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Case Report Open Access

Electrical Storm in a Patient with Acute Myocardial Infarction

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Abstract

60-year-old male admitted to hospital with chest pain. He was diagnosed as Acute Extensive Anterior Myocardial Infarction (AMI) and treated with stent placement. Postoperatively, the Electrocardiogram (ECG) showed third degree atrioventricular block. The patient went in cardiac shock and was treated with both temporary pacemaker and Intra Aortic Balloon Pumping (IABP). Despite IABP, antiarrhythmic drugs and β-blocker, Ventricular Tachycardia (VT) and Ventricular Fibrillation (VF) had been occupied frequently and cardioversion was performed 63 times.

Keywords: Myocardial infarction; Ventricular tachycardia; Ventricular fibrillation; Electrical storm

Introduction

Electrical storm is defined as "three or more distinct episodes of VF (Ventricular Fibrillation) or hemodynamically destabilizing VT (Ventricular tachycardia) within a 24 hours period [1]. It typically requires treatment with electrical cardioversion or defibrillation. The most common etiologies of electrical storm are those that are often responsible for ventricular tachydysrhythmias in general, including Acute Myocardial Ischemia or Infarction (AMI) and structural heart disease such as cardiomyopathy. Also, changes in the cellular membrane potential associated with inflammatory conditions (such as acute myocarditis) may lead to a dysrhythmia. On the other hand, frequent ventricular tachydysrhythmias and electrical storm may also occur in the structurally normal heart, such as the prolonged QT syndrome, Brugada syndrome [2,3], and serum electrolyte disturbances.

We present a case of a patient with AMI who developed electrical storm after Percutaneous Coronary Intervention (PCI), which was suppressed with conversion, and overdrive pacing (Figure 1).

Case Report

A 60-year-old man was admitted. He was still hypotensive and required dopamine to preserve favorable hemodynamics. What medications were used? Specifically, was beta blockade used as part of the acute MI protocol? If not, why not? What was the ejection fraction? Wall motion abnormalities?

On the 7th day in hospital, the patient had a cardiac arrest from ventricular tachycardia. Cardio Pulmonary Resuscitation (CPR) and two cardioversions (300 J) were performed. Then, amiodarone (150 mg) was administered intravenously, followed by a 0.5 mg/minute infusion. The patient regained consciousness and breathed spontaneously.

On the 8th day in hospital, VT/VF occurred frequently, despite cardioversion, drug therapy including amiodarone and lidocaine. Cardioversion was performed 21 times during 48 hours.

On day 10 of hospital admission, the episodes of VT were still frequent. The pacemaker pacing rate was increased to 90 beats/min. The frequency of VT episodes gradually declined. Ten days later, however, an electrical storm reoccurred. Intravenous metoprolol 5 mg was administered. Oral amiodarone, 600 mg/day for 1 week, and 400 mg/day for 1 week, followed 200 mg/day, also metoprolol 75 mg/day was added.

All antiarrhythmic drugs, including amiodarone and lidocaine, were reduced gradually and ceased before discharge without recurrence of VT/VF. A total of 63 cardioversions were performed during admission. He was discharged with aspirin, clopidogrel, valsartan, bisoprolol fumarate (7.5 mg/day) and simvastatin. No major adverse cadiac events occurred during the following 3 months.

Discussion

We have described a patient with incessant VT/VF. Several antiarrhymic drugs, electrical conversion, and overdrive pacing 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 β-blockers had a significant

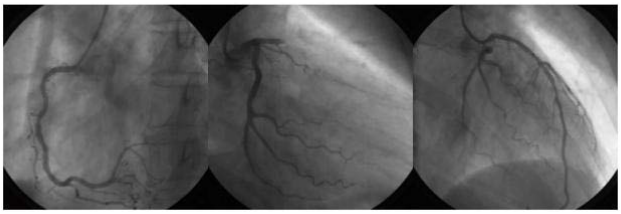

Figure 1: Coronary angiography showed occlusion of the proximal left anterior descending artery (LAD) that was treated with stent (Cypher 2.75 x 33mm, Cordis) lacement.

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reduction in primary outcome events compared to patients not on β-blockers. In our case, the patient was also treated with metoprolol associated with amiodarone, lidocaine, which had shown 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 catcholaminergic 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.

Furthermore, we found that overdrive pacing was effective in suppressing the electrical storm in a relatively short time. The same case was reported by Satoshi et al. [7]. Similarly, we started overdriving pacing as an adjunct to antiarrhythmic drug therapy; it is unclear whether the malignant arrhythmia was prevented only by the overdriving pacing.

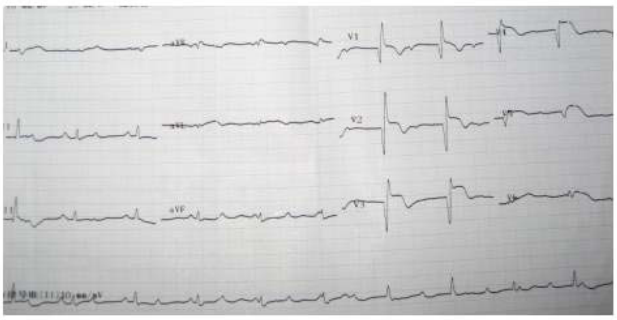

Figure 2: Three hours after having undergrone PCI, the ECG shows third-degree atrioventricular block with persistent ST-segment elevation and anterior Q waves.

During continuous ECG monitoring, however, we found that incessant VT/VF triggered by Monomorphic Ventricular Premature Beats (VPBs). Recently, radiofrequency ablation of the VPBs triggering incessant VT/VF after AMI is another therapeutic possibility and could prevent drug-resistant electrical storm [8]. This assumes a limited number of different premature beats. Hence, in our case, radiofrequency ablation could be proposed if the patient comes back with a new electrical storm.

In the present patient, once the electrical storm had passed, the malignant arrhythmia did not recur even without antiarrhythmic drug therapy, which suggests that an electrical storm during the early phase of AMI is not always associated with an increased risk of long-term mortality. If VT/VF is documented in the future, invasive electrophysiological testing should be considered for risk stratification.

In conclusion, we present this case to demonstrate the importance of recognizing sympathetically-mediated VT/VF in AMI. If the patient appears with arrhythmia early after PCI, (Figure 2) use of β-blockers or overdrive pacing early would be beneficial to prevent electrical storm and cardiac death postPCI.

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