

Surgical Management of Giant Left Atrium during Mitral Valve Surgery

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Abstract

Case Report

Rheumatic mitral valve disease, a preventable and eradicable condition, remains the primary indication of mitral valve surgery in developing countries. Delayed surgery is the main cause of giant left atrium (LA). This extreme dilatation of LA causes various complications compromising hemodynamic stability. We describe herein a patient with severe mitral stenosis and giant LA who had successful surgical correction by mitral valve replacement with reduction of the size of a LA.

Keywords: Giant left atrium, mitral valve stenosis, volume reduction.

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INTRODUCTION

Giant left atrium (LA) is a condition usually related to rheumatic mitral valve disease. Compression of surrounding structure, hemodynamic disturbance, atrial fibrillation and thromboembolism are the major complications of giant LA.

The clinical profile of our case is presented as well as a discussion of the definition, etiologies, indications for surgery and surgical management.

CASE REPORT

We report the case of a 42-year-old woman with a history of symptomatic severe rheumatic mitral stenosis under medical treatment. Dyspnea and palpitations were the main symptoms, which had worsened during the last month. Within her admission, she was conscious with normal blood pressure and tachycardia at 105 beats/min. Her neck veins were distended, and there were peripheral oedema signs of right-side heart failure. On auscultation, the presence of a mid-diastolic rumbling murmur. The electrocardiogram revealed rapid atrial fibrillation. The chest x-ray showed cardiomegaly, and the cardiothoracic ratio was 0.85. The patient underwent a transthoracic echocardiography (ETT), which revealed severe mitral stenosis (MVA = 0.4 cm² by pressure half time and a mean diastolic pressure gradient of 17 mmHg). Also, there was a giant left atrium (LA) with an anteroposterior diameter of 11.5 cm in the parasternal long axis view. The estimated left atrial volume was 867 ml. ETT showed mild tricuspid

regurgitation with a right ventricular systolic pressure of 55 mmHg and a tricuspid annulus diameter of 44mm. Intraoperative transesophageal echocardiography (TEE) was performed showing GLA filled with "heavy smoke" shadow without thrombi (Fig. 1).

Since the patient had dysphagia, chest computed tomography (CT) was done, which showed a giant left atrium measuring 10.6 x 14.6 cm with compression of the middle and lower lobes of the right lung (Fig. left).

After sternotomy, cardiopulmonary bypass (CPB) was started in standard fashion by aortic and bicaval cannulation. The IVC is cannulated low, and the SVC is directly cannulated as far distal as possible. In order to facilitate the later reduction of the LA, we proceeded to dissection of the adhesion between the LA and the postero-basal wall of the left ventricle while the heart was beating. Cardiac arrest and myocardial protection were obtained by the use of intermittent antegrade cold blood cardioplegia. Exposure of the mitral valve was achieved via a left atriotomy parallel to the interatrial groove. Mitral valve replacement was performed with a mechanical prosthesis, and endocardial exclusion of the left atrial appendage was done with 5-0 polypropylene suture material. Then, as described by Rios-Ortega *et al.*, [10], the left atriotomy is extended caudally through the oblique sinus to the inferior wall of the LA between the left and right pulmonary veins. We resected a 2.5 -cm wide strip and closed it with a 4-0 polypropylene running suture. The

incision is also extended cranially through the roof of the LA, and we resected a 1.5 -cm wide strip and closed it with a 4- 0 polypropylene running suture. Lastly, after the right atriotomy, a tricuspid annuloplasty using an annuloplasty ring was performed. The patient was easily weaned from CPB.

The postoperative course was uneventful. Anticoagulation therapy was initiated with an intravenous infusion of heparin four hours after the intervention, and oral anticoagulants were administered

within 24 hours after the operation. The patient was discharged two weeks after surgery. Postoperative follow-up by chest CT three months after surgery (Fig. right) showed a decrease in left atrial size from 10.6 cm to 7.2 cm in the anteroposterior diameter.

During the time she was hospitalized and after the surgery, the patient was delighted with the care she received and was optimistic about the outcome of her condition.

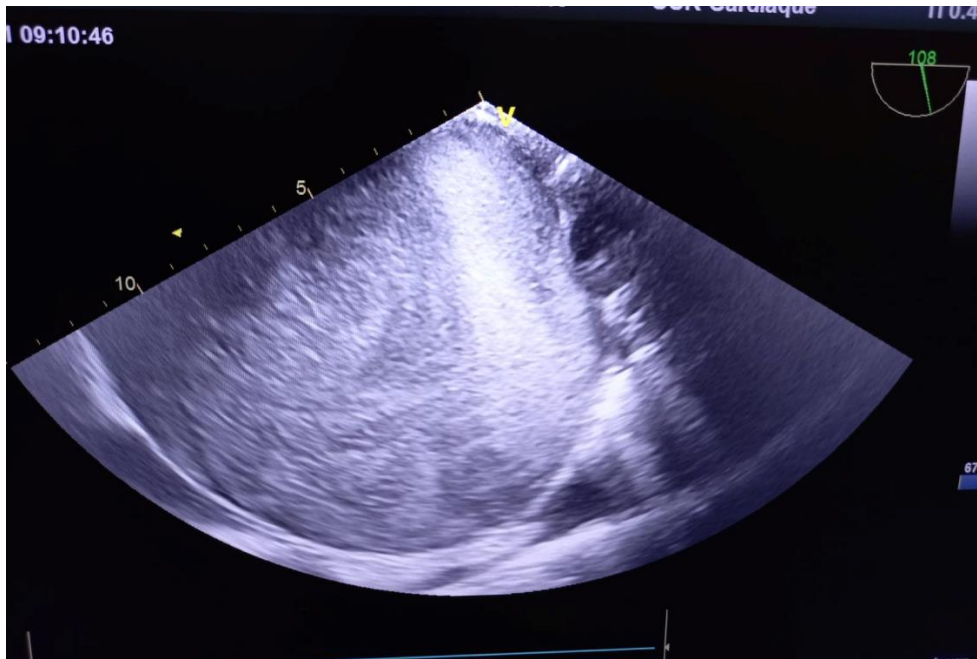


Fig. 1: Transesophageal echocardiography showing giant left atrium with “heavy smoke” shadow

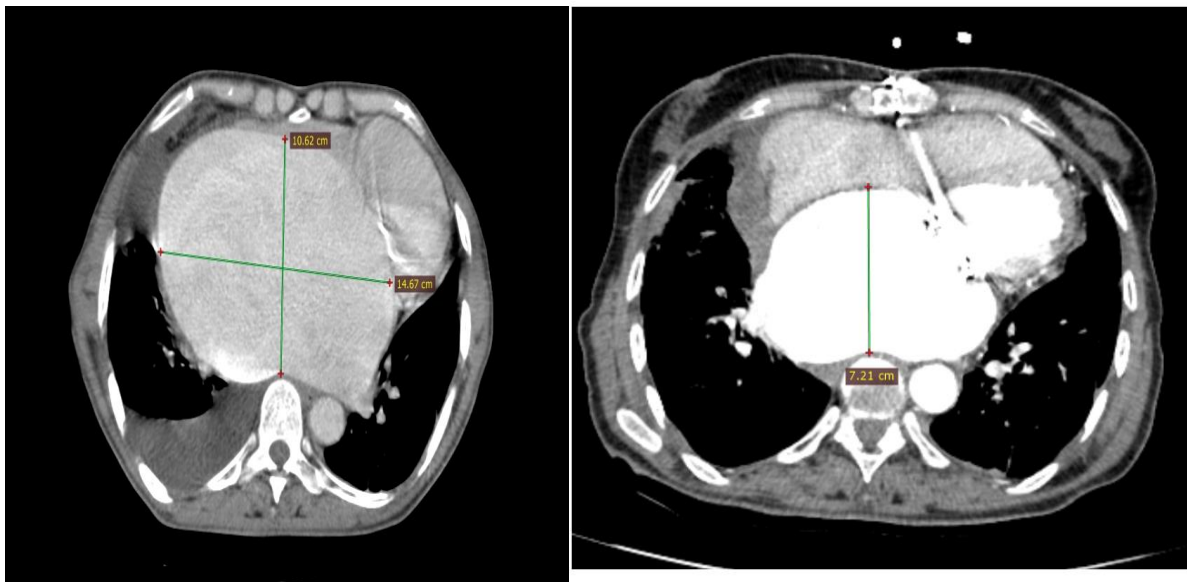


Fig. 2: Left: Chest computed tomography showing giant left atrium measuring 10.6 x 14.6 cm with compression of the right middle and lower lobes of lung
 Right: Postoperative chest computed tomography showing reduction of left atrium size

DISCUSSION

Giant LA is rare, with a reported incidence of 0.3% [1]. Hurst [2] defined a giant LA on CXR as "one that touches the right lateral side of the chest wall". He considered that "when the left atrium enlarges, it moves rightward and, in the giant left atrium syndrome, will touch the right lateral wall of the chest".

Many definitions of giant LA have been proposed, ranging from 6 to more than 10 cm in anterior-posterior diameter. Because rheumatic heart disease remains a public health issue in certain low- and middle-income countries where an enlarged LA is frequently observed, a diameter of more than 10 cm should be considered as a definition of a giant LA, as suggested by Ates and coworkers [3].

Pandit *et al.*, [4] reported the case of a 50-year-old female with a left atrial wall diameter of 19.4 cm as measured by echocardiography. Magnetic resonance imaging showed a LA size of 22.3 cm, which is the largest diameter that we find in the literature.

Rheumatic heart disease is the main cause of giant LA. The mechanism is explained by Hurst [2]: "rheumatic pancarditis damages the entire heart, including the left and right atria". However, other etiologies, such as functional mitral regurgitation [5], cardiac amyloidosis [6], and hypertrophic cardiomyopathy [7], have been reported.

The presence of compressive symptoms (dysphagia, hoarseness of voice), thrombus, or previous thromboembolic events are the main indications for LA reduction surgery. This reduction is concomitant with mitral valve surgery most of the time. Some surgeons believe that mitral valve surgery alone can lead to an eventual reduction in the LA size. Apostolakis *et al.*, [8] explained that is not true because the changes observed in the left atrium of patients with giant LA are significant and sometimes irreversible. Also, Abhayaratna and colleagues [9] argued that left atrial size is an important prognostic factor for cardiovascular outcomes.

Surgical techniques used to reduce the LA size vary from partial plication of the inferior atrial wall to partial auto-transplantation of the heart. For its safety and reproducibility, we used a technique described by Rios-Ortegas *et al.*, [10], which are a variation of the "spiral resection" proposed by Sugiki and coworkers [11]. This resulted in a postoperative improvement in NYHA functional class and compressive

symptomatology. Atrial reduction includes the caudally inferior wall between the pulmonary veins and cranially across the LA roof. The main disadvantage is the non-plication of the interatrial septum.

CONCLUSION

LA volume reduction while performing mitral valve surgery is a feasible and effective technique that should be considered for patients with giant LA.

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