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Case Report

Coronary Artery Bypass Grafting for Refractory Ventricular fibrillation after the Release of the Aortic Cross-Clamp in Patients Undergoing Aortic Valve Replacement: A Case Report

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Abstract

Background: Ventricular fibrillation (VF) is a known complication after the release of the aortic cross-clamp (ACC) during cardiopulmonary bypass (CPB) surgery. Various factors contribute to persistent refractory VF, making its management challenging. This case report describes the successful treatment of postoperative refractory VF by coronary artery bypass grafting (CABG) in a patient undergoing aortic valve replacement (AVR) with ACC release. **Case Presentation**: A 52-year-old woman with a history of hypertension and ischemic cerebral infarction presented with symptoms of chest tightness, dyspnoea and palpitations. She underwent a modified maze procedure of radiofrequency ablation, mitral repair, left atrial appendage closure and mechanical AVR under a CPB procedure. Following the ACC release, the patient experienced recurrent VF that was unresponsive to standard interventions such as lidocaine, amiodarone and direct current shocks. The suspicion of right coronary artery (RCA) insufficiency led to the decision to perform CABG using the great saphenous vein. After the CABG procedure, the patient's heart rhythm gradually returned to sinus rhythm, and she had an uneventful recovery. **Discussion and Conclusions**: Refractory VF after the ACC release can pose diagnostic and treatment challenges. In this case, RCA insufficiency was suspected as the cause of refractory VF. The absence of blood flow in the proximal RCA and the presence of distal blood return during cardiac surface exploration supported this suspicion. Coronary artery bypass grafting using the great saphenous vein successfully restored normal sinus rhythm, confirming the diagnosis. This case emphasises the importance of considering coronary artery stenosis or occlusion as a potential cause of refractory VF after the ACC release during CPB, with CABG serving as a viable alternative treatment.

Keywords

ventricular fibrillation; aortic cross-clamp release; coronary artery bypass grafting; aortic valve replacement; refractory arrhythmias

Background

The incidence of ventricular fibrillation (VF) after aortic cross-clamp (ACC) release during cardiopulmonary bypass (CPB) surgery is about 10%–80% [1]. There are various mechanisms for the occurrence of persistent refractory VF after the ACC release, including intraoperative surgical trauma, reperfusion injury, ischemia injury, inadequate coronary exhaust, mechanical stimulation of the left ventricular (LV) drainage catheter, inadequate myocardial protection, insufficient supply of cardioplegic solution, electrolyte and metabolic disorders and uncorrected hypothermia [2]. Although direct current (DC) shock has been the standard treatment for ventricular fibrillation, it may also aggravate myocardial damage and adversely affect cardiac function, especially when refractory VF occurs, and it may even be difficult to be weaned from CPB after multiple DC shocks. High-dose injection of the sodium channel blocker lidocaine, a class IB antiarrhythmic agent, reduces the incidence of VF during reperfusion after the release of the ACC in patients undergoing cardiac surgery [3]. Therefore, lidocaine has traditionally been used as an initial antiarrhythmic agent for VF that is resistant to cardioversion during CPB. Amiodarone, a multichannel blocker, slows ventricular conduction and heart rate by blocking sodium channels, blocks atrioventricular nodal conduction by blocking β-adrenergic receptors and calcium channels and prolongs atrioventricular repolarisation by inhibiting potassium channels. Therefore, administration of amiodarone before ischemia reduces the incidence of VF induced by reperfusion [4]. Nifekalant is a potassium channel blocker available only in Japan and is often used in place of amiodarone to suppress signs of life-threatening arrhythmias

such as VF and tachycardia. The advantage of nifekalant is that it is a pure potassium channel blocker, so it does not cause inotropic effects and has a shorter half-life than amiodarone [5]. In addition, warm blood perfusion is also a common method for VF after releasing the ACC. However, refractory VF remains a challenge for diagnosis and treatment. Here, the authors report a case of a patient who underwent an ACC procedure during aortic valve replacement (AVR) and received coronary artery bypass grafting (CABG) for the treatment of postoperative refractory VF.

Case Presentation

The patient, a 52-year-old woman, recently developed chest tightness and shortness of breath with palpitations after activity without obvious inducement, and then gradually developed symptoms of holding up at night and orthopnoea. She had a previous history of hypertension, ischemic cerebral infarction and tobacco use. Auscultation on ward rounds suggested rapid atrial fibrillation, but an electrocardiogram suggested an atrial flutter (Fig. 1). On physical examination, the patient's pulse was 72 bpm, the heart rate was 80 bpm, the heart rhythm was irregular and a diastolic water-spitting murmur could be heard in the aortic valve auscultation area. Transthoracic echocardiography showed that the ascending aorta was dilated to a maximum width of 4.1 cm; the LA dimension was 4.2 cm. The LV dimension was 5.3 cm at diastole. The thickness of the LV posterior wall was 0.9 cm, the ejection fraction was 56%, the echo of the aortic valve was enhanced and thickened and the AV area was 1.6 cm^2. The reflux area was 10.2 cm^2 in the AV, 6.1 cm^2 in the mitral valve and 3.0 cm^2 in the tricuspid valve. Coronary angiography showed no significant coronary disease. Preoperative coronary computed tomography angiography (CTA) showed 20%–30% proximal obstruction in both the left anterior descending and right coronary arteries and 10% proximal obstruction at the left circumflex artery. Electrolyte disturbance and an acid-base imbalance can easily occur in the perioperative period and are also a potential cause of ventricular arrhythmia. Blood gas tests were performed on patients immediately after entering the operating room, before relapse and after cardiopulmonary bypass. The blood gas analysis results are shown in **Supplementary Table 1**. The results indicated that the pH changed little in the three time periods, and PO_2 increased significantly after cardiac relapse. The content of HCO_3 decreased gradually.

The patient received a modified maze procedure of radiofrequency ablation, mitral repair, left atrial appendage closure and mechanical AVR successively under general anaesthesia and CPB. The patient's blood gas was normal at the start of surgery, the first cardioversion and the end of the cardiopulmonary bypass. Intraoperative temperature control was regulated by experienced CPB physicians.

First, the great cryptic vein bridge was prepared. From the left medial ankle of the lower limb along the great saphenous vein to the middle and upper leg, the blunt-free great saphenous vein was carefully ligated and cut off each branch of the great saphenous vein. 20 cm section of this vein was removed and placed in heparin saline for later use. The lower leg incision was sutured layer by layer and thereafter wrapped with pressure. Subsequently, a great saphenous vein bridge anastomosis was performed. After the lumen was cut with a small sharp knife, there was no blood in its proximal end and a small amount of blood back in the distal end. We hypothesize that this is due to plaque in the right coronary artery or calcified debris from the aortic valve blocking near the midsection, which could explain the recurrent ventricular fibrillation. We chose to enlarge the incision to the distal and proximal ends with a length of 5 mm and measured the distal patency aperture greater than 1.5 mm through the incision with a coronary artery probe to solve the blood flow problem, which could explain the phenomenon of repeated ventricular fibrillation. The incision was extended to the distal and proximal ends, with a length of 5 mm. The coronary artery probe was used to measure the distal patency diameter greater than 1.5 mm. The great saphenous vein was aligned with the coronary anastomosis and anastomosed continuously with a 7-0 Prolene™ double-headed line. After completing the anastomosis, it was checked to ensure no blood leakage. The aortic wall was clamped with aortic lateral wall forceps, and a hole with a diameter of 4 mm was made in the aortic lateral wall with a hole punch. The proximal ends of the right coronary great saphenous vein bridge and the aortic lateral wall were sutured continuously with a 6-0 Prolene line. After the above anastomosis was completed, the ascending aorta was opened, and 5–6 holes were inserted in the great saphenous vein bridge with a 7-0 Prolene needle to discharge the air accumulation in the vascular bridge. Then, the vascular bridge was opened without damage to the vascular clamp, and the heart rate gradually returned to a sinus rhythm, with a rate of 110 beats/min. Vascular bridge perfusion was good, the coronary flow was 32 mL/min, the perfusion index was 0.8 and VF did not recur.

Following the intracardiac operation, the ACC was released to restore autoperfusion. The spontaneous recovery of heartbeat was poor and recurrent VF occurred. Warm perfusion of the aortic root and DC shocks restored sinus rhythm, but the heart rate was slow. Epinephrine was used to increase the heart rate, but the effect was not obvious. A temporary pacing lead was placed on the surface of the right ventricle to prevent a slow postoperative heart rate. Epinephrine and dopamine were used to support cardiac function. The VF was found to be resistant to lidocaine, amiodarone and DC shocks. The position of the aortic valve and the ostium of the coronary artery were explored after cardiac re-arrest, but no errors were noted. After re-releasing the ACC, VF recurred despite multiple

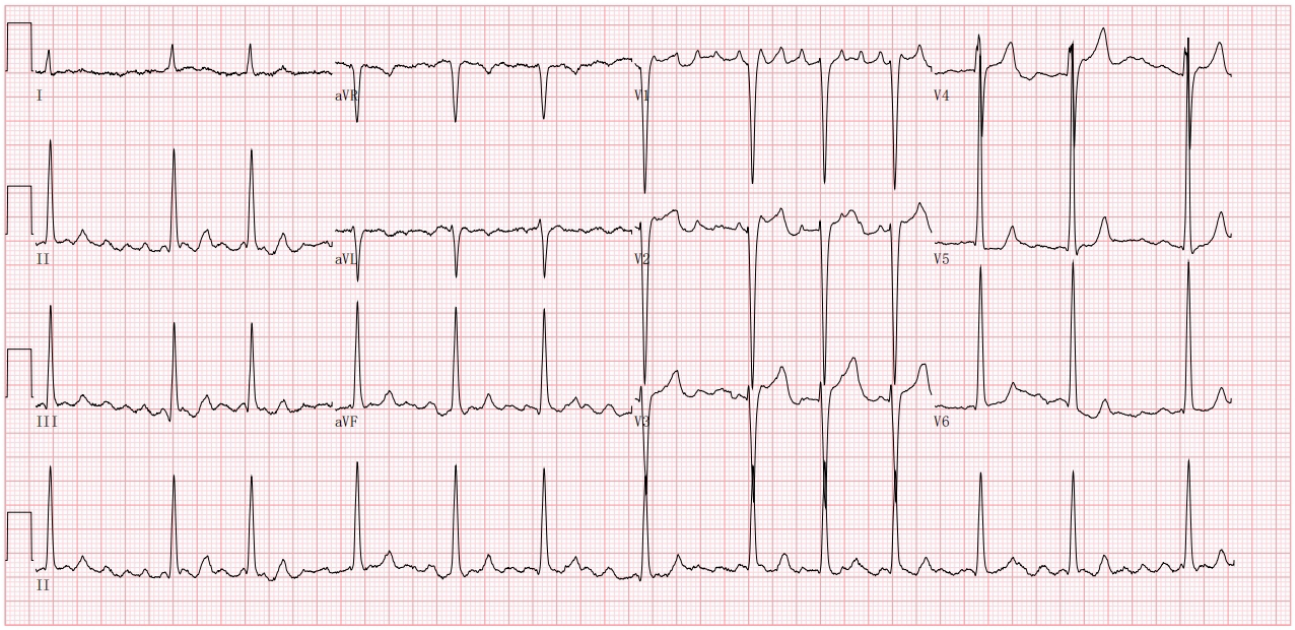

Fig. 1. Electrocardiogram on admission, showing ectopic rhythm, atrial flutter, poor R-wave increase in V1–V3 leads, left atrial hypertrophy and ST-T changes.

DC shocks (30 J). Since most of the nourishing arteries of the atrioventricular node start from the right coronary artery (RCA) at the atrioventricular junction, it was suspected that the refractory VF resulted from the RCA insufficiency. After communicating with the patient's family members, the great saphenous vein was used for CABG. After the lumen of the RCA was dissected, there was no blood in the proximal part and a small amount of blood return in the distal part. After the anastomosis, the ACC was released, and the heartbeat recovered well and gradually returned to sinus rhythm without VF. The patient had an uneventful postoperative recovery and was discharged on the tenth postoperative day.

Discussion and Conclusions

In this case, the patient had refractory VF after the ACC release, and there was still no cardioversion following the application of lidocaine, amiodarone and multiple DC shocks. Warm blood perfusion and cardiac surface exploration ruled out the possibility of an air embolism, and the possibility of artificial valve blocking ostia of the coronary artery was also ruled out. Because most of the blood supply of the atrioventricular node (92.3%) originates from the RCA at the atrioventricular junction, the RCA lesions are prone to arrhythmia according to clinical experience. Although preoperative coronary CTA reported an eccentric low-density shadow in the proximal segment of the RCA, and the corresponding lumen stenosis was 20%–30%, the authors strongly suspected that the VF had been caused by RCA insufficiency.

During the operation, after the lumen of the RCA was dissected, there was no blood in the proximal part and a small amount of blood return in the distal part, which was consistent with the authors' clinical judgement. It was thought that the plaque in the proximal segment of the RCA had been shed or the calcified debris of the AV had fallen into the RCA and blocked the proximal and/or middle segment of the RCA due to improper procedure, resulting in ischemia in the right coronary supply area; this could decrease the resting threshold of cardiomyocytes, increase extracellular potassium and decrease ATP-dependent potassium currents. These changes result in an 'injury current' that has been shown to increase abnormal automaticity, and the resulting rapid activation can aid the initiation of VF [6]. The primary treatment for VF caused by acute ischemia is coronary revascularisation. The authors performed CABG on the RCA using the great saphenous vein. After the anastomosis, the patient gradually returned to normal sinus rhythm without VF, which further supported the above decision.

Electrical storm syndrome is a life-threatening condition related to incessant ventricular arrhythmias. Urgent coronary angiography and revascularisation should be part of the management of patients experiencing electrical storms, as well as antiarrhythmic drug therapy and/or ablation of ventricular tachycardia. Acute myocardial ischemia can lead to ventricular fibrillation, and clinical observation has found that right coronary disease is prone to arrhythmia. Although the patient underwent a coronary CTA examination before surgery, there was no obvious stenosis in the right crown. However, the authors strongly suspect that the right coronary artery blood supply was involved during

the operation. After cutting the lumen of the right coronary artery trunk with a small knife, no blood was seen at the proximal end and a small amount of blood returned at the distal end, which confirmed the above conjecture. However, after the right coronary artery blood supply was restored, intractable VF disappeared. Based on this outcome, there is a causal relationship. Therefore, despite the exclusion of severe coronary stenosis by preoperative coronary angiography or CTA, the possibility of coronary artery severe stenosis or occlusion should be considered for refractory VF after ACC release during CPB, and CABG is an alternative method. The limitation of our study is that we only judged patients with arrhythmia due to RCA lesions based on clinical experience and did not further verify whether VF was caused by RCA insufficiency. What's more, we have checked the content of the article (**Supplementary Table 2**).

Availability of Data and Materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

XY has given substantial contributions to the conception or the design of the manuscript, DM to acquisition, analysis and interpretation of the data. All authors have participated to drafting the manuscript, MQ revised it critically. In addition, DM performed the operation as the surgeon and MQ as the assistant. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Research Ethics Committee of the First Hospital of Jilin University (ID: 2023-056), and informed consent was obtained from participant. All methods were carried out in accordance with relevant guidelines and regulations.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.59958/hsf.6713.

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