Successful Management Of Cardiac Electrical Storm In Wellens Syndrome: A Case Report

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July 11, 2022

Abstract

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ABSTRACT

A 42-year-old male patient diagnosed with Wellens syndrome, the angiography showed the narrowing not only in the left anterior descending artery but also in other arteries. Cardiac electrical storm can occur without complete revascularization and it could be managed successfully with the infusion of amiodarone and lidocaine along with cardiac defibrillation.

Keywords : Wellens syndrome, cardiac electrical storm, complete revascularization, infusion of amiodarone and lidocaine.

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 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 a critical and severe situation both on management of hemodynamically unstable arrhythmias and its association with a significantly 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 the electrical storm is to revascularize if patients experiencing 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 a case of Wellens syndrome suffering from the cardiac electrical storm and how we successfully managed the patient.

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.

The patient was treated with enoxaparin 1 mg/kg q12hr, DAPT (81 mg Aspirin and 75 mg Clopidogrel), 40mg Rosuvastatin, 5mg Perindopril, 25mg spironolactone, low-dose of dobutamine (5mcg/kg/min) and insulin human (rDNA). The primary percutaneous coronary intervention (PCI) was performed for the patient with the angiographic result of severe and diffuse stenosis of 90-95% in proximal LAD and significant stenosis of 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).

The patient was hemodynamically stable after that. On 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 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.

The next day, while the patient was under treatment for a maintenance dose of amiodarone and electrolytes

adjustment, he was still suffering from more than 5 times ventricular tachycardias (Figure 4) and intermittent ventricular fibrillation, 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 veins. After 30 minutes, the patient was stable hemodynamically with the sinus rhythm in ECG.

After that, we decided to transfer the patient to our 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.

DISCUSSION

In our case, the patient with typical ECG of Wellens syndrome unluckily had severe and diffuse stenosis of 95% proximal LAD and significant stenosis of 80-90% proximal LCx and 80% proximal MR. The treatment included DAPT, enoxaparin, angiotensin-converting enzyme inhibitors, dobutamine, 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 due to ventricular tachycardia which was successfully managed by cardiac defibrillations and amiodarone. Despite that, in the next day, the episodes of VTs continued to occur and the patient was diagnosed with cardiac ES which was controlled by 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. 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. 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, and 25mg of Spinorolactone, low-dose of Bisoprolol at 1.25mg, 2000 mg of Metformin and 10 mg of Dapagliflozin. We intended to perform an Implantable Cardioverter-defibrillator (ICD) to prevent sudden cardiac death. However, after 3 months of following up on 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 with the patient with the treatment.

Herein according to the joint EHRA, ACCA, and EAPCI task force in 2014 and 2017 AHA/ACC/HRS guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death, patients with the 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.[16, 17] 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 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 lidocaine in different veins should be considered as an option for controlling ventricular arrhythmias along with the control of electrolytes, analgesics, and sedatives.

CONCLUSIONS

In conclusion, the Wellens syndrome patients suffering from cardiac electrical storm could be managed successfully if complete revascularization could be carried out. Additional, we would like to propose the intravenous infusion of amiodarone and lidocaine in different veins as an option for controlling the cardiac electrical storm along with cardiac defibrillation.

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

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.

Acknowledgements

I would like to show my gratitude to my dear teacher, Anh Binh Ho, PhD, who guided me the right way to manage my patient and helped me to make my decision to write my case report.

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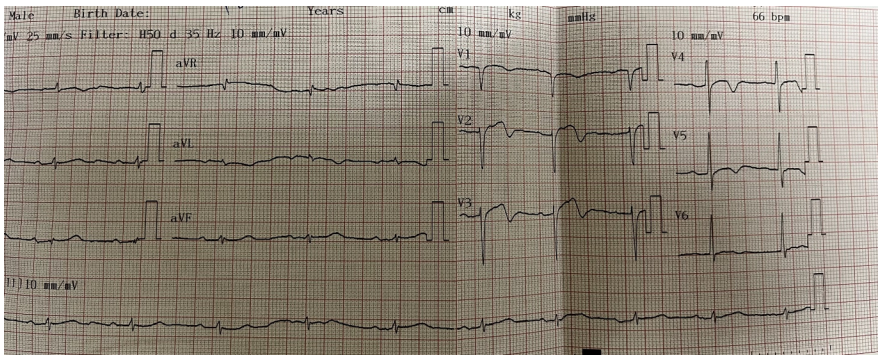

Figure 1. ECG findings on admission: Biphasic T waves in leads V2-V3

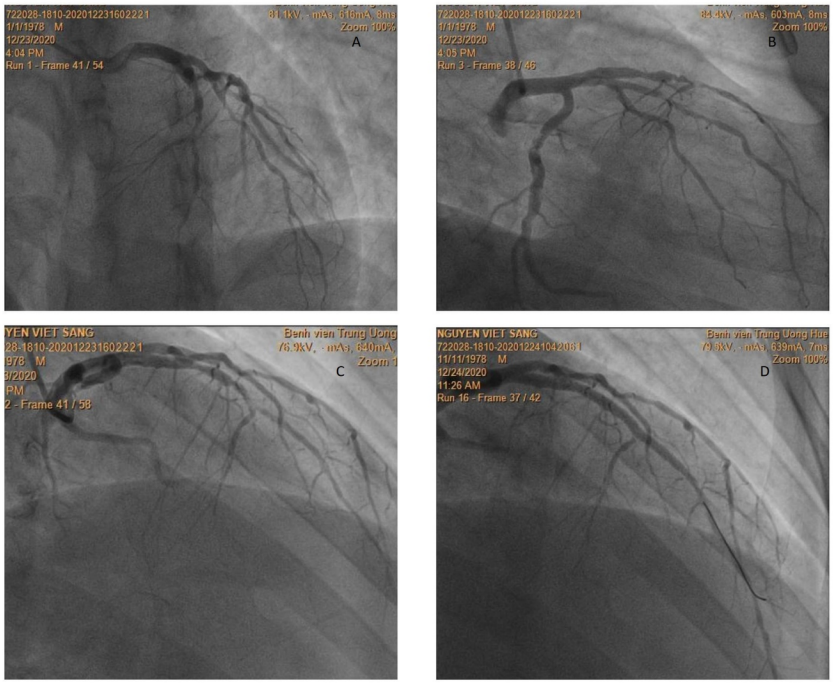

Figure 2. Coronary angiograms demonstrated the severe and diffuse stenosis in the proximal left anterior descending (LAD) (A, B, C), the significant stenosis also in the proximal left circumflex artery (LCx) and median ramus (MR) (B, C). Deploying a drug-eluting stent in LAD as a culprit lesion (D).

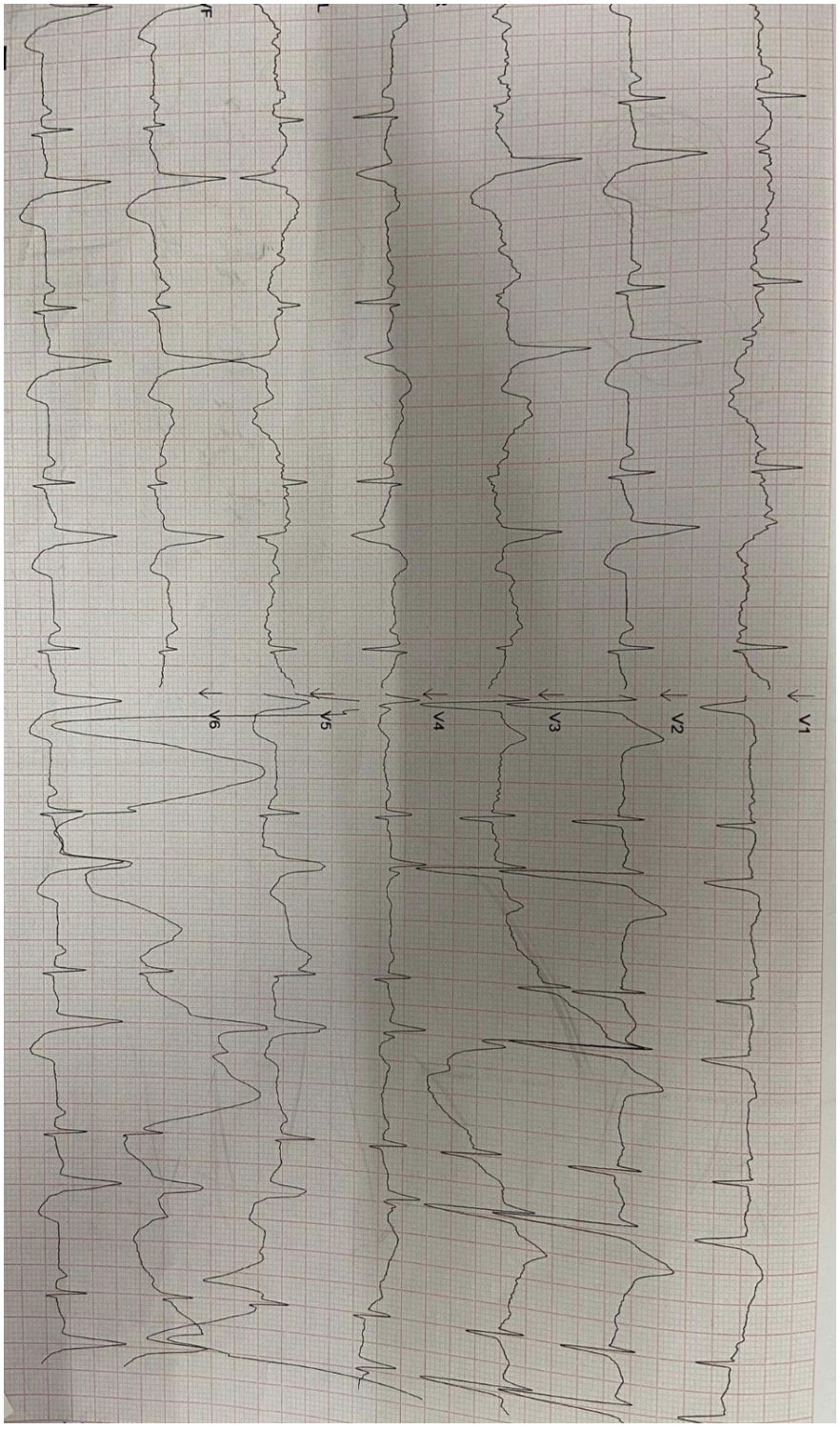

Figure 3. PVCs Bigeminy after successful management of sudden cardiac arrest

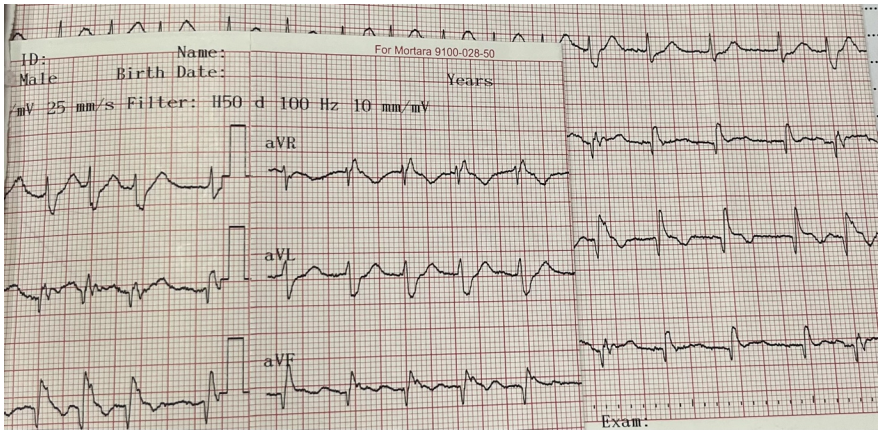

Figure 4. Monomorphic ventricular from the onset of electrical storm

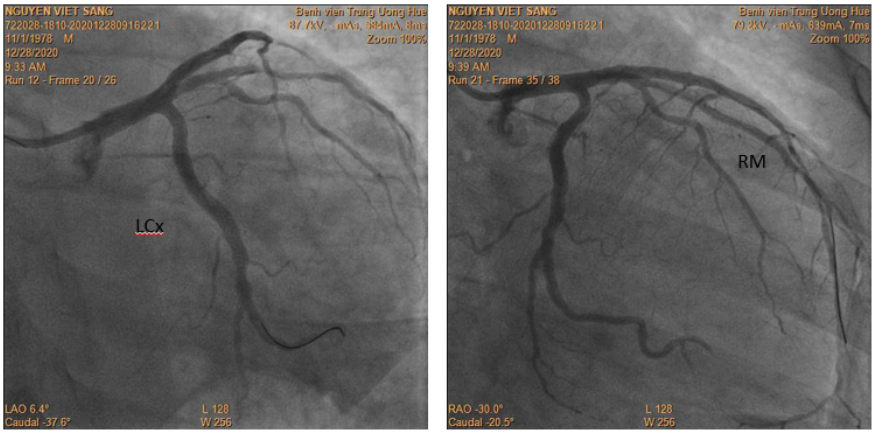

Figure 5. The final coronary angiograms after deploying percutaneous coronary interventions in LCx and RM with drug-eluting stents