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Case Report

Anaesthetic Management of a Large Atrial Septal Defect with Severe Tuberculous Constrictive Pericarditis: A Case Report

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Abstract

The combination of constrictive pericarditis (CP) and atrial septal defect (ASD) is a rare medical condition. Surgical intervention is typically considered the superior treatment option for patients with this condition. In this report, we present a rare case where a patient presented both a large ASD and severe tuberculous CP. The role of anesthesia is crucial in surgical procedures involving this complex cardiac conditions. Factors such as hemodynamic stability, fluid management, and preservation of cardiac function must be carefully considered. The utilization of transesophageal echocardiography (TEE) proved highly advantageous in this case, as it guided the medical team through various phases of treatment. By closely monitoring cardiac function with TEE, changes and improvements be accurately evaluated over time. This case report discusses the anesthetic management issues of this complex disease.

Keywords

atrial septal defect; tuberculous constrictive pericarditis; transesophageal echocardiography

Introduction

The combination of constrictive pericarditis (CP) and atrial septal defect (ASD) is a rare occurrence. While there have been reports on the etiology, diagnosis, and treatment of this condition, little attention has been given to the surgical and anesthetic challenges associated with it [1–11]. The long-term confinement of the pericardium leads to myocardium disuse atrophy, fibrosis, decreased compliance, diastolic dysfunction, and poor tolerance to hemodynamic fluctuations. Stress response and inappropriate volume status can disrupt the balance of oxygen supply and demand in cardiomyocytes, potentially leading to arrhythmia, heart failure, and even cardiac arrest. Therefore, precise anesthetic management is crucial for patients. This study focuses on the anesthetic management of a patient with a large ASD and severe tuberculous CP, providing insights into the pathophysiology and anesthetic implications of this complex disease. The CARE checklist was used when writing this case report in **Supplementary Table 1**.

Case Report

A 43-year-old man weighing 53 kg presented to the hospital with gradually increasing fatigue, shortness of breath, and abdominal distension. During the physical examination, a systolic murmur was detected at the II–III intercostal space of the right sternal border. The patient had previously received furosemide orally in a neighboring hospital. Upon arrival at our hospital, the patient tested positive for T-cell spot test (T-SPOT) and tuberculous (TB) trust. Laboratory tests upon admission revealed elevated N-terminal-pro hormone brain natriuretic peptide levels at 507, normal total bilirubin levels at 21.8 µmol/L, aspartate aminotransferase levels at 41 U/L, and alanine aminotransferase levels at 16 U/L. Transthoracic echocardiography (TTE) showed enlargement of the right heart and slight reduction of the left heart, along with a large ASD and thickened pericardium with calcification on the posterior, right, and diaphragmatic sides (Fig. 1A). Computed tomography (CT) revealed calcification and thickening of the pericardium, as well as dilation of the vena cava (Fig. 1B). Prior to surgery, the patient was administered a first-line anti-tuberculosis regimen consisting of rifampicin 450 mg/day, isoniazid 300 mg/day, pyrazinamide 750 mg/day, and ethambutol 750 mg/day. Surgery was scheduled after two months of anti-TB treatment.

Standard monitoring was utilized in the operating theatre. The patient was conscious with vital signs including a SpO_2 of 94%, respiratory rate of 18 breaths per minute, heart rate of 80 beats per minute, and blood pressure of 102/64 mmHg. Prior to anesthesia, defibrillation pads were positioned anteroposteriorly, and the defibrillation device was prepared. Arterial lines were inserted into the right radial artery under local anesthesia. The patient was induced with incremental doses of sufentanyl 50 µg, midazolam 4 mg, and cisatracurium 20 mg. Once the drugs had

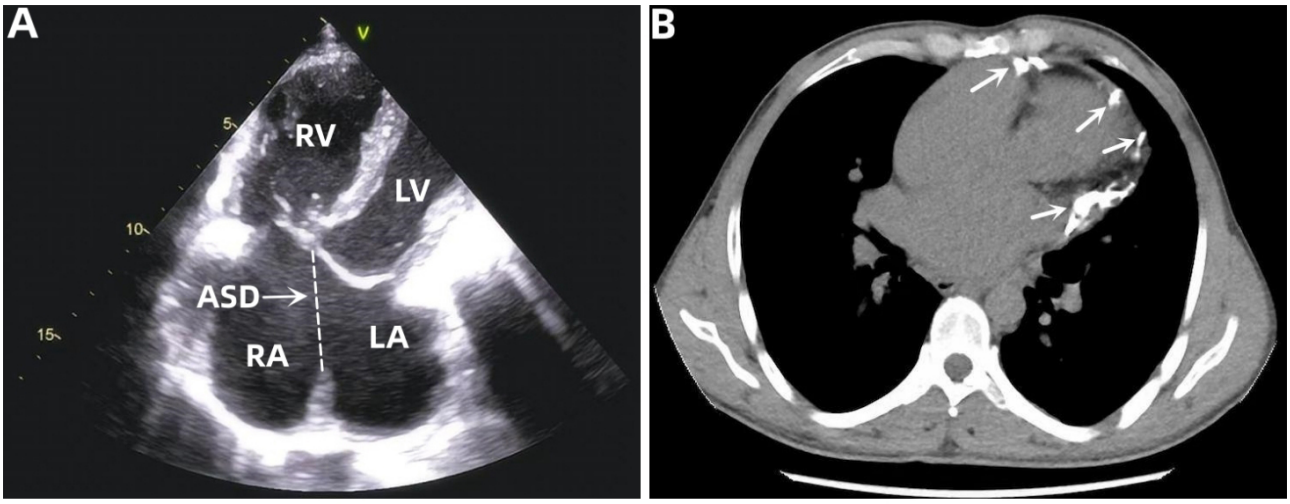

Fig. 1. Preoperative TTE and CT images of the patient. (A) TTE showed ASD and thickened pericardium. (B) CT showed pericardium was calcified (arrow) and thickened. RA, right atrium; RV, right ventricle; LV, left ventricle; LA, left atrium; TTE, transthoracic echocardiography; ASD, atrial septal defect; CT, computed tomography.

reached their full effect, a 7.5# endotracheal tube was inserted. During the anesthesia induction, the heart rate fluctuated between 70 and 80 beats per minute, while the systolic blood pressure ranged from 90 to 100 mmHg and the diastolic blood pressure remained within the range of 60 to 70 mmHg. Anesthesia was maintained using sevoflurane in an air/oxygen mixture, along with cisatracurium and sufentanyl. Central venous lines were placed in the right internal jugular vein, and a TEE probe was inserted to monitor cardiac function. The pericardium was found to be extensively thickened with calcification, and the ASD measured 4.4 cm on TEE. The pulmonary vein s/d ratio was 0.5, and the mitral doppler echocardiogram E/A ratio was 3.9 (>2) (Fig. 2), indicating severe diastolic dysfunction of the ventricle. The pericardium was dissected from the ascending aorta to the lateral and posterior wall of the left ventricle. Flutter occurred and the patient experienced a drop in blood pressure when the dissection reached the apex of the heart. Cardioversion was performed using 150 joules, successfully restoring the heart to sinus rhythm. The operation proceeded to address the pulmonary veins and arteries, as well as the right ventricle and right atrium. Cardiopulmonary bypass (CPB) was initiated after injecting 160 mg of heparin sodium. The posterior pericardium was dissected further, and a suitable-sized autologous pericardial slice was harvested to repair the ASD. However, ventricular fibrillation occurred when CPB was stopped due to a rapid increase in cardiac blood volume. Immediate defibrillation and restarting of CPB were performed. Right atrial drainage was placed, and blood was slowly pumped back into the patient under direct TEE vision to achieve the appropriate volume loading. Epinephrine dosage was adjusted under TEE guidance to optimize systolic function. Simultaneously, we assessed for residual intracardiac defects and shunts, observed an increase in pulmonary vein s/d, and an E/A ratio of 1.4 (Fig. 2). These indicators confirmed the success of the operation. CPB was successfully stopped, and the procedure was completed. The patient was under anesthesia for 8.8 hours, during which they received 500 mL of crystalloid solution, 500 mL of colloid solution, 400 mL of plasma, and had an autologous blood recovery volume of 300 mL. Additionally, the patient had a urine output of 3500 mL. The patient had a smooth recovery and was discharged on the 7th day. The postoperative pathological report confirmed the diagnosis. The patient was followed for 16 months post-surgery, and significant improvement in symptoms was observed with no recurrence of TB. This highlights the effectiveness of the surgical procedure and the anti-TB regimen administered prior to surgery.

Discussion

The pathophysiological changes in the combination of CP and ASD are complex and pose significant anaesthetic risks. This case report presents the first documented anaesthetic management of CP with ASD. Chronic CP is characterized by fibrosis and calcification of the pericardium, often resulting from infection, radiation injury, or tuberculosis. The constricted pericardium restricts the normal activity of both ventricles, impeding diastolic filling and obstructing blood return to the vena cava, leading to increased venous pressure [12]. When ASD is present concurrently, it exacerbates right heart failure due to early left-to-right shunting. The recommended approach in such cases involves pericardiectomy and repair of the atrial septal defect.

Anesthetic management of these patients poses significant challenges. The patient's anesthetic management can

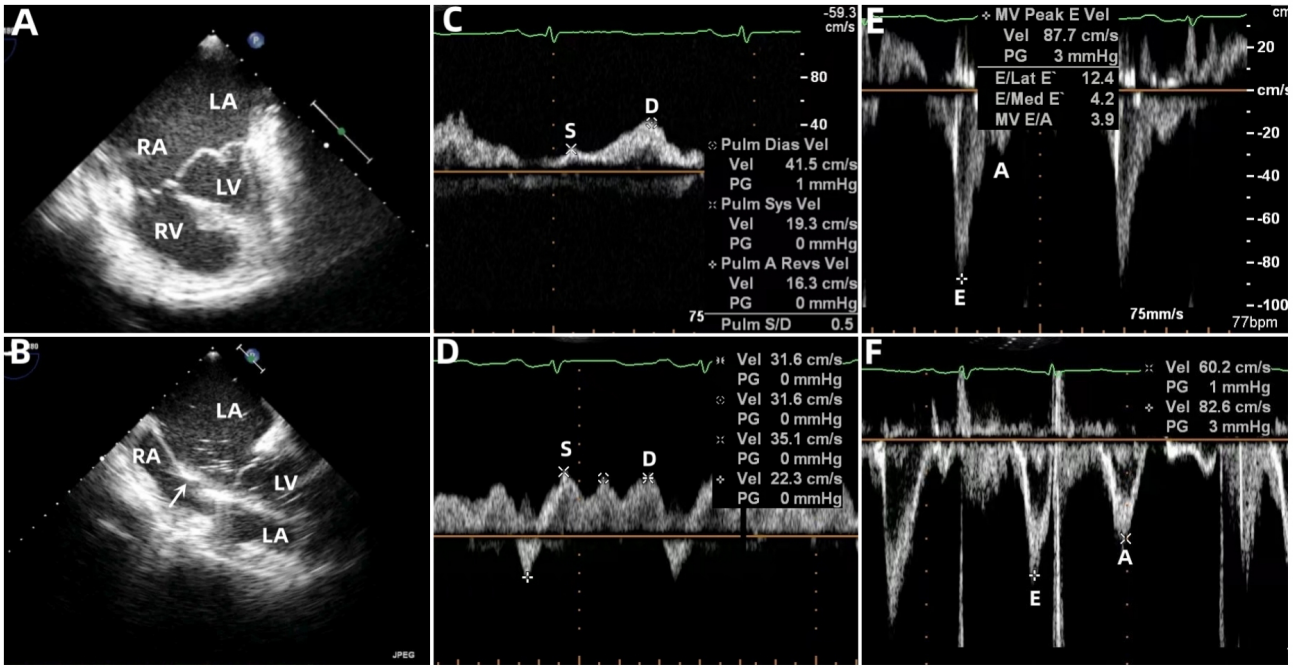

Fig. 2. Evaluation cardiac structure and function through TEE. (A) TEE revealed ASD and thickened pericardium. (B) TEE showed the postoperative atrial septum (arrow) and pericardium. (C) The preoperative pulmonary veins spectral Doppler. (D) The postoperative pulmonary veins spectral Doppler. (E) The preoperative mitral valve inflow spectral Doppler. (F) The postoperative mitral valve inflow spectral Doppler. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle; TEE, Transesophageal echocardiography; ASD, atrial septal defect.

be divided into three distinct phases, each with a specific focus. The first phase begins after anesthesia induction and continues until pericardial dissection. The second phase spans from after pericardial dissection to before atrial septal defect repair. Finally, the third phase starts after atrial septal defect repair and continues until the end of the surgery. During the first stage, the constricted pericardium limits diastolic filling of both ventricles, resulting in the hemodynamic paradox of low preload but high filling pressures [13]. Fluid therapy in these patients is particularly challenging. Excessive capacity can easily lead to pulmonary edema, while insufficient capacity can further compromise left ventricular filling and reduce stroke volume. Both central venous pressure (CVP) and pulmonary capillary wedge pressure are unreliable markers in these patients, as they overestimate volume status due to the significantly elevated pulmonary and systemic venous pressures [14]. Therefore, TEE is the most appropriate method to guide fluid management and monitor cardiac function. In the first stage, the s/d ratio of the pulmonary vein was 0.5, and the E/A ratio of the mitral doppler echocardiogram was 3.9 (>2), indicating severe diastolic dysfunction (Table 1). Left ventricular filling has been significantly restricted, and the left atrial contraction is unable to effectively pump blood into the left chamber. Consequently, a restrictive fluid strategy is implemented to control fluid intake. Biventricular diastolic dysfunction with elevated filling pressures results in a relatively fixed stroke volume, making maintenance of cardiac output dependent on increasing the heart rate. Therefore, it is necessary to maintain a fast heart rate of 70–80 beats per minute at this stage. During pericardial stripping, arrhythmias can frequently occur as a result of traction on the pericardium or stimulation caused by the use of an electric knife. However, intracardiac defibrillation is not effective in these cases due to the significant thickening and calcification of the pericardium. Therefore, it becomes essential to prepare defibrillation pads *in vitro* beforehand.

In the second stage, the main hemodynamic changes are attributed to a large ASD. The shunt volume of an ASD depends on the size of the defect, the pressure difference between the two atria, and the filling resistance of the two ventricles. Initially, blood is redirected from the left atrium to the right atrium due to the higher pressure in the left atrium, resulting in intracardiac shunting. This shunting causes increased volumes in the right atrium and right ventricle, leading to enlargement of the cardiac chambers in the right heart system. Consequently, it reduces the capacity of the left heart system and impairs systemic circulation perfusion. Removal of pericardial restriction further elevates the return blood volume, exacerbating the right ventricular volume load and increasing the risk of acute right heart failure. Therefore, proper fluid management is crucial, and utmost attention should be given to preserving right heart function.

Table 1. Assessment of diastolic function.

	Post-induction	Post-pericardial stripping	After repair of ASD
Peak E-wave velocity (cm/sec)	87.7	52.8	82.6
Peak A-wave velocity (cm/sec)	22.5	17.0	60.2
MV E/A ratio	3.9	3.1	1.4
PV S wave (cm/sec)	19.3	18.1	35.1
PV D wave (cm/sec)	41.5	31.6	31.6
PV S/D ratio	0.5	0.6	1.1

MV, mitral valve; PV, pulmonary vein.

In the third stage, the pericardium has been removed and the atrial septal defect has been repaired. The removal of the left-to-right shunt and mechanical restrictions resulted in a significant increase in left ventricular volume. However, patients with constrictive pericarditis exhibited varying degrees of myocardial cell atrophy and fibrosis, decreased compliance, and an inability to adapt to the sudden increase in volume. This imbalance between myocardial oxygen supply and demand can potentially lead to heart failure or malignant arrhythmias. Therefore, it is crucial to carefully maintain adequate preload under TEE guidance. Controlling fluid intake and using diuretics can help reduce volume overload. Research reports suggest that restrictive fluid infusion (2.68–7.46 mL/kg/h) is beneficial in reducing postoperative complications without increasing the risk of acute injury [15]. In this case, the patient received a continuous infusion of epinephrine at a rate of 0.05 µg/kg/min to increase myocardial contractility, and furosemide 10 mg was administered to reduce volume load.

Conclusion

In conclusion, large ASD with severe tuberculous CP is a rare condition that poses significant risks to the patient. Surgery is the most effective treatment option, although it presents numerous challenges. It is important to prioritize the preservation of the patient's heart function, with the guidance of TEE. Additionally, experienced anaesthetists and skilled surgeons are essential in minimizing the potential complications such as arrhythmias, heart failure, and heart rupture.

Availability of Data and Materials

The data of our case are available from the corresponding author on reasonable request.

Author Contributions

All authors contributed to the design and conceptualization of this case report. LL, MY, and HY were directly involved in the anaesthetic management of the patient. LL and MY were responsible for drafting the manuscript, while HY provided supervision for the writing process. 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

The case report was approved by the Ethics Committee of West China Hospital, Sichuan University (approval number 2023-2082), and written informed consent for publication has been obtained from the patient.

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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.5833.

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