Catastrophic cardiac arrest caused by acute pulmonary hypertension after removal of giant left atrial thrombus

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Title page

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Abstract

This is a report of severe mitral valve stenosis with giant left atrium thrombosis. Which gradually experienced difficulty in ventilation and persistent refractory hypotension, followed by ventricular fibrillation during the process of chest closure after cardiopulmonary bypass. TEE helps diagnose acute pulmonary hypertension. We present the case, although TEE plays an important role in the diagnosis and the decision of using mechanical devices, a Swan-Ganz catheter would be an effective hemodynamic monitoring device and can be used in conjunction with TEE in cardiac surgery.

KEYWORDS
mitral valve stenosis, giant left atrium thrombosis, pulmonary hypertension,

1 | INTRODUCTION

Mitral stenosis is associated in up to 17% with atrial thrombus. The changed anatomy and enlarged volume of left atrium, abnormal blood flow into the atrium, abnormal atrial contraction, decreased mitral valve area (MVA), and impaired endothelial function are the key determinants of increased risk of thrombus formation in these patients. Pulmonary arterial hypertension is often found in symptomatic patients with mitral stenosis. Mechanical factors, vascular remodeling, and endothelial changes mediate such hypertension, and neurohormones might affect the course. Occasionally, patients with pulmonary arterial hypertension who are hyper-responders present with structural changes in the pulmonary bed, and their hypertension is disproportionately severe and might incompletely resolve after the relief of mitral stenosis. We introduce a patient who underwent mitral valve replacement and thrombectomy, including tricuspid valve annuloplasty. During the process of chest closure, the patient gradually experienced difficulty in ventilation and persistent refractory hypotension, followed by ventricular fibrillation. VA-ECMO treatment is performed for patients with PH that are difficult to correct.

2 | CASE REPORT

A 53-year-old female patient was admitted to the hospital due to a history of progressive shortness of breath, progressive dyspnea, and more than 30 years. Two months before admission her activity became limited due to increasing shortness of breath, repeated coughing, and recurrent paroxysmal nocturnal dyspnea. The patient did not receive warfarin anticoagulation therapy and did not have any comorbidities.

Transthoracic echocardiography showed atrial fibrillation with enlarged atrial. Evidence of mitral restenosis (MVA 0.8 cm²) was seen. The tricuspid valve was moderately incompetent with an estimated pulmonary artery systolic pressure (PASP) of 75 mmHg. Preoperative transthoracic echocardiography (TEE) confirmed mitral stenosis and revealed an expanded and homogenous mass in the left atrium with an extent of 8.5 cm*9 cm (Figure 1A). Additional CT showed this mass filling the lateral and superior wall of the left atrium with an extent of 9cm*13cm. TEE confirmed regional extent including the atrial appendage (Figure 1B) and detected mobile parts with abundant smoke in the left atrium. In conclusion, the left atrial mass could be identified as a giant thrombus. Laboratory investigations revealed a low hemoglobin level (11.2 g/dL), low platelets count with mild hypoproteinemia.

Surgery was performed using standard CPB with moderate hypothermia and antegrade blood cardioplegia. A giant thrombus was difficult to remove because the adhesion between thrombus and the left atrium was very serious. Therefore, the thrombus is chopped up and taken out carefully. The mitral valve was replaced with St. Jude Medical valve. The tricuspid valve was repaired using Devag’s annuloplasty. Minimal inotropic support was used during weaning from bypass.

Sudden hypotension combined with poor ventilation during the process of chest closure, the administration of blood pressure drugs and inotropic drugs was ineffective, and then ventricular fibrillation occurred. Immediately perform chest heart compression and open the chest to prepare for CPB again. At the same time, he was given electric defibrillation 200J, intravenous epinephrine, and intravenous methylprednisolone 80mg. Fifteen minutes later, CPB was established again, and the patient returned to spontaneous circulation. TEE monitors that the artificial valve function is normal, the coronary blood flow is normal, and the left ventricular systolic function is normal. The PASP is directly measured as 90mmHg. After improving the internal environment and adjusting the ventilation and vasoactive drug dosage, the PASP dropped to 72mmHg. Then try to wean from CPB again smoothly guided by TEE. But during the separation of the second time, it was found that the PASP rose rapidly from 72 to 122mmHg. TEE monitoring through the left ventricular short-axis view revealed that the eccentric index of the left ventricle was greater than 1.1 during systole, showing a “D” sign (Figure 2A). The view of the right ventricular inflow and outflow view showed that the pulmonary artery was dilated (the diameter over 3cm) (Figure 2B). Meanwhile, the pressure of the systemic circulation was difficult to maintain. Then CPB was performed for the third time. Considering the patient’s severe PH which may be difficult to separate from CPB, our team decided to perform VA-ECMO
treatment. Unfortunately, the patient died of heart failure on the third day after surgery.

3 | DISCUSSION

A significant reduction was witnessed in the prevalence, incidence, and associated fatalities of rheumatic heart disease (RHD) in high-income countries in the late 20th century.\(^3\) At present, the patients with mitral valve stenosis in our center are still dominated by rheumatic diseases. Mitral stenosis remains an important cause of morbidity despite the ease with which it can be diagnosed and treated. The onset of atrial fibrillation, which is often caused by atrial inflammation and remodeling, is a pivotal moment in mitral stenosis. Atrial fibrillation occurs in 40–75% of patients who are symptomatic for mitral stenosis, precipitates such symptoms, greatly increases the risk of systemic embolization, and reduces cardiac output and exercise capacity.\(^4,5\) If the left atrial appendage thrombosis and there is no regular anticoagulation treatment, the embolus is very likely to cause cerebral infarction or other organ embolism.

Long-standing mitral valve disease is associated with enlargement of the left atrium as a compensatory mechanism due to increasing intracavitary pressure and volume. Such an enlargement is beneficial as it reduces pulmonary congestion, thus it protects the lung from PH and edema.\(^6\) However, with the gradual increase in left atrial pressure an associated increase in pulmonary venous pressure will eventually occur.\(^7\) As the course of the disease progresses and pressure conducts, pulmonary arteries develop vascular endothelial dysfunction and appear reactive vasoconstriction, neuroendocrine and inflammatory cell activation, NO reduction, increased endothelin secretion, and decreased vasodilation effect of BNP, and other pathophysiological changes that promote pulmonary vascular remodeling. The structure, leading to the occurrence of PH, further restricts the ability of the right ventricle to transfer blood to the pulmonary artery, causing right heart overload and right heart failure. At the same time, the left ventricular filling will be damaged again through ventricular interdependence.

PH-LHD (left heart disease) can be classified into two main subtypes: isolated postcapillary PH (IpcPH) and combined post and precapillary PH (CpcPH).\(^8\) When pulmonary vascular resistance (PVR) is \(< 3\) Wood units, PH is considered to be IpcPH, reflecting elevated left atrial pressure without intrinsic pulmonary vasculopathy. If the PVR is \(\geq 3\) Wood units, the elevated left atrial pressure is considered to be CpcPH which means that irreversible damage has occurred to the pulmonary vessels. Although the PCWP has been reduced to a certain extent after mitral valve replacement in this patient, the PVR has not been greatly reduced because of the pathological changes in the pulmonary vessel itself. During the process of chest closure, it can cause an increasing in chest pressure, further increasing pulmonary artery pressure and PVR. This leads to an increase in right ventricular pressure and an increase in tricuspid regurgitation. The increase in right ventricular pressure squeezes the left ventricle, further reducing the filling of the left ventricle. Meanwhile, the strong contraction of pulmonary blood vessels will lead to an increase in pulmonary ventilation resistance, increase the shunt in the lungs, and reduce the patient’s oxygenation. Therefore, the patient has circulatory instability and poor ventilation, which eventually causes circulatory shock and ventricular fibrillation.

TEE plays an important role in the monitoring of PH. But the disadvantage of TEE is the lack of continuity of monitoring. For patients with severe mitral stenosis and severe PH, should the Swan-Ganz catheter be placed routinely? The monitoring of pulmonary artery pressure and pulmonary venous pressure by Swan-Ganz catheter can guide the management of perioperative circulation and respiration, especially for early detection of PH. Few case reports describe percutaneous Swan-Ganz catheter removal, in which both chordae and papillary muscle ruptures have been observed.\(^9,10\) A Swan-Ganz catheter remains an effective hemodynamic monitoring device and can be used in conjunction with TEE, in cardiac surgery.\(^11\)

4 | CONCLUSIONS

We introduce a case of severe mitral valve stenosis with giant left atrium thrombosis performing cardiac surgery. The patient gradually experienced difficulty in ventilation and persistent refractory hypotension, followed by ventricular fibrillation. Although TEE plays an important role in the diagnosis and the decision of using mechanical devices. A Swan-Ganz catheter would be an effective hemodynamic monitoring device
and can be used in conjunction with TEE in cardiac surgery.

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**CONFLICT OF INTEREST STATEMENT**

None to declare.

**DATA AVAILABILITY STATEMENT**

The authors confirm that the data supporting the findings of this study are available within the article.

**ETHICS APPROVAL AND PATIENT CONSENT STATEMENT**

Written consent for the publication of this case report was obtained from the patient. Approval for a case report by the institutional ethics committee is not required.

**REFERENCES**


