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Recurrence of left ventricular pseudoaneurysm after multiple mitral valve replacements

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Abstract

Left ventricular pseudoaneurysm (LVPA) formation is a potentially lethal complication of myocardial infarction (MI) and mitral valve (MV) replacement that requires prompt diagnosis and treatment. A female patient who had been complaining of exertional dyspnea underwent a two-dimensional transthoracic echocardiogram (TTE) which revealed a functioning mechanical MV with severe paravalvular leak, severe tricuspid regurgitation (TR) and severely elevated pulmonary artery systolic pressure. Moreover, echo-lucent space at the postero-lateral portion of the left ventricle near the MV was seen, suggestive of a large LVPA. Transesophageal echocardiography (TEE) and computed tomography (CT) angiography confirmed these findings. Afterwards, the patient had a surgical repair for the LVPA along with mitral and tricuspid valve (TV) replacement. Three months later, the patient presented with symptoms of congestive heart failure. The LVPA had recurred at the same location of the previous pseudoaneurysm and given the high risk for reoperating on the patient, close monitoring and medical management was deemed as a better option.

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Introduction

Left ventricular (LV) wall rupture and left ventricular pseudoaneurysm (LVPA) formation are two potentially lethal but rare complications of myocardial infarction (MI) and mitral valve (MV) replacement that occurs in less than 0.2% and 0.1%, respectively [1]. It undergoes rapid enlargement and carries a significant risk of being ruptured ranging from 30% to 45% [2]. Since LVPAs present with nonspecific symptoms such as chest pain, shortness of breath and palpitations, its diagnosis is often delayed.

Case Report

A 55-year-old female followed at the outpatient valve clinic, with a history of atrial fibrillation, type 2 diabetes mellitus, hypertension, rheumatic heart disease, and previous multiple cardiac surgeries. The first cardiac surgery was a tricuspid valve (TV) repair at the age of 33, the second operation was a MV replacement with bio-prosthesis, and the third was replacing the bio-prosthesis with a mechanical valve due to infective endocarditis. The patient complained of worsening exertional dyspnea (NYHA III) and chest discomfort combined with signs of heart failure.

Laboratory tests indicated anemia (hemoglobin: 79 g/L, MCV: 84.5 fL, MCH: 23.7 pg, MCHC 281 g/L, reticulocytes: 122.4/L, haptoglobin <0.1 g/L, LD 484, ferritin 297 ng/mL, ALT 51,1 U/L). A two-dimensional transthoracic echocardiogram (TTE) performed six months after the surgery showed severe concentric LV hypertrophy, a normally functioning mechanical MV with a severe lateral paravalvular leak. Furthermore, a large echo-lucent space with a narrow neck (about 17 mm in diameter) on the postero-lateral portion of the LV near the prosthetic MV, suggestive of a giant LVPA was shown (Figure 1). Transesophageal echocardiography (TEE) confirmed the presence of a LVPA with blood flow within it, localized below the left atrial appendage with no communica-

tion as well as severe MV paravalvular leak (Figure 2). Computed tomography (CT) angiography revealed a sub-mitral annulus LVPA defect located posteriorly. The neck was 16 mm, and its extension was about 24 mm (Figure 3).

The patient underwent cardiac surgery for the fourth time, where yet again, it was an MV replacement with a bioprosthetic valve 33 mm, and Epic bioprosthetic 31 mm TV and LVPA repair. The neck of the LVPA was seen just at the end of the A1 scallop, continuing posteriorly and a tear was identified on the posterior annulus. The LVPA was repaired utilizing a bovine pericardial patch. Intraoperative measurements of LVPA were 6 cm deep and 4x5 cm communication neck with the LV. The patch was also used to repair the tear on the posterior annulus. The MV prosthesis was sutured by passing through the annulus and the sewing ring of the prosthesis. The TV had undergone annuloplasty three times, and although the tricuspid leaflets were mildly involved by the rheumatic disease, the team decided to replace it with a bioprosthetic valve. Intra-operative TEE confirmed the complete closure of the LVPA orifice with no blood flow within it, and the persistence of a moderate paravalvular leak requiring repair. However, a residual paravalvular leak remained after the repair, yet it was acceptable since it was mild (Figure 4). The patient then developed a complete atrioventricular block ten days after surgery, requiring pacemaker implantation. The rest of the hospital admission was unremarkable, and the patient was discharged fifteen days post-surgery.

Three months following the surgery, the patient presented to the outpatient clinic with signs and symptoms of congestive heart failure and was readmitted. TTE showed mild paravalvular leak and the presence of a small cavity at the exact location of the previous LVPA with flow inside. The findings suggested recurrence of the LVPA confirmed by CT angiography, however smaller in size in comparison to the previous one (Figure 5). The patient was managed medically increasing her medications' dosage along with close follow-up. At the next visit, she was hemodynamically stable and NYHA II.

Discussion

This is a case of a patient with history of four cardiac surgeries: three MV replacements and a recurrent LVPA. The patient developed LVPA and severe paravalvular leak secondary to her third MV replacement. The diagnosis was made by utilizing multimodality imaging: the complexity of the heart lesions was described by TTE and TEE but the dimension and relation with adjacent structure of the LVPA was best described by CT angiography. Urgent cardiac surgery was the only therapeutic option but not as a long-term resolution. These complications were likely related to loosened stitching between the implanted pericardial patch, mitral annulus and the lateral LV side.

LVPA, also known as a false aneurysm, is defined as a contained rupture or tear of the myocardium [3]. LVPA rarely occurs in clinical practice and is most commonly associated with MI. On the other hand, it is an extremely rare complication post-MV repairs, occurring in 0.02-1% of all MV replacements [1].

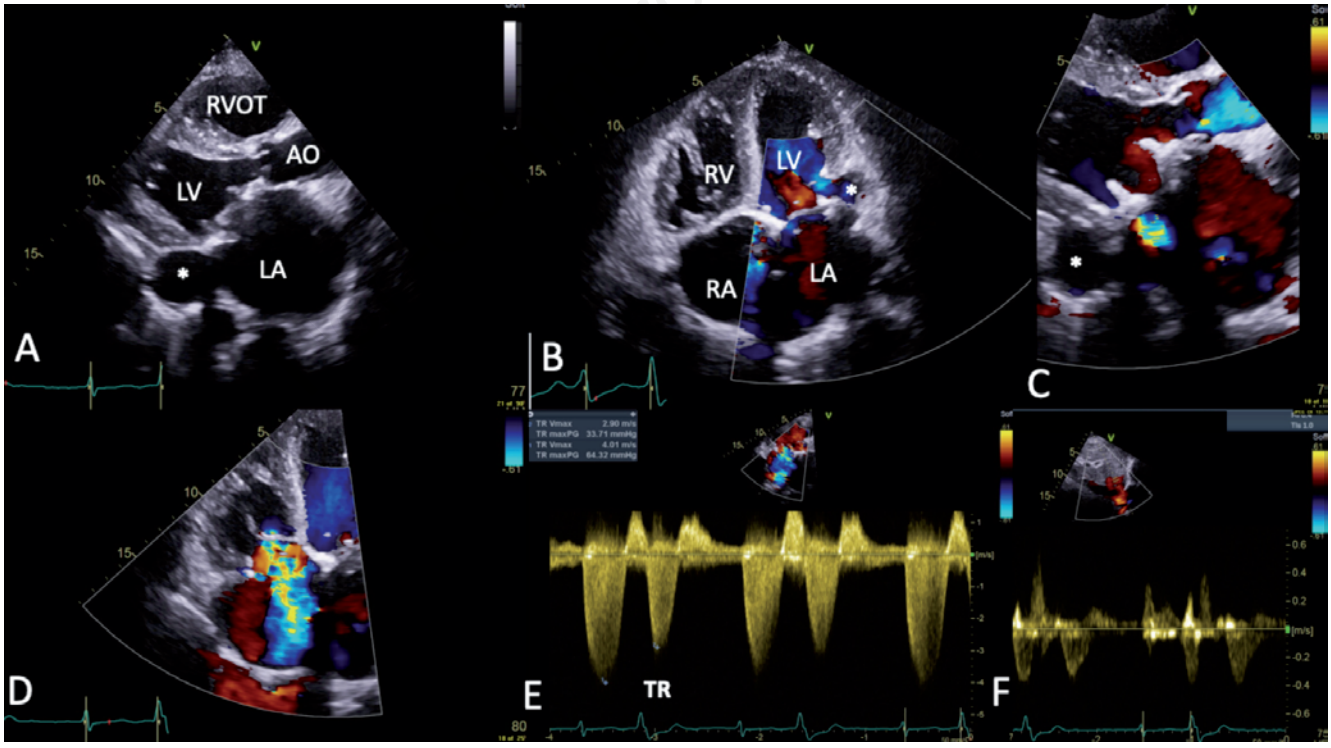

Figure 1. Upper row transthoracic echocardiography (TTE) showing: A) Parasternal long access. B) Apical four chambers with color Doppler showing paravalvular leak around the mitral prosthesis and flow between the LV and the pseudoaneurysm. C) Parasternal long access with color Doppler showing paravalvular leak around the mitral prosthesis; asterisk represents the pseudoaneurysm; lower row represented the tricuspid regurgitation (TR). D) Apical four chamber view with color Doppler across the tricuspid valve (TV) showed TR. E) Continuous wave signal showed TR peak velocity of 4 m/s with peak gradient of 64 mmHg, premature ventricular contractions are noted in bigeminy pattern during the study. F) Pulsed wave of hepatic vein showed systolic reversal.

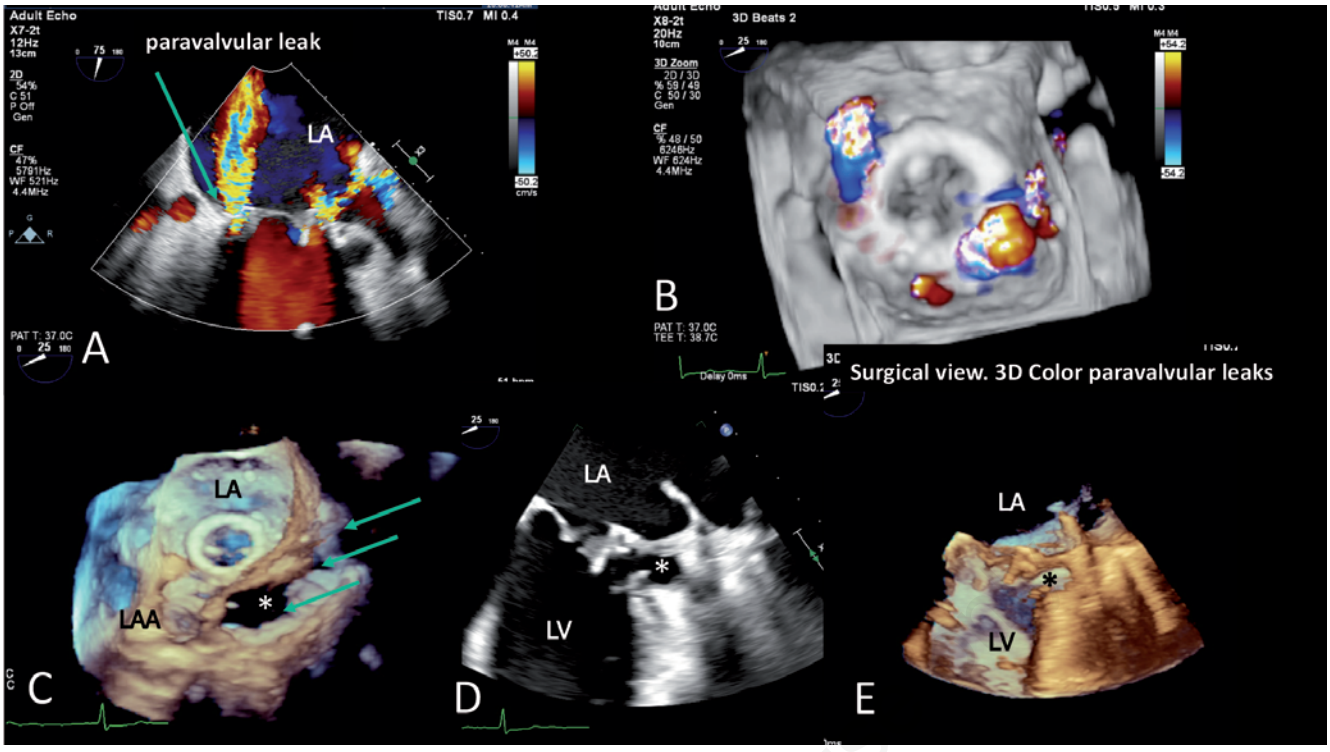

Figure 2. TEE showing color Doppler (A) paravalvular leak across the mitral prosthesis; B) same view with pseudoaneurysm; C) TEE 3D view showing the paravalvular leak around the mitral prosthesis. Asterisk represents the pseudoaneurysm.

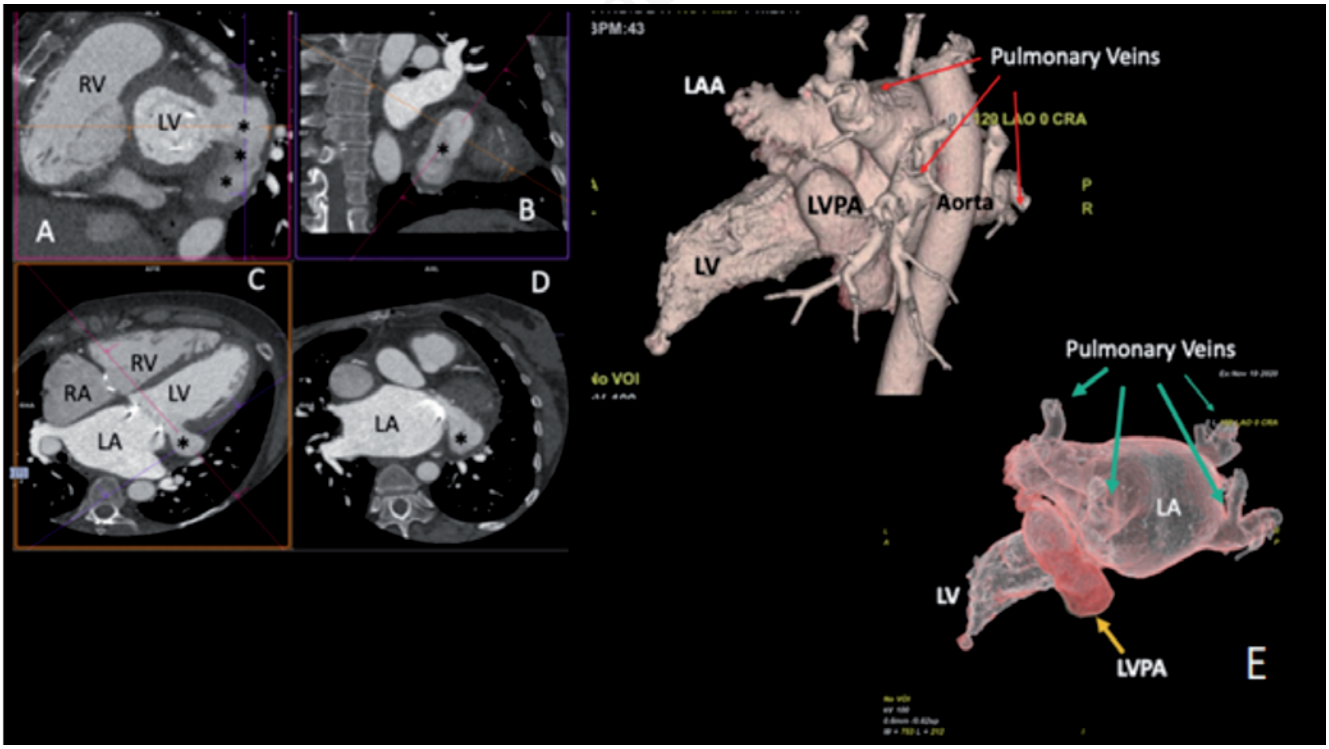

Figure 3. Cardiac CT scan showing: A) Short access view with pseudoaneurysm. B) Sagittal plain of the pseudoaneurysm. C) Four chambers view demonstrating the pseudoaneurysm between the crossing lines. D) Upper cuts at the level of left atrium (LA); asterisk represent the pseudoaneurysm. E) 3D reconstruction of pseudoaneurysm with illumination of the great vessels.

Predisposing factors are reoperation, oversized prosthesis, endocarditis, female gender, mitral stenosis, advanced age, small LV volume and small body size. From the surgical point of view, multiple factors could be responsible for the development of a LVPA. For instance, excessive mitral tissue removal during valve repair can damage the annulus resulting in a LVPA. Inadvertent

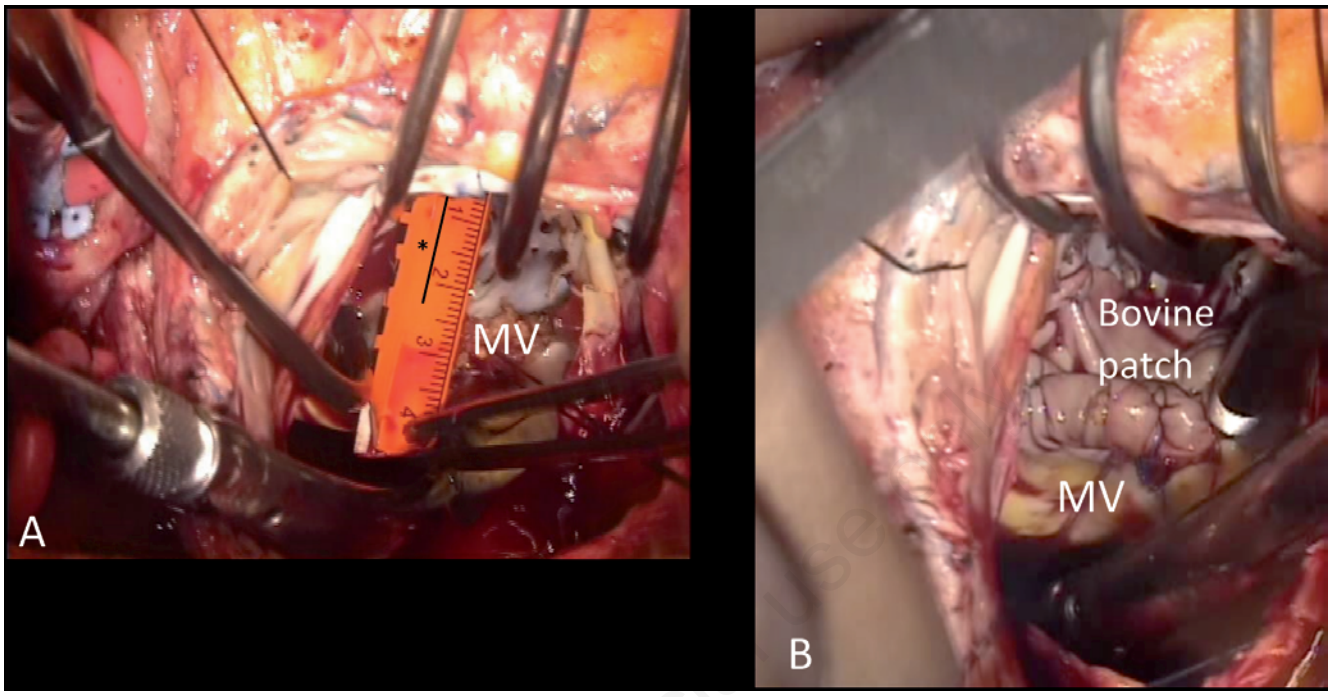

Figure 4. A) Intra-operative image before closure of pseudoaneurysm with mitral valve and the pseudoaneurysm beneath the valve; measurement of neck of the pseudoaneurysm around 2.4 cm. B) Post closure of the pseudoaneurysm with bovine patch.

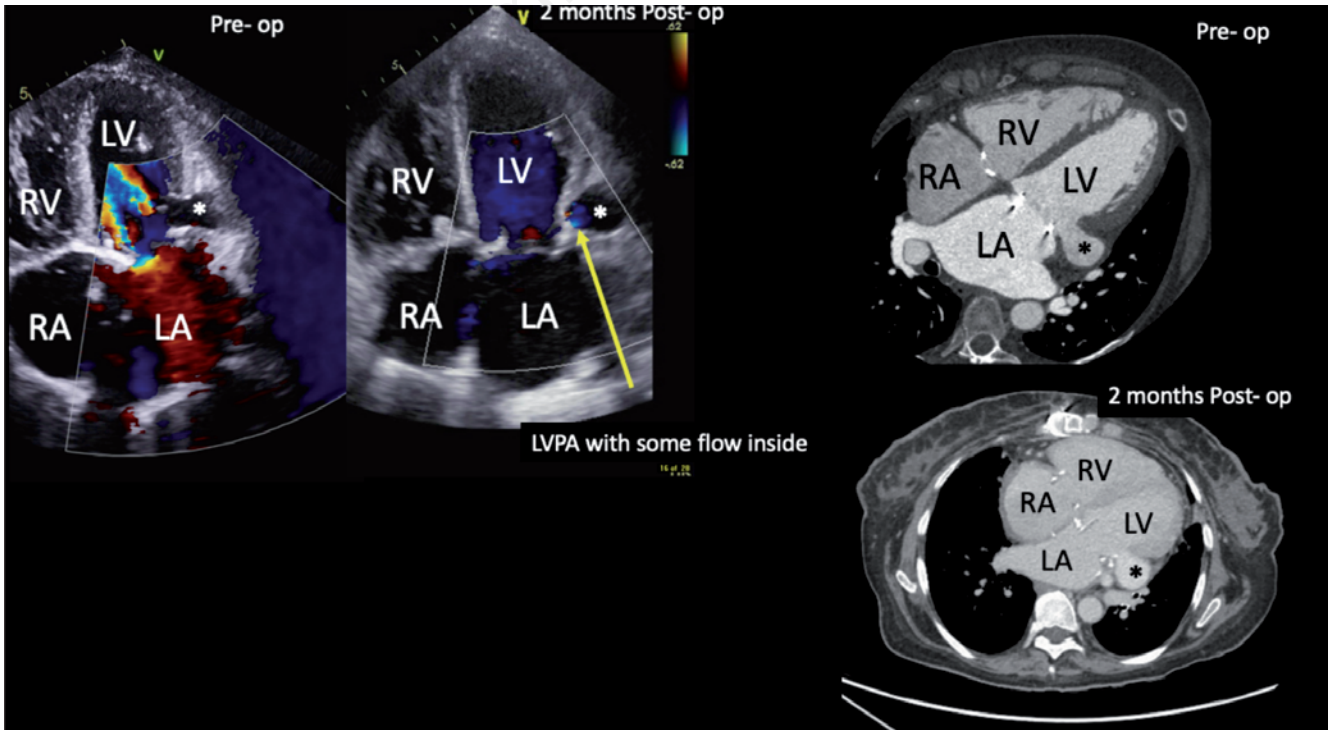

Figure 5. Comparison TTE and CT pre- and 2 months post-surgery. Asterisk represents pseudoaneurysm.

incision and/or forced traction of the annulus, deeply placed sutures along with excessive traction on them can affect the annulus negatively. Beside the aforementioned, insertion of an oversized mitral prosthesis can stretch the annulus ultimately leading to LVPA [4].

LVPA is reported to be most commonly associated with MI (55%), followed by surgery (33%), with MV replacement being the most common type, and trauma accounting for 7% of the cases [5]. Depending on the time of diagnosis, LVPA can be classified into three groups: the first class is early LV wall rupture, where it happens intraoperatively. The second is delayed, which occurs several hours or days postoperatively. Lastly, the late LV wall rupture, which usually occurs months to years after the operation [2].

The clinical presentation of LVPA varies based on its size and location. LVPA can present as chest pain and dyspnea, but patients can still be asymptomatic. A case series from Mayo Clinic on 52 patients revealed that 48% of patients were asymptomatic [5]. Our patient was more of a complex case since symptoms were related to congestive heart failure due to impaired biventricular systolic function, pulmonary hypertension and hemolytic anaemia secondary to severe mitral paravalvular leak. The LVPA was found and described in its extension and relation with the adjacent structures using the multi-imaging modality. Due to the high risk of LVPA rupture, an early diagnosis is crucial in the management and outcome of these patients. Previously, LVPA diagnosis was made by LV angiography, although it is rarely requested nowadays [5]. Echocardiography and CT angiography are the diagnostic approach for such patients [1], as in our case. Surgical repair is the preferred therapeutic option, although it is associated with a high mortality rate (23%). Medical therapy is recommended for patients with chronic LVPA that is <3 mm in size or for patients that are considered high risk for re-operation. Medical treatment aims to reduce LVPA enlargement and LV wall stress by lowering the afterload and reducing the risk of complications such as thromboembolism, though it is linked to 48% risk mortality [5].

Conclusions

We presented the case of a patient with a history of multiple cardiac surgeries which started at the age of 33 years due to rheumatic valve disease, further complicated by LVPA and its recurrence after the third MV replacement. This patient presented with almost all the possible risk factors for developing an LVPA including several reoperations, endocarditis, friable tissue, female gender and some surgical technicalities. Although LVPA is uncommon, it is potentially lethal. Cardiac surgery is the treatment of choice for patients with such condition, but in cases where patients are at high risk for reoperation, medical management and close follow-up is the treatment of choice.

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