

Case Reports

Effective Thrombectomy for Biventricular Thrombosis With an Endoscope via the Aortic and Tricuspid Heart Valves

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Abstract

The simultaneous formation of thrombi in the left and right ventricles is extremely rare, and this condition's clinical characteristics and prognosis remain unknown. Therefore, treatment methods are still controversial. This case highlights the potential for endoscopic thrombectomy as a viable option for the treatment of giant floating biventricular thrombi in a patient with restricted cardiac function. This approach via the aortic and tricuspid heart valves allowed for effective and complete removal of the thrombi, thereby preventing further embolic complications.

Keywords: Thrombosis; thrombectomy; low cardiac output; cardiac surgical procedures; endoscopes

Case Report

Presentation and Physical Examination

A 56-year-old woman who had self-discontinued treatment for hypertension 1 year previously presented at the hospital; she had been experiencing dyspnea on exertion and bilateral lower leg edema for 2 weeks. The patient was admitted for treatment. Transthoracic echocardiography (TTE) revealed cardiac dysfunction, and large floating thrombi were observed in the apex of both ventricles. At hospital admission, the woman's height was 148.0 cm, her body weight was 71.0 kg, and oxygen saturation as measured by pulse oximetry was 94% on room air. Pulmonary sounds were clear, with coarse crackles, and heart sounds were normal but with a systolic murmur. Bilateral lower leg edema was also present. Plain chest radiography in the sitting position revealed a cardiothoracic ratio of 65.8% and bilateral pleural effusion (Fig. 1). Electrocardiography showed sinus rhythm, with a heart rate of 107/min and left ventricular (LV) hypertrophy.



Fig. 1 Chest x-ray with the patient in a sitting position shows cardiomegaly and bilateral pleural effusion.

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Blood chemistry tests (with normal ranges in parentheses) showed a white blood cell count of $7.93 (3.30-8.60) \times 10^9/L$, hemoglobin level of $106.00 (116.00-148.00) g/L$, platelet count of $3.79 (1.58-3.48) \times 10^9/L$, creatine kinase level of $1.52 (0.68-2.56) \mu kat/L$, lactate dehydrogenase level of $6.65 (2.07-3.71) \mu kat/L$, C-reactive protein level of $2.20 (0.00-1.40) mg/L$, dimerized plasmin fragment D level of $62.43 (0.00-5.48) nmol/L$, and N-terminal pro-brain natriuretic peptide level of $821.00 (0.00-14.80) pmol/L$. Activated protein-C of 105% (70%-140%) and protein-S antigen of 91.0% (60%-150%) showed normal ratios. Transthoracic echocardiography showed severe akinesis of the LV inferior wall and apex; LV internal diastolic and systolic dimensions of 59 mm and 52 mm, respectively; left atrial diameter of 44 mm; and LV ejection fraction (LVEF) of 25%. Mitral valve regurgitation was mild, with no prolapse, and 2 mobile masses within the LV and right ventricular (RV) cavities were present (Fig. 2). The LV thrombus, which was 41×26 mm in diameter, swung inferiorly to posteriorly and was mobile. Right ventricular systolic function was decreased, with akinesis of the free-wall apex. The RV thrombus in the apex, which was 23×14 mm in diameter, was also mobile. Tricuspid valve regurgitation was mild to moderate, and RV systolic pressure was 48 mm Hg. A computed tomography scan showed infarctions in the spleen and kidney and no clinically significant stenoses in the coronary arteries. In addition

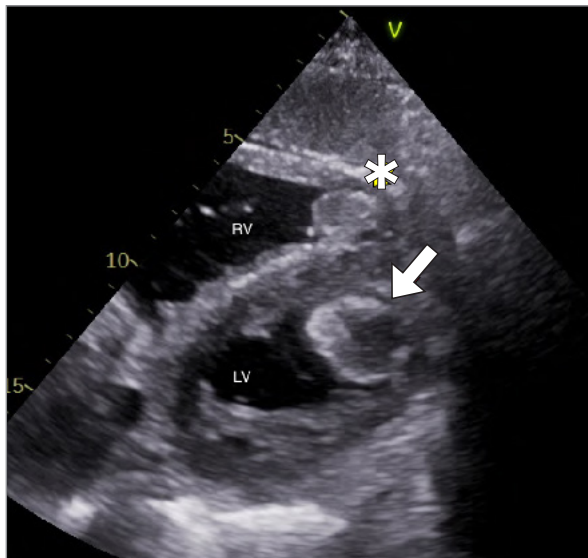


Fig. 2 Transthoracic echocardiogram shows the thrombi in the bilateral ventricles. The arrow indicates the thrombus in the left ventricle (LV), and the asterisk shows the thrombus in the right ventricle (RV). Both thrombi were spherical and mobile.

Key Points

- Giant floating thrombi forming simultaneously in the left and right ventricles is a rare condition. Emergency thrombectomy was performed in this patient to prevent the occurrence of critical events such as sudden death, cerebral infarction, or pulmonary artery embolism.
- Because of severely restricted LVEF, thrombectomy through the aortic and tricuspid valves was useful to avoid damaging the cardiac musculature.
- An endoscope and forceps were used to remove the fragile blood clots completely and to shorten aortic cross-clamp time. The patient's postoperative course was uneventful.

Abbreviations

LV, Left ventricular
LVEF, Left ventricular ejection fraction
RV, Right ventricular
TTE, Transthoracic echocardiography

to the biventricular thrombi and bilateral pleural effusion, splenic infarction and bilateral kidney infarction were observed (Fig. 3).

Medical History

The patient had a medical history of hypertension and diabetes, for which she had discontinued treatment 1 year ago.

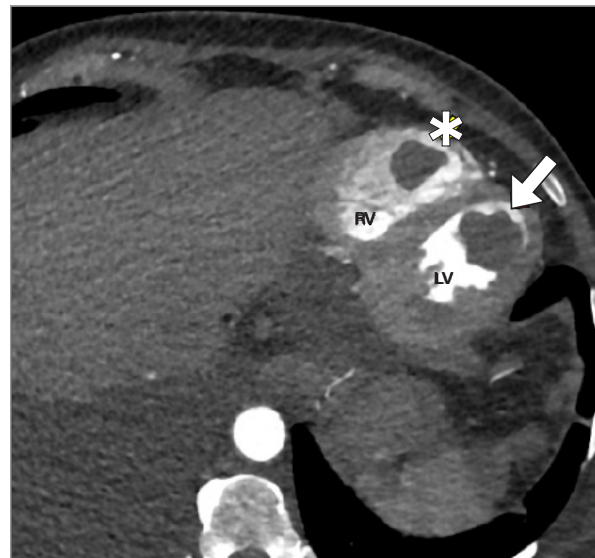


Fig. 3 Computed tomogram shows the thrombi in the bilateral ventricles. The arrow indicates the thrombus in the left ventricle (LV), and the asterisk shows the thrombus in the right ventricle (RV).

Technique

To prevent fatal embolism, emergent biventricular thrombectomy and tricuspid valve annuloplasty were performed. After induction of general anesthesia, a median sternotomy was performed. Cardiopulmonary bypass was established by cannulating the ascending aorta for perfusion and the superior and inferior venae cavae for drainage. An LV vent was inserted via the right upper pulmonary vein, and after cardiac arrest and aortotomy, it was placed in the left ventricle. Cardiac arrest was obtained with antegrade cardioplegia. Although visualization of part of the thrombi in the left ventricle was confirmed through the aortic valve, an endoscope

was used to view the whole left ventricle. A spherical thrombus was observed in the apex of the left ventricle, along with red and white thrombi filling the trabeculae (Fig. 4). The thrombi were fragile and difficult to grasp with forceps, so they were removed with forceps and cell saver suction under endoscopic guidance. A large amount of saline was used to wash the left ventricle, and the endoscope was used to confirm that all blood clots had been completely removed. After a portion of the LV wall was biopsied to obtain a pathological specimen, the aortotomy was closed. Subsequently, the right atrium was incised, and an RV thrombus was confirmed through the tricuspid valve. The thrombus consisted of

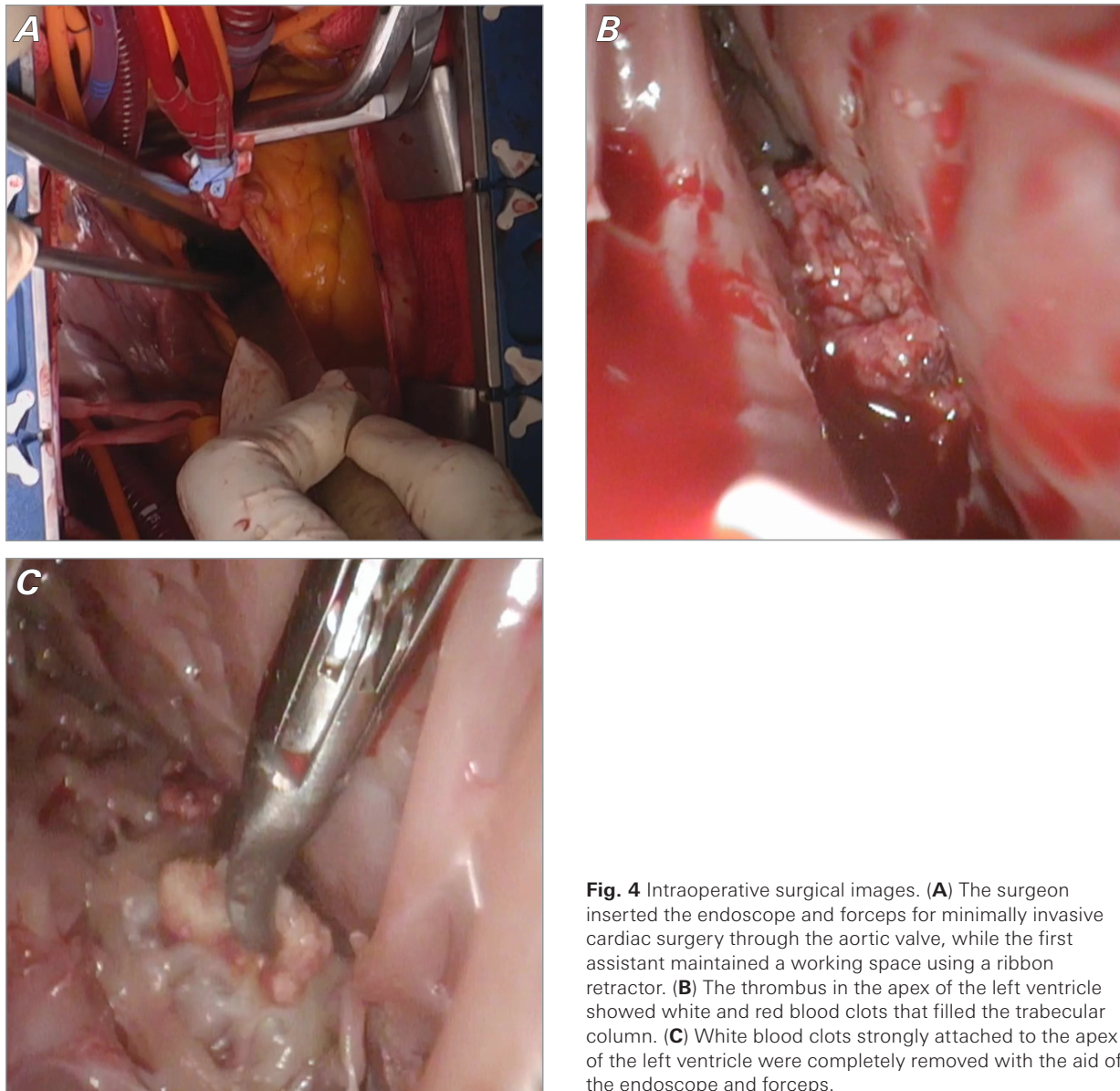


Fig. 4 Intraoperative surgical images. **(A)** The surgeon inserted the endoscope and forceps for minimally invasive cardiac surgery through the aortic valve, while the first assistant maintained a working space using a ribbon retractor. **(B)** The thrombus in the apex of the left ventricle showed white and red blood clots that filled the trabecular column. **(C)** White blood clots strongly attached to the apex of the left ventricle were completely removed with the aid of the endoscope and forceps.

red and white clots entangled with the tricuspid valve chordae, and the RV trabecular space was also filled with minute thrombi. As with the LV thrombus, the RV thrombus was removed with forceps and suction. The right ventricle was washed with a large amount of saline, and the endoscope was again used to confirm that all thrombi had been completely removed. After biventricular thrombus removal, tricuspid valve annuloplasty via the De Vega method (29 mm) was performed. Weaning from cardiopulmonary bypass was uneventful, and transesophageal echocardiography did not show thrombi in the bilateral ventricles. The total operating time was 3 hours, 27 minutes, and aortic cross-clamp time was 65 minutes.

Pathologic examination of the biopsied LV musculature showed no specific inflammation. Oral warfarin was started after surgery. Postoperative TTE showed LV end-diastolic and end-systolic diameters of 53 mm and 34 mm, respectively; an LVEF of 24%; mild tricuspid valve regurgitation; and RV systolic pressure of 38 mm Hg. Cardiovascular magnetic resonance imaging on the 10th postoperative day showed no thrombi in either ventricle and late gadolinium enhancement in the middle layer of the LV myocardium (Fig. 5). The patient was discharged from the hospital on the 11th postoperative day without any complications.

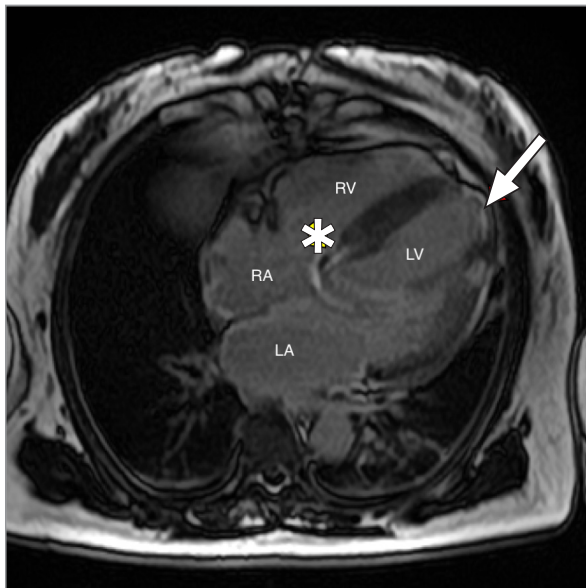


Fig. 5 Cardiovascular magnetic resonance imaging after thrombectomy shows late gadolinium enhancement in the middle layer (asterisk) and the apex (arrow) of the left ventricular myocardium. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Outcome

The patient tolerated the procedure well, with no immediate complications. Six-month follow-up TTE revealed improvement in the patient's cardiac function and removal of the biventricular thrombi (Fig. 6). Surgical thrombectomy with an endoscope via the aortic valve did not damage the LV wall. Moreover, the use of the endoscope, which enabled the complete and easy removal of all thrombi, proved to be an effective treatment for intraventricular thrombus removal. The patient was discharged with an improved clinical status.

Latest Follow-Up

At 1 year after surgery, the patient has shown no remarkable change, and her LVEF has improved on TTE.

Discussion

A situation in which thrombi form simultaneously in the left and right ventricles is extremely rare, and this condition's clinical characteristics and prognosis are not fully known. Some reports have shown the effectiveness of anticoagulant therapy for intraventricular thrombus¹ and good results obtained with surgical thrombectomy.²

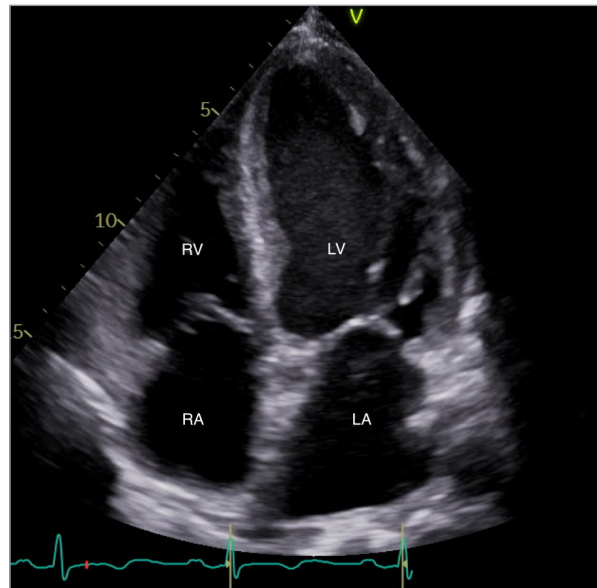


Fig. 6 Six-month follow-up transthoracic echocardiogram reveals improvement in the patient's cardiac function and removal of the biventricular thrombi. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

In some cases, however, both treatment methods have resulted in death or critical complications, such as cerebral infarction and pulmonary artery embolism. Therefore, treatment methods for intraventricular thrombi remain controversial.

This patient showed biventricular thrombi with heart failure and severely restricted cardiac function, and embolisms were observed in multiple organs on computed tomographic scanning. Transthoracic echocardiography showed large, floating, spherical thrombi. Therefore, to prevent fatal complications, an emergency thrombectomy was performed. The standard surgical approach for an apical thrombus is left ventriculotomy. However, it was thought that an approach from the LV myocardium could worsen the patient's heart failure because of her severely restricted LVEF of 25% or cause difficulty in weaning from intraoperative cardiopulmonary bypass. A right-sided left atrial approach through the mitral valve would make it difficult to view the LV cavity. In addition, in light of a case report of residual thrombus after endoscopic thrombus removal through the left atrium,³ this approach was considered inappropriate. Therefore, a thrombectomy through the aortic valve was performed. This approach has the benefit of avoiding ventriculotomy, which is particularly effective in preserving LV function in patients with low EF. Moreover, by using a ribbon retractor to avoid the right coronary cusp of the aortic valve, the LV cavity can be observed. Although there are some difficulties in inspecting the whole LV cavity, video-assisted surgery is useful to resolve such situations. Kikuchi et al⁴ reported endoscopic thrombus removal through the aortic valve for LV thrombi caused by acute myocardial infarction. In the present case, an endoscope enabled the easy removal of the white membranous thrombus that adhered to the apex of the LV cavity and provided confirmation that no minute blood clots remained between the trabeculae. Moreover, for surgical treatment, it is possible to submit pathologic specimens from LV myocardial biopsy in addition to thrombus removal to prevent future fatal complications. Pathologic results sometimes provide a definitive diagnosis of the etiology of cardiac dysfunction, which can lead to notable findings in determining treatment. The pathologic results, in this case, showed no specific inflammation, and preoperative blood tests showed no suspicion of protein C or protein S deficiency. Postoperative TTE showed that the LVEF did not improve, which suggested that cardiac function was not restricted due to acute heart failure. In addition, postoperative cardiovascular

magnetic resonance imaging showed late gadolinium enhancement in the middle layer of the myocardium, which was different from the site where the blood clot was attached, indicating hypertrophic cardiomyopathy, sarcoidosis, or viral myocarditis. Because detailed preoperative tests were not possible because of the need for emergency surgery, it will be necessary to determine a diagnosis using outpatient cardiac magnetic resonance imaging or gallium scintigraphy, which will reveal the etiology of the cardiac dysfunction.

Regarding anticoagulant therapy, long-term results have shown the usefulness of warfarin for patients with myocardial infarction complicated by LV thrombus.⁵ Moreover, it is difficult to prevent LV thrombus associated with endocardial changes, such as those after myocardial infarction, with direct oral anticoagulants.⁶ In this case, thrombi were mixed with red and white clots, and the white clots were strongly attached to the LV wall, which was thought to be accompanied by intimal changes. Therefore, heparin and warfarin were started after the surgery. The patient is currently being followed up as an outpatient, and no recurrences of thrombus in either ventricle have been observed.

Article Information

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