Arrhythmias are often relatively well tolerated hemodynamicall y, because of the function of the donor heart. Once severe native aortic regurgitation has developed, the best probable therapeutic option is re-transplantation (ie, replacing the native heart with an orthotopic transplant while leaving the original heterotopic transplant in situ). However this approach can only be used while the patient’s general condition remains suitable for transplantation.

In conclusion, this case demonstrated a new mechanism for acquired aortic valve regurgitation after HHT. We speculate that tethering of the aortic valve leaflets with thrombus may also explain some cases of aortic regurgitation in other situations in which the aortic valve rarely opens (e.g., during long-term support with a left ventricular assist device).

References

Giant Left Ventricular Pseudoaneurysm After Mitral Valve Replacement and Myocardial Infarction

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Left ventricular pseudoaneurysm is a rare but serious complication of mitral valve replacement or myocardial infarction. Prompt surgical correction is mandatory in cases of a large left ventricular pseudoaneurysm. A 70-year-old man had a giant left ventricular pseudoaneurysm after myocardial infarction and mitral valve replacement. The orifice of the pseudoaneurysm was closed with an expanded polytetrafluoroethylene patch and the pseudoaneurysmal wall was almost completely resected.


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Left ventricular pseudoaneurysm develops after rupture of the ventricular wall in an area of pericardial adhesion; this has been recognized historically as a possible sequela of mitral valve replacement and myocardial infarction. Left ventricular pseudoaneurysm is a rare but serious complication. Although there may be a role for nonoperative treatment in cases of small pseudoaneurysms, the presence of large pseudoaneurysms usually mandates surgical intervention. We describe a patient with a giant left ventricular pseudoaneurysm caused by mitral valve replacement and myocardial infarction.

A 70-year-old man was admitted to our hospital with no symptoms. He had a past history of acute myocardial infarction 2 years previously with the culprit lesion in the left circumflex coronary artery. He had also undergone percutaneous transluminal coronary angioplasty at another hospital, but it was unsuccessful. After a few weeks he had dyspnea on effort and he was diagnosed with a New York Heart Association function class II. Echocardiography revealed mitral regurgitation (Sellers’ classification, class IV) that was due to destruction of the posterior papillary muscle. He underwent mitral valve replacement with a 27 St. Jude Medical (Little Canada, MN) mechanical prosthesis. His recovery was uneventful and he was discharged home on postoperative day 20. Echocardiography was regularly performed at the outpatient clinic and revealed a gradually enlarging left ventricular pseudoaneurysm (Fig 1). No thrombotic material was detected within the pseudoaneurysm. The prosthetic mitral valve was functioning normally without paravalvular leak on echocardiography. Computed tomographic studies also demonstrated the lesion that had originated from the left ventricular posterolateral wall and measured 7 cm in diameter, and at this stage he was referred to our hospital. We thought this case was an indication for left ventricular aneurysmectomy, and preoperative cardiac catheterization was performed. The left circumflex coronary artery was occluded at number 11 and the distal portion was not visualized. Left ventriculography revealed a giant left ventricular pseudoaneurysm (Fig 2). Results of pressure study with a Swan-Ganz catheter were in the normal range. Myocardial perfusion imaging revealed no viability of the left ventricular posterolateral wall, so additional coronary artery bypass grafting was not planned.

After the patient was positioned, the right femoral artery and vein were exposed and the left lateral thoracotomy was performed in the fifth intercostal space. We selected this approach because median sternotomy had been performed at the previous operation and exposure of the lesion by retracting the heart could thus be avoided. With selective ventilation, the left lung was collapsed, and the left ventricular pseudoaneurysm was found (Fig 3); it was almost free of adhesions and originated from the left ventricular posterolateral wall. First we tried to establish cardiopulmonary bypass through cannulation of the right femoral artery and right femoral vein; however it was unsuccessful because of insufficient venous drainage. We were able to establish cardiopul-
monary bypass by additional cannulation of the pulmonary trunk. After ventricular fibrillation was induced electrically, the pseudoaneurysm was incised longitudinally. The orifice of the pseudoaneurysm was large and measured 6 cm in diameter. The edge of the orifice was densely fibrotic, and the posterior papillary muscle was in the proximity of the edge. The posterior leaflet of the mitral valve and chordae showed no remarkable abnormality. The mechanical prosthesis and its surroundings were normal. The orifice of the pseudoaneurysm was closed with an expanded polytetrafluoroethylene patch. The pseudoaneu-

Fig 1. Preoperative echocardiogram (parasternal short-axis view) showing a large left ventricular pseudoaneurysm originated from the posterolateral left ventricular wall. Dotted line = the diameter of the orifice (5.4 cm measured by echocardiogram). (LV = left ventricle; O = orifice of pseudoaneurysm; P = pseudoaneurysm.)

Fig 2. Preoperative left ventriculography in the left anterior oblique projection showing a large left ventricular pseudoaneurysm. (LV = left ventricle; O = orifice of pseudoaneurysm; P = pseudoaneurysm.)

Fig 3. An intraoperative view of a giant left ventricular pseudoaneurysm was recognized (in the lower middle). (P = pseudoaneurysm.)

rysmal wall was almost completely resected, and it was closed by means of direct suture. Pathologic examination of the pseudoaneurysmal wall revealed no myocardial tissue. The patient’s recovery was uneventful, and he was discharged home on postoperative day 14. The patient was well and symptom free 7 months postoperatively.

Comment

Acquired left ventricular pseudoaneurysms develop after myocardial infarction, surgery, trauma, or infection [1]. Postsurgical pseudoaneurysms occur after replacement of the mitral valve or arise at a previous ventriculotomy site [2]. This complication occurs in 0.02% to 2.0% of mitral valve replacements [2, 3]. Predisposing factors include resection of the posterior leaflet, overzealous decalcification of the annulus, insertion of an oversized prosthesis, and redo mitral valve replacement. The main cause of pseudoaneurysm in our case was mitral valve replacement, because the enlarging pseudoaneurysm was found after mitral valve replacement, but the combination of myocardial infarction facilitated the formation of a giant pseudoaneurysm. Although the presence of a narrow neck is strongly suggestive of a pseudoaneurysm [4], the orifice of the pseudoaneurysm in our case was very large and was impossible to repair by direct closure. A pseudoaneurysm more than 3 cm in diameter is an operative indication in chronic and asymptomatic pseudoaneurysms [4], and this indication applied in our case.

The left thoracotomy approach was a better option because ablation of extensive adhesions is usually required at reoperation when approached by a repeated median sternotomy, as in our case. Furthermore, exposure of the lesion by retracting the heart is avoided by the left thoracotomy approach [5]. Pseudoaneurysms are repaired from outside or inside of the heart. The advantages of internal repair are: (1) better exposure of the subannular apparatus is obtained, making repair straightforward; (2) additional cardiac abnormalities can be repaired simultaneously; and (3) the left circumflex coronary artery is better protected than with...
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REFERENCES


Mitral Valve Replacement in a Patient With a Collapsed Lung and a Giant Abscess

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Mitral valve replacement was performed on a 75-year-old man with a history of pulmonary tuberculosis. Computed tomography showed a collapsed left lung and counterclockwise rotation of the heart due to a hard abscess. Surgery was performed through a median sternotomy, and extensive pericardial suspension was useful for obtaining an adequate view. Despite poor pulmonary function, the patient was extubated on the day of surgery and had an uneventful postoperative course. Cardiac surgery can be performed in patients with a single functional lung if their preoperative respiratory function is good enough to have daily life without dyspnea.

Cardiac surgery is rarely performed in patients with a single functional lung. Several patients who have undergone cardiac surgery after pneumonectomy have reported success. We report a patient with a collapsed lung and a giant abscess due to tuberculosis who underwent successful mitral valve replacement.

A 75-year-old man was admitted to our hospital with dyspnea and edema of the legs, and he was found to have mitral regurgitation and congestive heart failure. He had been diagnosed with pulmonary tuberculosis 50 years ago, but he had not suffered from any medical complaints during daily life before the mitral regurgitation had developed. A grade 3 systolic murmur was heard along the left sternal boarder and at the apex. Chest roentgenogram revealed a giant abscess with fluid collection, which caused almost total collapse of the left lung and hyperinflation of the right lung (Fig 1). Computed tomography showed deformity of the left chest wall and a giant abscess with a calcified shell and fluid collection in the left chest cavity. The left lung had collapsed and the heart had been rotated counterclockwise around its long axis due to the hard abscess (Fig 2). Pulmonary function tests revealed severe obstructive and restrictive lung disease. His vital capacity was 1.80 L (55.6% of the predicted value), and the forced expiratory volume in 1 second was 0.86 L (36.9% of the predicted value), which increased to 1.15 L after bronchodilator inhalation. The room-air arterial blood gases showed a normal pH with hypercapnia and hypoxemia; the carbon dioxide tension (PaCO₂) was 52.6 mm Hg, and the oxygen tension (PaO₂) was 75.1 mm Hg. Echocardiography confirmed severe mitral regurgitation due to prolapse of the anterior leaflet. Electrocardiography showed atrial fibrillation, and coronary angiography showed normal coronary arteries.

Surgery was performed through a median sternotomy. The right pleura, which crossed the midline, was carefully peeled away from the pericardium so as not to open the right pleural cavity. The heart was rotated counterclockwise, placing the right atrium in a more posterior position than usual. Only the right side of the posterior pericardium was elevated extensively, and the right atrial appendage could be seen.

Cannulation for cardiopulmonary bypass was achieved. An arterial cannula was inserted into the ascending aorta and two venous cannulas were inserted into the superior vena cava directly and the inferior vena cava through the right atrium with forceful retraction of the aorta and right ventricle. The aorta was cross clamped and the heart was arrested using warm blood cardioplegia. The mitral valve was exposed through a transseptal superior approach, and the exposure was facilitated by stay sutures around the margin of the incision. The valve was replaced with a bi-leaflet mechanical prosthesis (27 mm) (Sulzer-Carbomedics Inc, Austin, TX). Weaning from cardiopulmonary bypass was easily achieved.

The patient was extubated on the day of the operation with intravenous administration of aminophylline. Postoperative recovery was uneventful, and results of the