

# Malignant Ventricular Arrhythmias After Surgery for Uncomplicated Congenital Atrial Septal Defect-Case Report

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## Case report

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# Abstract

## Background:

Malignant ventricular arrhythmias (MVA) occurring subsequent to a repair of uncomplicated congenital heart disease is scarcely described in literature.

## Case presentation:

One adult patient following congenital atrial septal defect (ASD) repair underwent immediate postoperative refractory MAV and ventricular fibrillation. The recurrent episodes of shocks cannot be suppressed by drugs. Emergent re-exploration was performed and repeated closure of ASD and DeVega's annuloplasty were completed. The patient had uneventful recovery and no occurrence of arrhythmia.

**Conclusion:** Malignant ventricular arrhythmias are rare and should never be overemphasized even during the repair of uncomplicated congenital heart defect. Re-exploration should be taken into consideration when MVA occurred in the early stage postoperatively.

## Background

Malignant ventricular arrhythmia (MVA) early after cardiac surgery is an uncommon arrhythmic complication but has a negative impact on mortality[1]. Some literature documented MVA and sudden death in complicated congenital heart disease such as tetralogy of Fallot[2, 3]. However, MVA subsequent to a repair of uncomplicated congenital heart disease is scarcely described. In this case, we reported one adult patient following congenital atrial septal defect (ASD) repair which underwent immediate postoperative refractory MVA and ventricular fibrillation.

## Case Presentation

A 49-year-old woman admitted to our hospital had been suffering from effort-induced chest tightness and shortness of breath for ten years, with the diagnosis of congenital ASD plus mild tricuspid regurgitation. Physical examination disclosed no abnormalities. Routine pre-operative assessment demonstrated her normal sinus rhythm, blood biochemical test and complete blood count analysis. The patient was in NYHA functional class I. Coronary angiography revealed maximal diameter stenosis was 30% in left anterior descending artery, and right coronary artery was normal. The patient denied history of arrhythmia. The arterial blood gas analysis on the first day of admission exhibited normal lung function parameters.

After a four-day preparation, the patient received a repair of atrial septal defect plus tricuspid valvuloplasty with cardiopulmonary bypass (CPB). The operation was conducted via a midline sternotomy. The ascending aorta and both vena cavae were cannulated directly. CPB under mild hypothermia (34 °C) was instituted. The heart was arrested by antegrade cold blood cardioplegia. The

right atrium was opened. There was a superior sinus venosus large ASD 2.0 × 2.5 cm in size. The defect was closed with a 3.0 cm dacron patch with continuous 5/0 prolene sutures. Tricuspid valvuloplasty (TVP) was performed with DeVega's annuloplasty. The heart restores normal sinus rhythm after removal of cross-clamp. The patient weaned from CPB smoothly. The duration of CPB was 63 minutes and cross-clamp was 17 minutes. The patient was transferred to the intensive care unit (ICU) with intubation without any unusual sign during procedure of closing. At the admission of ICU, blood pressure (BP) were stable (102/63 mmHg) and electrocardiogram revealed normal sinus rhythm. However, just within one hour, unanticipated ventricular tachycardias and sequent ventricular fibrillation (VF) occurred (Fig. 1). Then the patient was promptly resuscitated with defibrillation and antiarrhythmic agents (amiodarone, lidocaine, and epinephrine). Emergent bedside echocardiography dictated normal left ventricular ejection fraction without pericardial tamponade. Arterial blood gases analysis and laboratory investigations were unremarkable when MVA occurred. After an initial uneventful recovery, the patient had recurrent episodes of ventricular tachycardias and VF requiring multiple defibrillation shocks. The greatest of these is that, there is no obvious ST-segment and T-wave (ST-T) changes during the interval between attacks which may demonstrate coronary ischemia caused by coronary stenosis or intracoronary air embolism.

After a comprehensive analysis and discussion, the heart team decided to conduct an emergent re-exploration. All of the previous patch and sutures were replaced and the repeated closure of ASD and DeVega's annuloplasty were completed by another senior cardiac surgeon. The patient restores normal sinus rhythm after removal of cross-clamp. The second duration of CPB was 49 minutes and cross-clamp was 18 minutes. The patient weaned from CPB smoothly with temporary pacemaker implantation. About 8 hours after re-exploration, the tracheal tube was removed after the patient had become fully awake. On the second day postoperatively, the patient was transferred to the ward. No major arrhythmias or adverse clinical events were recorded during the postoperative ICU and hospital stay. Furthermore, the postoperative echocardiogram and various examinations before discharge were within normal limits. During the six-month following-up, the patient had an uneventful recovery and no occurrence of arrhythmia.

## Discussion

Sudden cardiac death (SCD) mostly resulting from ventricular arrhythmia remains a cause of mortality in 19–30% of adults with congenital heart defects[4]. Prime risk factors of MVA are summarized in Table 1. The precise mechanisms remain debatable. Underlying mechanisms involve: 1) abnormal or enhanced automaticity in ventricular myocytes and Purkinje fibers, 2) triggered activity induced by early or late afterdepolarizations, 3) the reentry around a scar and functional block, 4) the reentry due to heterogeneity of ventricular repolarization [5].

Table 1  
Factors Related to MVA in Cardiac Surgery

<b>perioperative myocardial infarction</b>
low cardiac output
preoperative ventricular arrhythmia
sympathomimetic drugs (catecholamines methamphetamine)
metabolic and electrolytes disorders (hypokalemia)
ischemic heart disease
age
specific family history
structural heart disease
cardiac channelopathy
long QT syndrome
Brugada syndrome
cardiac tamponade
cardiopulmonary bypass (trauma from cannulation, cross-clamp times, cardioplegia)
extreme hemodynamic fluctuation
coronary artery bypass graft
extensive scars and wide surgical sutures
heart transplant
MVA = malignant ventricular arrhythmia

In this case, the heart team decided to perform emergent re-exploration based on the following considerations. First, the recurrent episodes of ventricular tachycardias and VF cannot be suppressed by drugs. Emergent bedside echocardiography, blood gases analysis, electrocardiogram and laboratory investigations were unremarkable when MVA occurred, which helped to eliminate possibilities of cardiac tamponade, coronary stenosis or intracoronary air embolism and electrolyte imbalances. This was an otherwise healthy adult, with no history of medications, heart attack and severe coronary artery disease, and the implantation of intra-aortic balloon pump was not considered as the first therapy. Second, the MVA occurred so frequently in the early postoperative stage, which indicated a high correlation with the operation. Therefore, the heart team speculated that there were two possibilities: on the one hand, trauma from surgical sutures may be served as a potential source of MVA; on the other hand, the implementation of tricuspid valvuloplasty probably caused stenosis or occlusion of the right coronary artery during the

beating of the heart. To some extent, the fact that no MVA reoccurred after the re-exploration proved our assumption. Nonetheless, there is no definite evidence to explain the occurrence of MVA. Future research will shed more light on this issue.

## **Conclusions**

To the best of our knowledge, this is the first case in the literature. Our experience emphasized the possibility of manipulated injury when malignant ventricular arrhythmias occur in the early stage after uncomplicated congenital atrial septal defect repair. Prompt recognition and intervention are essential for this particular issue to avert potentially fatal consequences.

## **Abbreviations**

ASD

Atrial septal defect

ICU

Intensive care unit

MVA

Malignant ventricular arrhythmia

## **Declarations**

### **Ethics approval and consent to participate**

Ethics approval and consent for this publication were waived by Fuwai Ethics Committee.

### **Consent for Publication**

Written informed consent was gained from the patient for the publication of this article.

### **Availability of data and materials**

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

### **Competing interests**

The authors declare that they have no competing interests.

### **Funding**

none.

## Authors' contributions

Author Ying Liang undertook the main work of data collection, follow-up, analysis, and writing; Author Yulong Guan, revised and interpreted the manuscript, and will do the subsequent contact.

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Not applicable

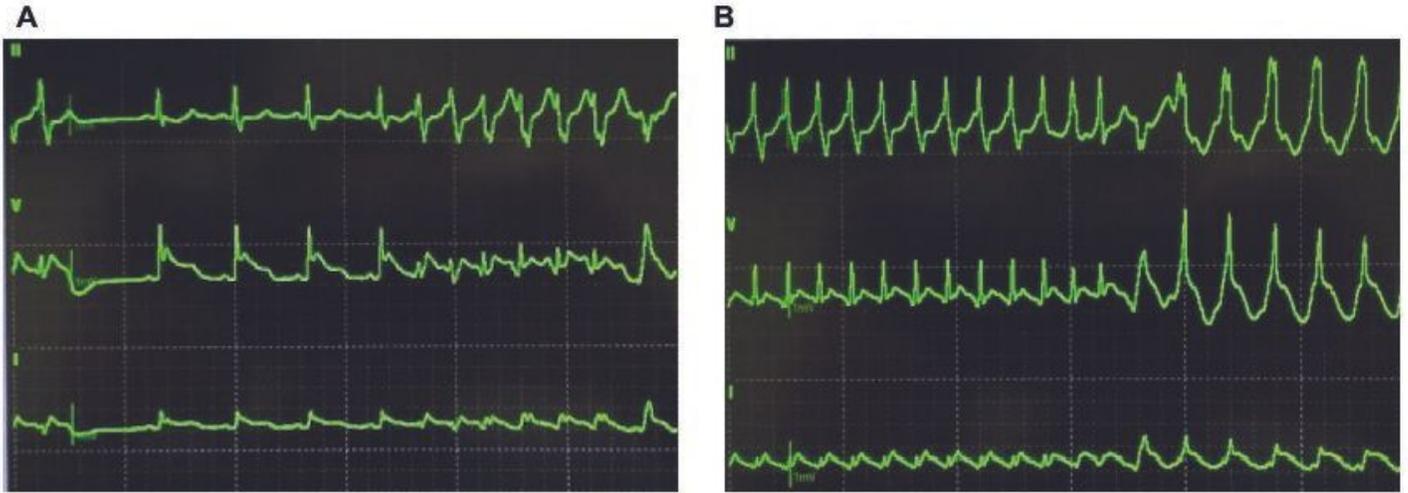
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## Figures



**Figure 1**

Electrocardiogram when MVA occurred Legend: A. ventricular tachycardia B. supraventricular tachycardia and ventricular tachycardia. MVA Malignant ventricular arrhythmia

## Supplementary Files

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