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CASE REPORT

Electrical Storm Due to Active Myocardial Ischemia in the Right Coronary Artery Territory – Case Report

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ABSTRACT

Electrical storm is defined by at least three episodes of sustained ventricular tachyarrhythmias or appropriate shocks given by implantable cardiac defibrillator devices (ICD), occurring within a period of 24 hours. In the present manuscript, we present the case of a 69-year-old female patient with previous aortocoronary bypass, who was admitted from the Emergency Department after presenting several episodes of syncope in prehospital settings and presented 4 episodes of sustained ventricular tachycardia which required electrical cardioversion. The arrhythmia disappeared after percutaneous revascularization of a chronic occlusion in the right coronary artery. In this case, the implantation of an ICD was avoided, as a reversible cause of ES has been identified and treated.

Keywords: electrical storm, ventricular tachycardia, myocardial revascularization, heart failure

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INTRODUCTION

The expression "electrical storm" (ES) was first described in the 1990's, to indicate a scenario of electrical instability due to repeated ventricular arrhythmias in a short period of time. This entity was associated with a high mortality rate, requiring urgent therapeutic interventions, intensive cardiovascular care, multiple electrical cardioversions, and hemodynamic support.[1,2] The presence of more than three sustained episodes of ventricular tachyarrhythmias, or appropriate shocks given by implantable cardiac defibrillator devices (ICD) occurring during a period of 24 hours is the current accepted definition of ES.[3] Sustained ventricular arrhythmias include sustained ventricular tachycardia (lasting more than 30 seconds, with subsequent hemodynamic impairment or requiring therapeutic intervention for termination), and ventricular fibrillation.[4]

Sudden cardiac death (SCD), as a possible consequence of ES, is accountable for more than half of the total cardiovascular deaths, from which 25% represent the first manifestation of an asymptomatic, silent underlying cardiovascular disorder.[5,6] Out-of-hospital cardiac arrest is linked to a still low survival rate of 10%, being even lower in cases occurring at the patient's home (6% survival rate), compared to cardiac arrest occurring in public places, when the association of by-stander resuscitation, the use of automatic external defibrillators, and early arrival of first responders can lead to an increase in survival

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rates.[7] On the other hand, in-hospital cardiac arrest has a survival rate as high as 24%, depending on the underlying cause and the availability of intensive care and advanced life support.[3,7]

ES is associated with increased mortality rates, albeit the exact number is still unknown. Being a life-threatening condition, it requires emergency hospitalization. The MADIT II (Multicenter Automatic Defibrillator Implantation Trial) study, conducted on 719 patients with ICD, out of which 4% (n = 27) presented ES, revealed that there was a 17.8-fold increase in the 3-month death rate (95% CI 8–39.6, p <0.01) for subjects that presented ES compared to those with no arrhythmic events. This risk was maintained even after 3 months (HR for death 3.5, 95% CI 1.2–9.8, p = 0.02).[8] The clinical presentation of ES includes a variety of symptoms that can range from lack of any symptoms to syncope or pre-syncopal states. In some cases, it can lead to cardiogenic shock in subjects with repeated episodes of ventricular arrhythmias associated with hemodynamic impairment and requiring critical cardiac care.[9] The presence of ES in patients with non-ischemic cardiomyopathy is also associated with increased mortality rates, as well as with higher rates of patients requiring heart transplantation (up to 54%).[10] ES is also associated with a higher frequency of rehospitalizations and with a significant impairment of quality of life; a subanalysis of the SHIELD (SHock Inhibition Evaluation with azimiLiDe) trial revealed a 3 times higher rate of hospitalizations related to arrhythmias (p <0.0001), in comparison to patients who presented isolated ventricular tachycardia or fibrillation.[11] The impact of ES on patient survival, hospitalization rates, and quality of life is clear, either directly, or as an expression of severe cardiovascular or systemic disease.[12]

In the present manuscript, we present the case of a 69-year-old female patient, who was admitted to our clinic from the Emergency Department, after presenting several episodes of syncope in prehospital settings. The patient signed a written informed consent, in which she approved the publication of her medical information, and the study procedures were performed according to the ethical principles stated in the Declaration of Helsinki. The publication of this case was also approved by the ethics committee of the hospital.

CASE PRESENTATION

A 69-year-old female patient, with a history of coronary artery disease and heart failure (HF), presented to the Emergency Department of a secondary care hospital after 3 syncopal episodes occurring in prehospital settings, associated with symptoms of acute HF. The patient had a history of known coronary artery disease (CAD), with previous anterior acute myocardial infarction (AMI), resuscitated cardiac arrest, and multivessel CAD, for which she underwent coronary artery bypass grafting (left internal mammary artery – left anterior descending artery; right mammary artery – first marginal artery; and saphenous vein graft to second left obtuse marginal artery), 3 years prior to current presentation. After the surgical revascularization, during the course of 2 years, she presented 3 other hospital admissions for HF-related symptoms, for which she was treated with intravenous diuretics.

The current presentation to the Emergency Department resulted in admission to a secondary care hospital, for monitoring and pharmacological stabilization of the decompensated HF. Echocardiographic examination revealed a severely impaired left ventricular (LV) function with an ejection fraction of 25%, presence of an LV aneurysm with apical thrombosis, and a hemodynamically significant mitral regurgitation, for which she was administered continuous intravenous furosemide, angiotensin converting enzyme inhibitors, beta blockers, and anticoagulation (fractioned heparin). During hospitalization, she presented 4 episodes of sustained ventricular tachycardia in a period of 12 hours (observed during continuous ECG monitoring), with syncope and hypotension (80/40 mmHg), for which external electrical synchronous cardioversion (200 J) was administrated, followed by intravenous administration of amiodarone (900 mg over a 12-h period).

After electrical stabilization, the patient was referred to a tertiary care hospital, in a PCI center, for further investigations and treatment. During transportation, the patient presented 3 other episodes of self-limiting ventricular tachycardia, for which no electrical cardioversion was required.

Upon admission in the tertiary care unit, the patient was stable and asymptomatic, and the ECG tracing revealed sinus rhythm with left bundle branch block, ventricular extrasystole with "R-on-T" phenomenon, and a prolonged corrected QT interval (550 milliseconds) (Figure 1). Laboratory revealed no electrolyte imbalance, but there were increased levels of troponin I (11.1 ng/mL) and creatine-kinase MB (329 ng/mL).

The patient was admitted to the Intensive Cardiovascular Care Unit for observation, being scheduled for invasive coronary angiography on the following day. During the course of the next 12 hours, the patient presented several polymorphous ventricular extrasystoles, which triggered

3 episodes of non-sustained ventricular tachycardia for which intravenous magnesium sulfate (2.5 g), and continuous lidocaine infusion was initiated (with a rate of 1 mg/min). Lidocaine was preferred over amiodarone due to the prolonged QTc interval. The patient also presented 2 episodes of sustained ventricular tachycardia (Figure 2) with hemodynamic impairment, with a sudden decrease of blood pressure to 75/40 mmHg, obnubilation and diaphoresis followed by syncope, for which external electrical defibrillation was performed.

Invasive coronary angiography showed patency of the previous aortic coronary bypass grafts (Figure 3 A, B, C), and identified a total occlusion of the right coronary artery (RCA) (Figure 4) with no other significant associated lesions. The RCA was considered the culprit lesion responsible for triggering the ES, in the absence of other obvious causes. Interventional revascularization of the RCA was performed, with percutaneous implantation of 2 drug-eluting stents (one in the distal vertical segment, and one in the posterior interventricular artery), followed by several postdilation with non-compliant balloons required by extensive calcifications. The post-procedural result was good, with a TIMI III flow after revascularization (Figure 5).

After revascularization, the patient did not present any other episode of electrical instability, and no repeated sustained or non-sustained ventricular tachycardias were recorded.

The patient was discharged 3 days after revascularization, on dual antiplatelet therapy, oral anticoagulation, ACE inhibitors, diuretics, and beta blockers.

DISCUSSIONS

The present case was a female patient with ES presenting a history of significant CAD and secondary HF, who underwent coronary revascularization that led to the cessation of the arrhythmic episodes. The syncopal episodes occurring in prehospital settings were probably caused by recurrent ventricular tachycardias. Although there was no evidence of ES before hospital admission, the patient had presented multiple syncopal episodes during sustained ventricular tachycardias that occurred during the hospitalization period.

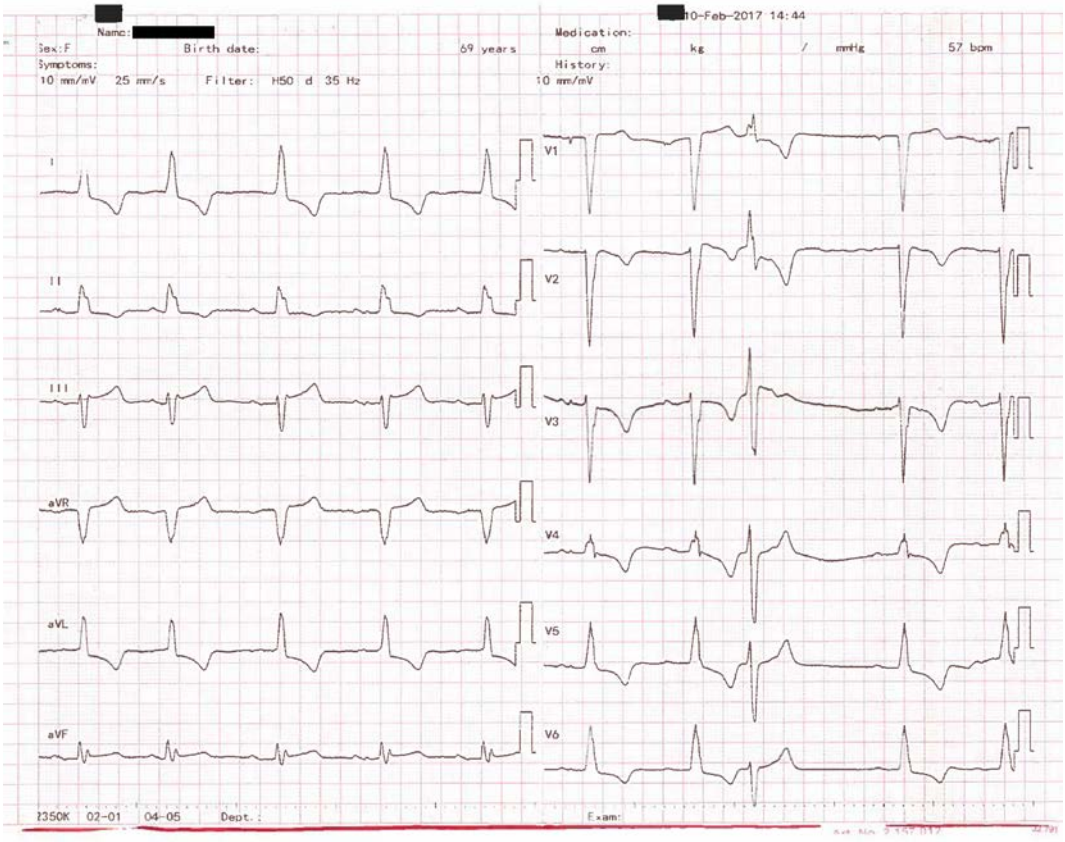

FIGURE 1. Transthoracic 12-lead ECG tracing showing sinus rhythm, left bundle branch block, and ventricular ectopic beats with "R-on-T" phenomenon

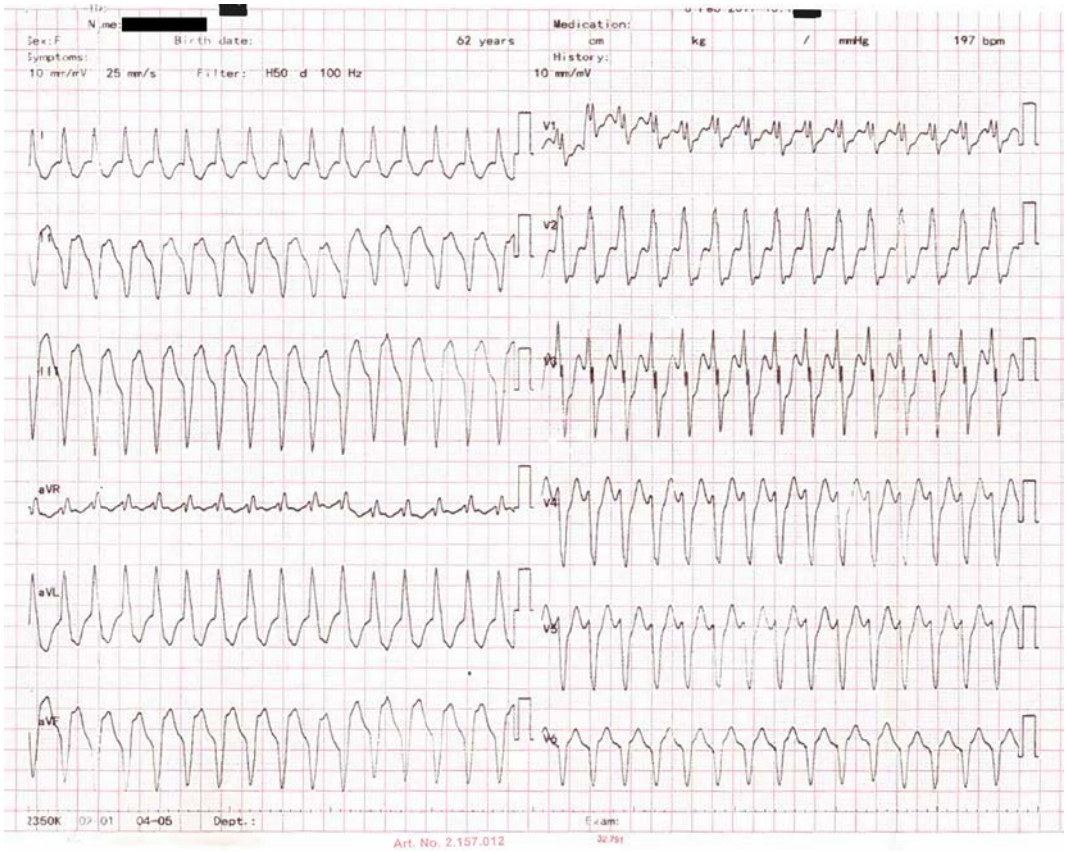

FIGURE 2. Transthoracic 12-lead ECG showing sustained ventricular tachycardia

The most frequent cause of sustained ventricular tachyarrhythmias is the presence of structural heart disease, which triggers re-entry mechanisms around the fibrotic area within the myocardium. Myocardial fibrosis occurs as a structural modification in ischemic and non-ischemic cardiomyopathies, after AMI with subsequent LV remodeling.[13] Other triggers include electrolyte disturbances, worsening HF, and even psychological stress due to adrenergic activation. Risk factors for ES include a severely impaired LV function, end-stage renal disease, or the presence of initial ventricular tachycardia as associated with the development of ESs.[14] The presented case was a patient with ischemic dilated cardiomyopathy, occurring after a previ-

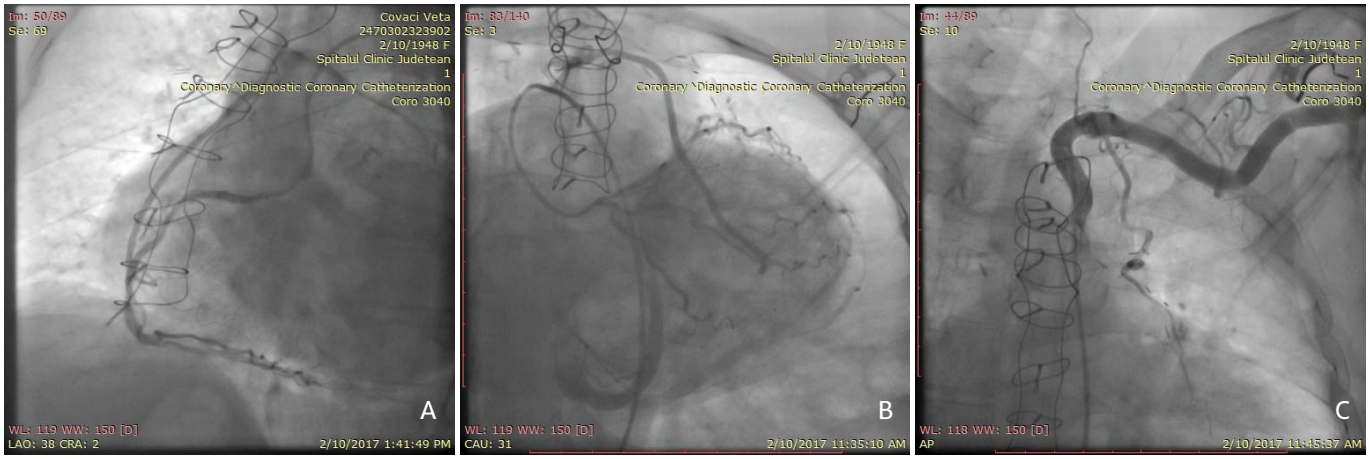

FIGURE 3. Invasive coronary angiography showing normal flow at the level of the coronary artery bypass grafts, and extensive multivessel coronary artery disease. **A** – patency of the bypass graft to first marginal artery; **B** – patency of the bypass graft to second left obtuse marginal artery; **C** – patency of the bypass graft between left internal mammary artery and left anterior descending artery

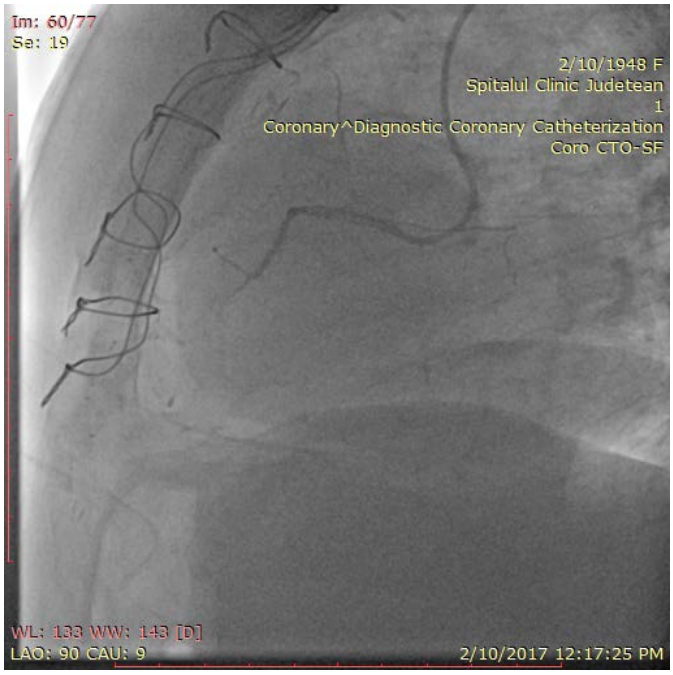

FIGURE 4. Angiography of the right coronary artery showing 60% aorto-ostial stenosis, followed by occlusion in the vertical segment

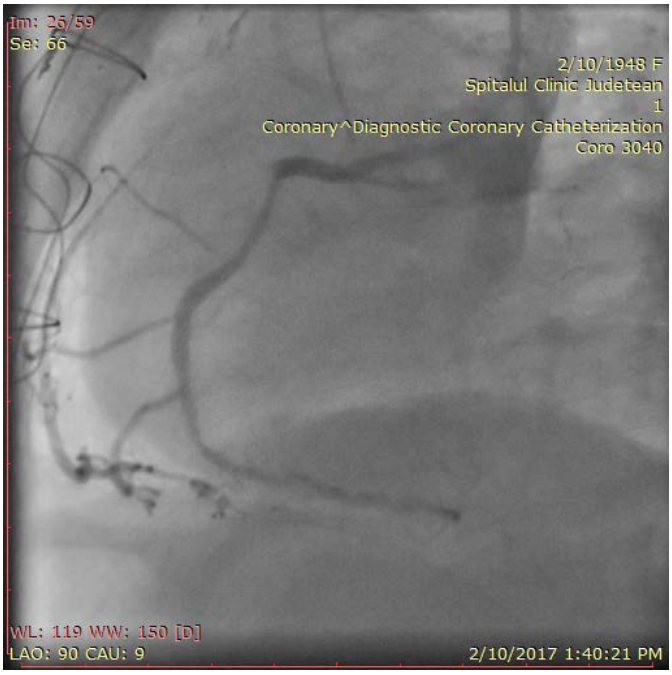

FIGURE 5. Angiographic aspect of the right coronary artery following revascularization

ous anterior myocardial infarction, with subsequent LV remodeling and aneurysm.

The management of ES requires emergency diagnostic and therapeutic interventions, as well as hospital admission regardless of the presence or absence of hemodynamic instability. Firstly, the triggers and risk factors for the ES should be sought and treated, which refers to identification of myocardial ischemia, acute HF or decompensated chronic HF, electrolyte imbalance, medications, and other systemic disease.[12] In the present case, myocardial injury and active ischemia were revealed true, being demonstrated by elevated levels of cardiac necrosis enzymes (troponin I and CK-MB), which directed patient management towards invasive coronary angiography and subsequent percutaneous revascularization.

The pharmacologic management of ES includes the use of antiarrhythmic medication, which can be further upgraded to electrical and interventional therapies, according to the patient's condition. Antiarrhythmic medication used in ES include beta blockers, amiodarone, sotalol, azimilide, dofetilide, or their combinations.[12] The patient had been already on efficient treatment with beta blockers for ischemic heart failure, and was administered 900 mg of amiodarone in the secondary care unit. This led to a severe prolongation of the corrected QT interval, which may have contributed to further initiation of ventricular arrhythmias. Due to the lack of response to amiodarone, the patient was switched to continuous lidocaine infusion and magnesium sulfate. Despite this, she continued to present electrical instability and became hemodynamically unstable, requiring electrical cardioversion with 200 J external electrical synchronous shock.

Patients with ES are also to be evaluated for hemodynamic instability and treated accordingly. Furthermore, early sedation and use of analgesia should be considered in patients with adrenergic stimulation, in those who require multiple shock therapies, or in patients with ICDs who have received multiple shocks.[13,15] In case of monomorphic ventricular tachycardia, catheter ablation of the re-entry pathway for definitive treatment of ES is superior to medical treatment and significantly reduces the arrhythmia burden and ICD shock.[16]

In the presented case, the patient did not present any other episodes of ventricular tachyarrhythmias after revascularization, which suggests that the cause of ES was the myocardial ischemia in the territory supplied by the RCA. Furthermore, the patient did not benefit from intracardiac defibrillator, which was not indicated as there was a correctable cause for the electrical storm.

Implantation of an ICD is contraindicated in the acute phase of ES, but the current therapeutic guidelines recommend device therapy for the secondary prevention of SCD in patients who had suffered an ES, with untreatable structural cardiac disease, after the exclusion of correctable causes such as acute coronary syndromes or dyselectrolitemia.[3]

CONCLUSIONS

The presented case illustrates a clinical scenario in which chronic myocardial ischemia due to RCA occlusion became suddenly the trigger for several episodes of ventricular tachyarrhythmias, with intermittent hemodynamic instability, requiring repeated electrical cardioversion. ES was treated by revascularization of the chronically occluded RCA. In this case, the implantation of an ICD was avoided, as a reversible cause of ES has been identified and treated.

CONFLICT OF INTEREST

Nothing to declare.

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