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An interesting implantable cardioverter defibrillator treatment for lethal ventricular arrhythmias caused by coronary artery spasm

A case report

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Abstract

Rationale: Coronary artery spasm (CAS) could cause serious lethal ventricular arrhythmias. While implantable cardioverter defibrillators (ICDs) have been recommend for secondary prevention of sudden cardiac death related to lethal ventricular arrhythmias. However, in resuscitated sudden cardiac death caused by CAS, the effect of ICD is still not well clear.

Patient Concerns: A 60-year-old male presented with 2 episodes of syncope. Coronary angiography showed normal coronary arteries. Twenty-four hour Holter electrocardiograms revealed that there were repeatedly transient marked ST segment elevation in the all leads except avR lead, junctional rhythm, and subsequently nonsustained ventricular tachycardia.

Diagnoses: Ischemic-induced lethal ventricular arrhythmias caused by CAS.

Interventions: Both calcium channel blocker (diltiazem, 180 mg twice daily) and nitrate (isosorbide dinitrate 40 mg twice daily) were initially administrated, and ICD was subsequently implanted as a secondary prevention.

Outcomes: In the early stage of CAS, ICD therapy terminated the lethal ventricular arrhythmias. Conversely, after the administration of epinephrine, ICD therapy, even combined with external defibrillation, failed in resuscitating sudden cardiac death.

Lessons: For the sudden cardiac death related to lethal ventricular arrhythmias caused by CAS, ICD therapy is an efficient secondary prevention base on administrating coronary vasodilators. Furthermore, administration of epinephrine should be avoided during cardiorespiratory resuscitation of sudden cardiac death caused by CAS.

Abbreviations: CAS = coronary artery spasm, ICD = implantable cardioverter defibrillator.

Keywords: coronary artery spasm, implantable cardioverter defibrillator, lethal ventricular arrhythmias

1. Introduction

Coronary artery spasm (CAS) could lead to sudden cardiac death due to serious lethal ventricular arrhythmias. Despite calcium antagonists have been demonstrated to be an effective treatment for CAS and has a potential role for preventing ventricular arrhythmia, the ventricular arrhythmia may recurrent at any time. Implantable cardioverter defibrillators (ICDs) have been recommend for secondary prevention of sudden cardiac death related to lethal ventricular arrhythmias. However, in resuscitated sudden cardiac death caused by CAS, the effect of ICD is still not well clear. In the present report, we showed a case of ICD

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treatment in a man who suffered from recurrent episodes of lethal ventricular arrhythmias induced by CAS.

2. Case presentation

Written informed consent was obtained from patient's family, and institutional Ethics Committee of Zhongshan Hospital of Sun Yat-sen University approved this case report.

A 60-year-old male was admitted with 2 episodes of syncope. At each time of syncope, several-minute retrosternal chest pain presented firstly, consciousness lost suddenly, and recovered normal quickly. Findings of physical examination, laboratory tests, 12-leads electrocardiograms (Fig. 1A), x-ray, echocardiography, magnetic resonance imaging of head, and electroencephalography were normal. Coronary angiography indicated normal coronary arteries. Twenty-four hour Holter electrocardiograms revealed that there were repeatedly transient marked ST segment elevation in the all leads except avR lead, junctional rhythm, and subsequently nonsustained ventricular tachycardia (Fig. 1B). Therefore, a definitive diagnosis with ischemic-induced lethal ventricular arrhythmias caused by CAS was established. Both calcium channel blocker (diltiazem, 180 mg twice daily) and nitrate (isosorbide dinitrate 40 mg twice daily) were initially administrated, and ICD was subsequently implanted as a secondary prevention.

After ICD implantation, the patient stayed in a persistent state of anxiety. On the sixth day after implantation, the patient present repeated chest pain with marked diffuse ST segment

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Figure 1. Electrocardiograms of different periods. (A) Twelve-lead electrocardiogram showed normal. (B) Twenty-four hour Holter electrocardiograms revealed transient marked ST segment elevation in all leads except avR lead, junctional rhythm, and subsequently nonsustained ventricular tachycardia. (C) Ventricular fibrillation could not been terminated by external defibrillation.

elevation, exhibited sustained ventricular tachycardia and ventricular fibrillation. The ICD accurately detected and successfully treated by multiple intracardiac shocks to restore sinus rhythm. However, after repeated heart attacks, the blood pressure could not been hold normal, even with administration of three vasopressors (dopamine, norepinephrine, and epinephrine). Suddenly, the patient's condition turned down sharply. Ventricular fibrillation could not been terminated by both electrical therapies of ICD and external defibrillation (Fig. 1C), and electromechanical dissociation finally appeared.

3. Discussion

Prevalences of CAS in patients with angina were wide differences among different countries. Several pathogenetic mechanisms, such as endothelial dysfunction, primary smooth muscle cells hyperreactivity, abnormal production of growth factors, and adventitial abnormalities, have been demonstrated to participate in CAS.^[1] CAS could lead to transient myocardial ischemia and infarction and result in lethal ventricular arrhythmias.^[2,3]

Lethal ventricular arrhythmias could be decreased by coronary vasodilators based on pathogenetic mechanisms of CAS.^[2] Both calcium channel blockers and nitrates are the guideline medicine for relaxing vascular smooth muscle and preventing CAS.^[3] However, CAS in some patients showed less response to these drugs so that lethal ventricular arrhythmias cannot be suppressed. For this intractable CAS, how to resuscitate patients from sudden cardiac death due to lethal ventricular arrhythmias became a particular critical issue.

ICD has been a proven effective device for terminating lethal ventricular arrhythmias. Furthermore, several case reports have demonstrated that ICDs were also useful for lethal ventricular arrhythmias caused by CAS.^[4–6] However, there is still no large-scale clinical trial to support for it. On the contrary, Letsas et al^[7] reported a multivessel spasm case with lethal ventricular arrhythmias and continuous ICD therapy was no efficacy for

ventricular fibrillation. Therefore, in the field of lethal ventricular arrhythmias caused by CAS, the effect of ICD remained controversial.

The present case was quite different from those previous reported cases. He underwent 2 opposite responses to ICD therapy. In the early stage, ICD therapy terminated the lethal ventricular arrhythmias, even the electrocardiograms indicated multivessel CAS. The recurrence of CAS might be largely due to the persistent anxiety state of the patient. It showed that ICD was actually an effective device for secondary prevention of sudden cardiac death related to lethal ventricular arrhythmias. Conversely, in the end stage, ICD therapy, even combined with external defibrillation, failed in resuscitating sudden cardiac death. One reason might be the routine administration of norepinephrine and epinephrine which enhanced CAS and myocardial ischemia.^[8,9] Zhang and coworkers^[9] reported two cases of diffuse CAS aggravated by intravenous epinephrine and dopamine during cardiorespiratory resuscitation and immediately reversed by intracoronary injection of nitroglycerin. Another reason might be rupture of undiscovered vulnerable plaques caused by CAS that resulted in continued myocardial ischemia and myocardial infarction. Bil and coworkers^[10] not only applied intravascular ultrasound to clearly reveal vulnerable plaques in a patient with CAS, but also intelligently used a bioresorbable vascular scaffold to fix the problem of plaque sealing.

4. Conclusion

For the sudden cardiac death related to lethal ventricular arrhythmias caused by CAS, ICD therapy is an efficient secondary prevention base on administrating coronary vasodilators. Furthermore, administration of epinephrine should be avoided during cardiorespiratory resuscitation of sudden cardiac death caused by CAS.

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