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Successful Management of Cardiac Electrical Storm in Wellens Syndrome: A Case Report

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ABSTRACT

Wellens syndrome patients are at risk for acute myocardial infarction with a large acute anterior wall because of the lesion in left anterior descending coronary artery and percutaneous coronary intervention is the definitive treatment to relieve the occlusion in left anterior descending coronary artery. Electrical storm is defined as the three or more episodes of ventricular tachycardia or ventricular fibrillation in any interval of 24 hours. The appearance of cardiac electrical storm is a critical situation with poor outcome. We would like to present a case of Wellens syndrome, with the significant stenosis in left circumflex artery and Ramus intermedius or Median Ramus artery apart from the culprit lesion in left ascending artery, suffering from the cardiac electrical storm after percutaneous coronary intervention in the left ascending coronary artery - the culprit artery and it was managed successfully with the complete revascularization in all lesions thanks to the intravenous infusion of amiodarone and lidocaine in different veins as an option for controlling the electrical storm.

Keywords: Wellens syndrome; Cardiac electrical storm; Complete revascularization; Infusion of amiodarone; Lidocaine.

List of Abbreviations: CPR: cardiopulmonary resuscitation; DAPT: dual antiplatelet therapy; ECG: electrocardiography; ES: electrical storm; IV: intravenous; LAD: left anterior descending; LCx: left circumflex artery; MR: median ramus; PCI: percutaneous coronary intervention; PVC: premature ventricular contractions; VF: ventricular fibrillation; VT: ventricular tachycardia

INTRODUCTION

Wellens syndrome, also called left ascending artery (LAD) coronary syndrome or widow maker, was first described by de Zwaan et al in patients with unstable angina during a pain-free period with electrocardiographic (ECG) changes[1]. There are two types of the syndrome, including type A and type B manifesting with biphasic T waves and deeply inverted T waves in leads V2-V3, respectively[2]. If the patients present with these types of ECG, it is highly specific for severe, proximal stenosis of the left anterior descending coronary artery[3]. Normally, when Wellens syndrome patients come to the emergency department or cardiovascular center, they do not have chest pain with normal or slightly elevated cardiac enzymes[4]. However, it is crucial to attach the importance of the ECG patterns because these patients are at high risk for acute myocardial infarction with a large acute anterior wall. Percutaneous coronary intervention (PCI) is the definitive treatment to relieve the occlusion in LAD[5,6].

The definition of cardiac electrical storm (ES) is as the three or more episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) in any interval of 24 hours[7]. There are many causes of ES, which can be divided into two categories scar-mediated re-entry due to previous myocardial infarction and reversible causes (acute ischemia, acute decompensated heart failure, electrolyte abnormalities, drug toxicity, sepsis and thyrotoxicosis) [8]. Electrical storm is considered as a critical and severe situation both on management of hemodynamically unstable arrhythmias and its association with significant elevated sympathetic tone, which are likely to trigger further arrhythmias[9]. Normally, patients with ES are treated with antiarrhythmic medications serially and suffer repeatedly from electrical shocks[10]. The key intervention in electrical storm is to revascularize if patients experiencing with myocardial infarction and to block the sympathetic system through beta-blockers, especially propranolol; combined with analgesics and sedatives along with the control of serum electrolytes (particularly Mg2+, K+) at high levels by the infusion of intravenous electrolyte solution[7,11-13].

We would like to present our case with the history of diabetes type 2 hospitalized with the ECG of Wellens syndrome along with the angiography showing the severe and diffuse stenosis of left anterior descending artery (LAD) and the significant stenosis of left circumflex artery (LCx) and Median Ramus (MR) artery was treated successfully with complete revascularization, antiarrhythmic drugs, intravenous sedative and electrolyte adjustment.

CASE REPORT

A 42-year-old male patient with a history of type 2 diabetes under the treatment of 2000mg/day of Metformin presented with typical angina chest pain and dyspnea for 3 days before being hospitalized. The ECG findings showed the pattern of type A Wellens Syndrome (Figure 1), the echocardiography indicated the ejection flow reduced at 28% with contractile dysfunction in the wall motions of the left ventricular. The cardiac enzyme hs-Troponin T was 1.00 ng/ml.

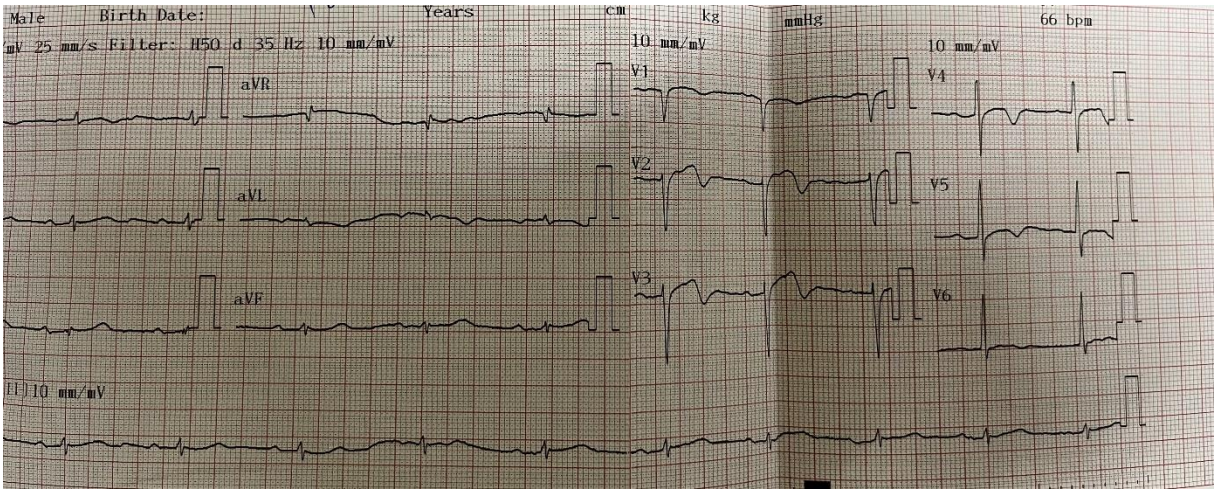

Figure 1: ECG findings on admission: Biphasic T waves in leads V2-V3

The patient was treated by enoxaparin, DAPT (Aspirin + Clopidogrel), rosuvastatin, angiotensin-converting-enzyme inhibitors, spinorolactone, low-dose of dobutamine and insulin human (rDNA). The primary percutaneous coronary intervention (PCI) was performed for the patient with the angiographic result of severe and diffuse stenosis 90-95% in proximal LAD and the significant stenosis 80-90% in proximal LCx and proximal MR (Figure 2). We decided to deploy a drug-eluting stent in the LAD based on the ECG findings of Wellens syndrome (Figure 2).

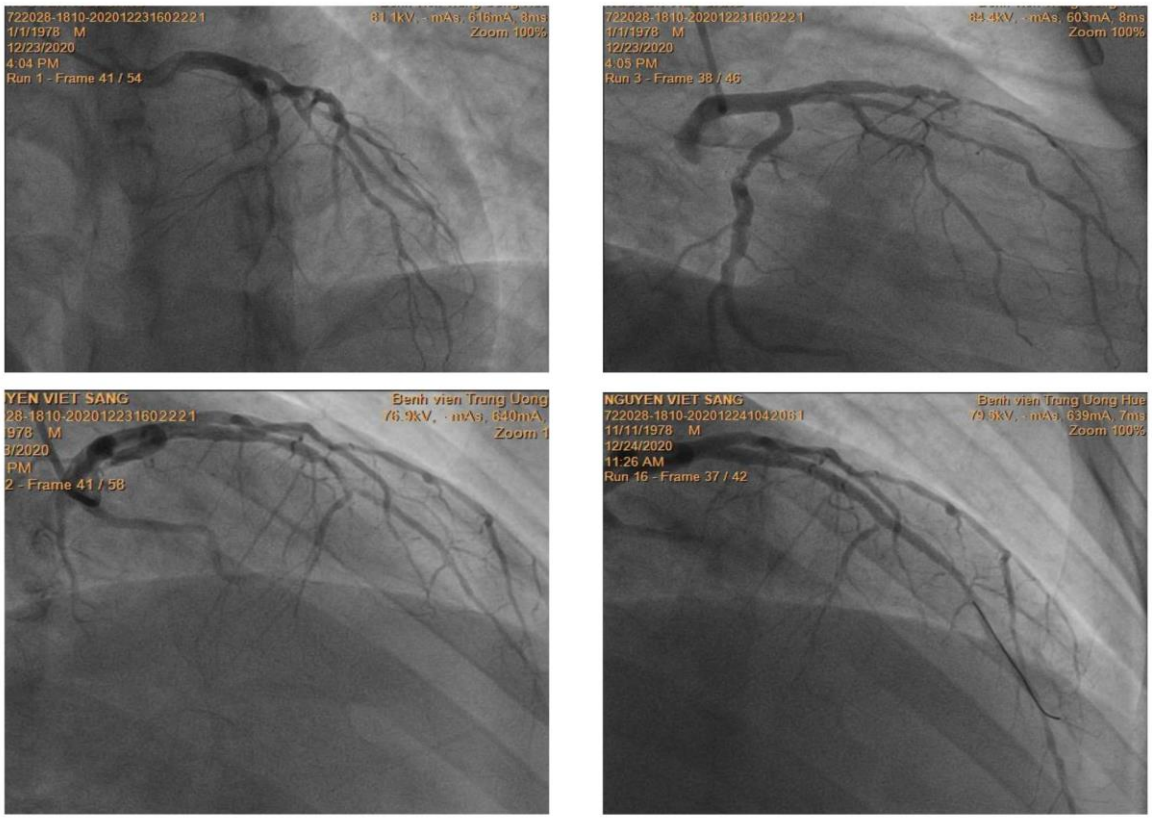

Figure 2: Coronary angiograms demonstrated the severe and diffuse stenosis in the proximal left anterior descending (LAD), the significant stenosis also in the proximal left circumflex artery (LCx) and median ramus (MR). Deploying a drug-eluting stent in LAD as a culprit lesion

The patient was stable after that. On the day two after PCI, the patient suddenly suffered from cardiac arrest and was treated successfully with cardiopulmonary resuscitation, three times of defibrillation and IV adrenaline. The ECG after 30 minutes showed the premature ventricular contractions (PVC) bigeminy (Figure 3), it was managed by amiodarone with 150mg IV bolus and the IV maintenance dose of 1mg/min for 6 hours and 0.5mg/min IV after that. By doing so, the ECG finding was sinus rhythm with occasional PVCs. At that time, the serum potassium and magnesium were 3.8 mmol/L and 1.1 mmol/L, respectively, which was under the IV infusion of electrolytes including 2-gram kali chloride 10%- and 1.5-gram magnesium sulfate. Because of signs of congestive heart failure including rales at both lung bases, we were afraid of indicating beta blockers for the patient.

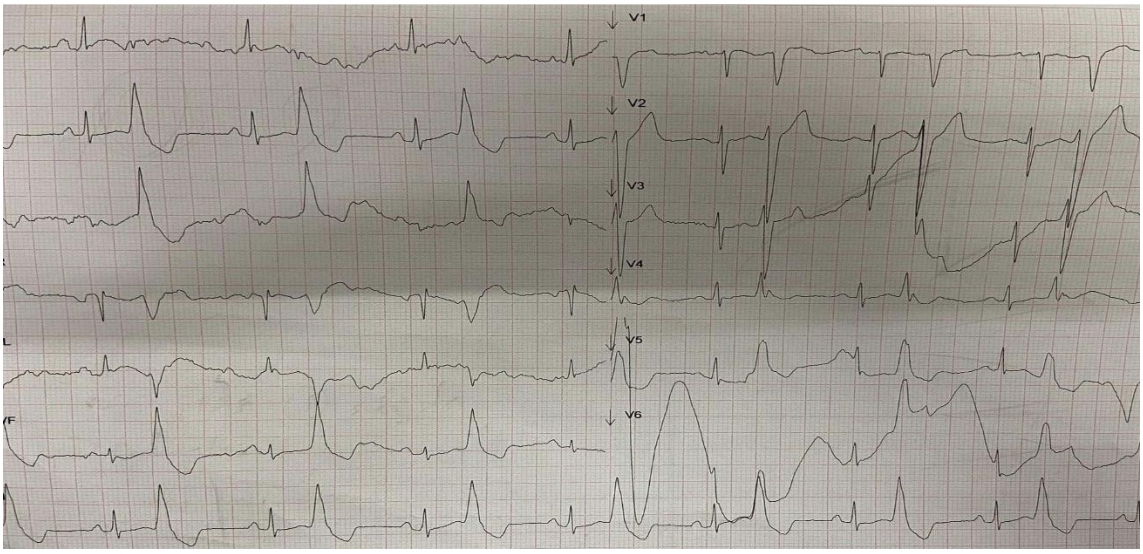

Figure 3: PVCs Bigeminy after successful management of sudden cardiac arrest

The next day, while the patient was under treatment for a maintenance dose of amiodarone and electrolytes adjust, he was still suffering from more than 5 times of ventricular tachycardias (Figure 4) and intermittent ventricular fibrillations, which is also called cardiac electrical storm. The patient was managed with the combination of CPR and IV epinephrine, a number of defibrillations, 10mg diazepam for sedative and 1gram acetaminophen infusion of analgesics, IV potassium and magnesium, especially with the addition of one more antiarrhythmic drug called lidocaine with 1mg/kg for the bolus dose and 1mg/min for continuous infusion dose in different vein. After 30 minutes, the patient was stable hemodynamically with the sinus rhythm in ECG.

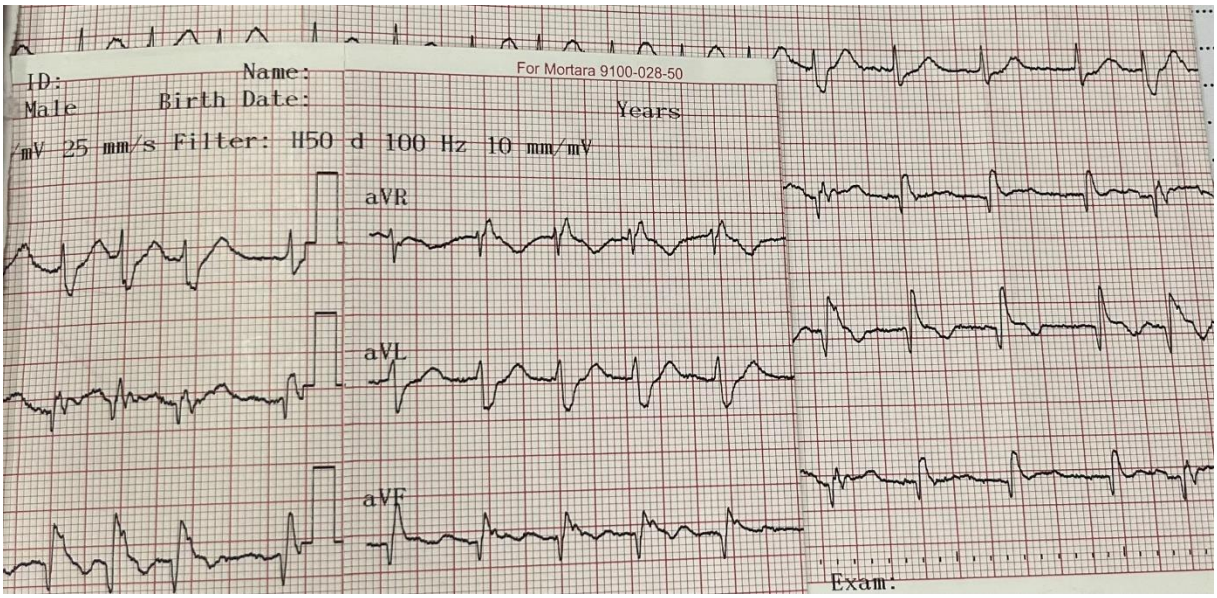

Figure 4: Monomorphic ventricular from the onset of electrical storm

After that, we decided to transfer the patient to cath-lab in order to perform PCI with drug-eluting stents in LCx and MR arteries (Figure 5). Thanks to the complete revascularization, the patient was stable with sinus rhythm. The patient was followed up within a week and was discharged thanks to the hemodynamic stable condition with sinus rhythm and no arrhythmias. However, the cardiac fraction ejection did not improve. Therefore, the patient was treated with DAPT (81 mg Aspirin and 75 mg Clopidogrel), high-dose of rosuvastatin at 40mg, 5 mg of perindopril, 25mg of spinorolactone, low-dose of bisoprolol at 1.25mg, 2000 mg of Metformin and 10 mg of dapagliflozin. After 3 months of following up, the patient was stable with sinus rhythm and the cardiac function was improved from 28% to 48%, which also meant we did not indicate implantable cardioverter-defibrillator for the prevention of sudden cardiac death due to ventricular fibrillation and ventricular tachycardia.

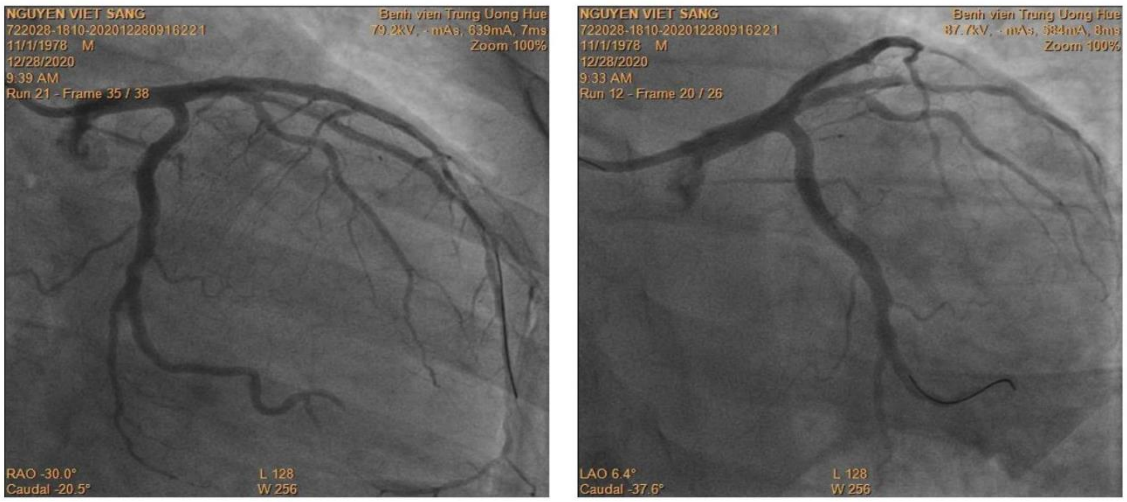

Figure 5: The final coronary angiograms after deploying percutaneous coronary interventions in LCx and MR with drug-eluting stents

DISCUSSION

Wellens syndrome patients frequently manifest symptoms similar with acute coronary syndrome; the ECG pattern of the patients presents from 14 to 18% of unstable angina patients in the study of Wellens et al[2]. The Wellens syndrome is highly specific for stenosis or a temporary obstruction of the LAD coronary artery caused by the rupture of an atherosclerotic plaque. If it is not identified early and treated properly with percutaneous coronary intervention (PCI), the patients are at risk of a large acute anterior wall myocardial infarction. Therefore, it is crucial for physicians and cardiologists to identify Wellens patterns on the ECG as soon as possible in order to have better management for the patients[1,5,6]. In our case, the patient unluckily had severe and diffuse stenosis of 95% proximal LAD and significant stenosis of 80-90% proximal LCx and 80% proximal MR.

Cardiac electrical storm (ES) is defined as the three or more episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) within 24 hours[7]. There are many causes of ES, which can be divided into two categories scar-mediated re-entry due to previous myocardial infarction and reversible causes such as acute ischemia, acute decompensated heart failure, electrolyte abnormalities, drug toxicity, sepsis and thyrotoxicosis[8]. Cardiac electrical storm is a relatively rare complication but poor prognosis of acute myocardial infarction that can result in death if it is not controlled immediately. A study of non-ST-elevation acute coronary syndrome patients who underwent cardiac catheterization within 48 hours showed VT/VF at 7.6%[14]. While there were 5.7% patients developed sustained VT or VF with two thirds of these events happening prior to the end of the catheterization and 90% within 48 hours from the procedure according to a clinical trial[2]. Typically, ESs require treatment with electrical shocks and coronary revascularization along with blocking the sympathetic tone, antiarrhythmic drugs, analgesics and sedatives along with electrolyte control[7,11-13].

In our case, without the history of syncope, and even when being hospitalized, there was no report of VT and VF. The treatment included DAPT, exonaparin, angiotensin-converting enzyme inhibitors, dobutamin and insulin human (rDNA) along with primary PCI for the culprit lesion in LAD according to the ECG of Wellens syndrome, the patient was being followed up at the cardiac intensive care unit room. On the second day after PCI, the cardiac arrest occurred spontaneously, the patient was managed by cardiopulmonary resuscitation (CPR), epinephrine, three times cardiac shocks because of VTs, after 30 minutes, the ECG was present with bigeminy premature ventricular contractions (PVC) which was managed by amiodarone bolus with 150mg IV bolus and the IV maintenance dose of 1mg/min for 6 hours and 0.5mg/min IV after that, along with intravenous infusion of potassium and magnesium. Despite that, in the next day, the episodes of VTs occurred and the patient was diagnosed with cardiac ES and was controlled by the combination of CPR, cardiac shocks, diazepam for sedatives and acetaminophen infusion of analgesics, IV potassium and magnesium, particularly with the addition of another antiarrhythmic drug called lidocaine with 1mg/kg for the bolus dose and 1mg/min for continuous infusion dose in different vein, along with the continuous dose of amiodarone at 0.5mg/min. With all of aforementioned treatment, the ECG was back with sinus rhythm and the patient was stable hemodynamically and immediately transferred to the catheterization room in order to perform PCI for the LCx and the MR which were 80-90%, 80% stenosis in the proximal segment, respectively, and were thought to be as a cause of the ventricular arrhythmias for our patient. After PCI for LCx and MR artery, the patient did not show any arrhythmias in the ECG and the clinical examination was progressively better without chest pain and shortness of breath. However, the ejection

fraction did not show any improvement. He was treated with DAPT (81 mg Aspirin and 75 mg Clopidogrel), high-dose of rosuvastatin at 40mg, 5 mg of perindopril, 25mg of spinorolactone, low-dose of bisoprolol at 1.25mg, 2000 mg of Metformin and 10 mg of dapagliflozin. We intended to perform Implantable Cardioverter-defibrillator (ICD) to prevent sudden cardiac death. However, after 3 months of following up the above treatment, the patient was stable with sinus rhythm and the cardiac function was improved to 48%, therefore, we decided not to do so and keep following up the patient with the treatment.

Herein according to the joint EHRA, ACCA and EAPCI task force in 2014 and 2017 AHA/ACC/HRS guideline for Management of patients with ventricular arrhythmias and the prevention of sudden cardiac death[16,17], patients with acute coronary syndrome, presenting with the condition of incomplete revascularization as mentioned in our case with the significant stenosis of LCx and MR besides the culprit lesion in LAD, are at increased risk for the development of arrhythmia. Currently, there has been a lack of larger trials comparing the benefit from complete revascularization and incomplete revascularization. Generally, it is recommended that non-culprit lesion revascularization should be performed by a staged percutaneous coronary intervention in order to achieve the complete revascularization. Therefore, after the successful PCI of the culprit lesion, it is advisable to treat other lesions in the duration of hospitalization[18].

In addition, we believe that if one antiarrhythmic drug could not control the rhythm, the additional one in a different group should be considered. We suppose that intravenous infusion of amiodarone and lidocatin in different veins should be considered as an option for controlling the ventricular arrhythmias along with the control of electrolytes, analgesics and sedatives.

CONCLUSIONS

In conclusion, we would like to propose three main experiences from our case:

1. Typically, the patients with the characteristics ECG of Wellens syndrome present with the significant stenosis or even occlusion of proximal in LAD coronary artery. However, in our case, apart from the lesion in LAD, there were also the significant stenosis of LCx coronary artery and even Median Ramus artery. We would like to raise the awareness of other lesions along with the LAD in Wellens syndrome.
2. Cardiac electrical storm might occur even after the percutaneous coronary intervention in the culprit lesion if there is also another significant stenosis. This is the reason why we would like to suggest cardiac interventionists to perform percutaneous coronary intervention in other significant stenosis during the hospitalization in order to prevent the arrhythmias.
3. Along with cardiac defibrillation, electrolytes management, analgesics and sedatives, we would like to propose the intravenous infusion of amiodarone and lidocaine in different veins as an option for controlling the electrical storm prior to percutaneous coronary intervention with complete revascularization.

Ethical Approval

The need for ethics approval for this case report was waived.

Consent for Publication

For the publication of this case report, written informed consent was obtained from the patient.

Disclosure

The authors report no other conflicts of interest in this work.

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