ESC European Society of Cardiology

European Heart Journal - Case Reports (2020) **4**, 1–5
doi:10.1093/ehjcr/ytaa091

CASE REPORT
Cardiac imaging

Late atrioventricular groove disruption presenting 7 years after mitral valve replacement: a case report

Ciro Mancilha Murad [1]*, Letícia Braga Ferreira [1], Rochelle Coppo Militão Rausch [2], and Cláudio Léo Gelape[3]

[1]Department of Cardiology, Hospital das Clínicas, Universidade Federal de Minas Gerais, 110, Avenida Professor Alfredo Balena, Belo Horizonte MG 30.130-100, Brazil; [2]Department of Cardiology and Cardiovascular Imaging, Hospital das Clínicas, Universidade Federal de Minas Gerais, Avenida Professor Alfredo Balena n 110, Belo Horizonte MG 30.130-100, Brazil; and [3]Department of Cardiovascular Surgery, Hospital das Clínicas, Universidade Federal de Minas Gerais, Avenida Professor Alfredo Balena n 110, Belo Horizonte MG 30.130-100, Brazil

Received 27 September 2019; first decision 10 December 2019; accepted 1 April 2020; online publish-ahead-of-print 1 May 2020

Background Left ventricular rupture is the most feared complication in mitral valve surgery. Despite its low incidence, mortality rates can reach up to 75%. It usually presents on the operating room with a dissecting haematoma followed by massive bleeding after discontinuing cardiopulmomary bypass. However, cardiac rupture may be contained by adherent pericardium or scar tissue leading to chronic formation of a pseudoaneurysm (PSA).

Case summary A 44-year-old man came to our institution with acute heart failure triggered by community-acquired pneumonia. He underwent mitral valve replacement with a mechanical prosthesis 7 years before and reported suffering from chronic worsening dyspnoea for 18 months. He underwent chest computed tomography scan and cardiac magnetic resonance imaging (CMRI), which showed two extensive left ventricular (LV) multilobulated PSAs. An operative approach was chosen and a tear was found on the posterior atrioventricular groove (AVG), communicating left ventricle with the PSA, which was closed with bovine pericardium patch. After weaning from cardiopulmonary bypass, he presented a diffuse life-threatening bleeding. The surgeons packed his chest with compresses before closing the sternum and he was operatively revised after 48 h. Post-operative CMRI showed that one of the PSAs remained connected with the LV. Despite of all, 1 year after hospital discharge, he remains asymptomatic without signs of heart failure.

Discussion This case illustrates PSAs' potential to grow for a long period before causing symptoms, the complexity and risks of chronic AVG disruption surgery and the importance of careful annular manipulation and debridement as preventive measures in mitral valve surgery.

Keywords Ventricular pseudoaneurysm • Cardiac rupture • Atrioventricular groove disruption • Mitral valve replacement • Case report

Learning points

- Cardiac rupture following mitral valve replacement may present as a pseudoaneurysms in the chronic period.
- Pseudoaneurysms may grow for a long period before causing symptoms, demonstrating the importance of continuous surveillance after valve surgery.
- Preventive measures should be carried out in mitral valve surgery, avoiding annular injury with excessive debridement and tissue resection.

* Corresponding author. Tel: +55 31 9 8898 8843, Email: ciromurad@hotmail.com

Handling Editor: Nikolaos Bonaros

Peer-reviewers: Laszlo Gobolos and Thomas Schachner

Compliance Editor: Stefan Simovic

Supplementary Material Editor: Ross Thomson

Introduction

Left ventricular rupture is the most feared complication in mitral valve surgery. Despite low incidence ranging from 0.6% to 1.2%, mortality rates can reach up to 57–75%.[1,2] This complication can be classified into three types based on the location of the epicardial tear.[3,4] Type I occurs at the level of posterior atrioventricular groove (AVG), also referred as AVG disruption, Type II occurs at left ventricular posterior wall at the base of papillary muscle, and Type III occurs between the base of papillary muscle and posterior AVG. Ventricular rupture may present as a dissecting haematoma followed by massive bleeding, after discontinuing cardiopulmonary bypass (CPB), in the operating room.[1,5] Further, it can manifest hours to days after surgery, with haemodynamic instability and bleeding from drains. However, late presentation, days to years after surgery,have been described.[1,6] In this situation, cardiac rupture is contained by adherent pericardium or scar tissue leading to formation of a pseudoaneurysm (PSA) in the chronic period.[1,7]

Timeline

Date	Event
2011	Mitral valve replacement due to rheumatic valve disease.
09 June 2018	Initial presentation with signs of severe acutely decompensated heart failure triggered by community-acquired pneumonia.
14 June 2018	Echocardiogram demonstrates bi-ventricular dysfunction and an anechoic structure adjacent to the left ventricle.
14 June 2018	Computed tomography scan showed large multilobulated mediastinal formations.
20 June 2018	Cardiac magnetic resonance imaging (MRI) defined the structures as pseudoaneurysms.
04 July 2018	First surgical procedure complicated by haemorrhagic shock.
06 July 2018	Surgical reassessment.
18 July 2018	Follow-up cardiac MRI: inferior pseudoaneurysm (PSA) without contrast filling. The second PSA remained connected to left ventricle.
23 July 2018	Patient discharge.
31 August 2018	First outpatient appointment: resumed daily activities without dyspnoea or signs of overt heart failure.
18 December 2018	Transoesophageal echocardiography: left ventricle ejection fraction of 51%, normal right ventricle function, mitral prosthesis with a moderate paravalvular leak on anterior topography.
02 September 2019	Last outpatient appointment: remains asymptomatic, conservatively managed.

Case presentation

A 44-year-old man presented to our hospital complaining of productive cough and fever for 5 consecutive days. He also reported suffering from chronic worsening dyspnoea, orthopnoea, and abdominal discomfort for ∼18 months. He underwent mitral valve replacement due to rheumatic valve disease (severe regurgitation and mild stenosis) with a mechanical prosthesis 7 years before. His medical history additionally consisted of moderate asthma and atrial fibrillation. After initial surgery, the patient remained asymptomatic for 6 years. He had serial echocardiograms showing mild ventricular dysfunction without significant valve prosthesis dysfunction or thrombosis that could explain the worsening symptoms. Cardiovascular physical examination revealed elevated jugular venous pulse, irregular cardiac rhythm with S3, a lower left parasternal heave and a pathologic pulsation in the left second intercostal space. Hepatomegaly and diffuse bilateral pulmonary rales and wheezes were also discovered. Due to pulmonary consolidations on superior and inferior right lobes on chest X-Ray (*Figure 1*) and elevated inflammatory markers, the initial diagnosis was acute decompensated heart failure triggered by community-acquired pneumonia, and treatment was initiated with intravenous loop diuretics and antibiotics. Other relevant laboratory tests showed leucocytosis, mild anaemia (HgB 112 g/L), mild hypoxaemia, and a supratherapeutic international normalized ratio (5.94).

Further evaluations by chest computed tomography (CT) and echocardiogram were performed because of persistent respiratory symptoms, elevated inflammatory markers and worsening cardiovascular symptoms.

Echocardiogram showed left ventricular dilation and mild dysfunction with ejection fraction (EF) of 43%, right ventricular dilation and dysfunction, mild pericardial effusion, normally functioning mechanical mitral prosthesis and an intriguing anechoic structure adjacent to the left ventricle posterior lateral wall, near the mitral prosthesis with possible left ventricle communication (*Figure 2*). Thorax CT confirmed pneumonia with concomitant pulmonary congestion. Furthermore, it revealed two massive multilobulated formations, located in superior and inferior aspects of mediastinum, with partially calcified walls (Supplementary material online, *Figure S1*). Cardiac magnetic resonance imaging (CMRI) was performed for better assessment of these structures, showing one trilobed formation, located at left ventricle inferior wall, causing diaphragm compression, and one bilobed formation, located at left ventricle anterior wall and extending to the lateral wall. (*Figure 3*). Both structures communicated with the left ventricle through perivalvular, contrast-filled necks. The absence of ventricular muscle layer and passive filling during ventricular systole characterized these structures as PSAs

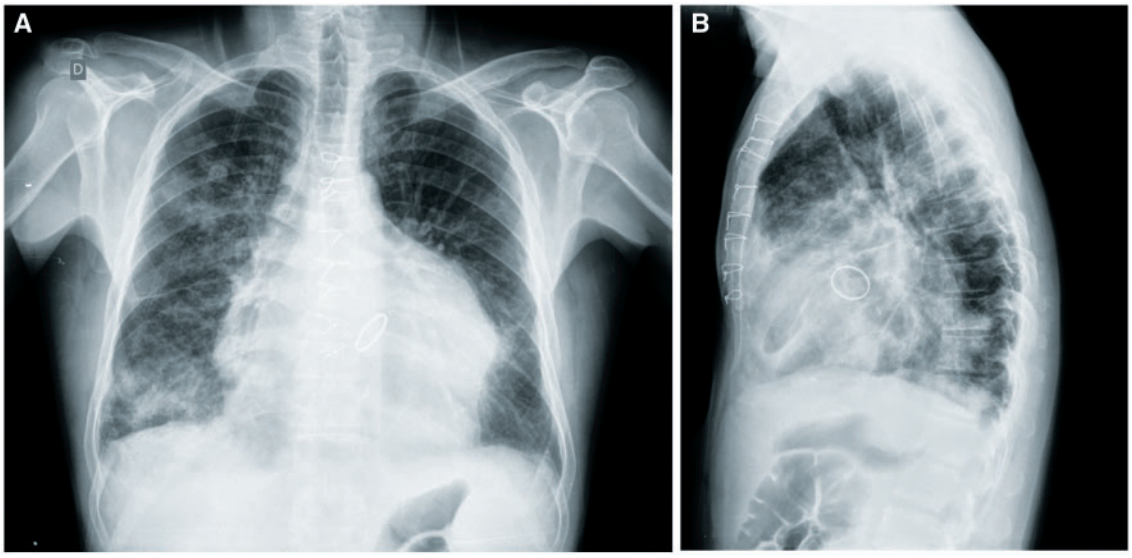

Figure 1 Chest X-ray showing marked cardiomegaly, signs of pulmonary congestion, and opacities on the right superior lobe and right inferior lobe. (*A*) Posteroanterior view. (*B*) Lateral view.

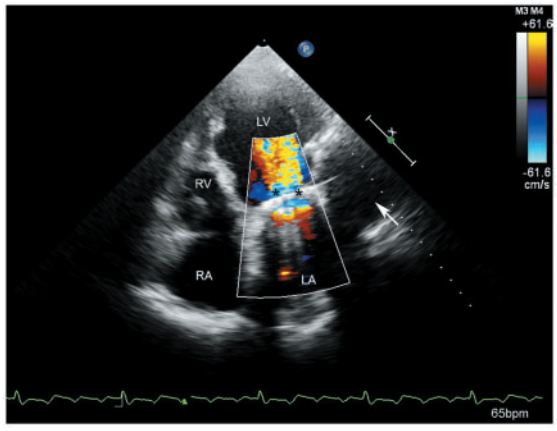

Figure 2 Transthoracic echocardiogram, apical four-chamber view. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. **Mitral valve. White arrow: anechoic structure adjacent to the left ventricle.

(Supplementary material online, *Video S1*). On coronary CT angiography, we also identified a circumflex branch occluded which was confirmed by coronary angiography (Supplementary material online, *Figure S2*).

Owing to the massive size of PSAs and the presentation with new-onset severe heart failure, an operative approach was chosen. During the procedure, after initiation of CPB, mechanical mitral prosthesis was explanted permitting posterior ventricular wall exploration. It was impossible to find the PSA neck. The option was to perform an incision of the PSA external wall, and a tear was found on the posterior AVG, below the insertion site of mitral prosthesis, communicating the PSA with the left ventricle. This communication was closed with bovine pericardium patch using single pledgeted sutures and a new mechanical prosthetic valve number 25 was implanted with interrupted stiches. After weaning from CPB and protamine reversal, patient presented diffuse bleeding on the site of dissection from PSA wall, left ventricle, and diaphragm. Surgeons did not find bleeding on suture lines, nor any bleeding with possible surgical approach. Therefore, they packed the chest with compresses as a life saving measure before closing the sternum. After 48 h, patient's chest was reopened in order to remove the towels. Further, post-operative course was uneventful.

Prior to discharge, patient was re-evaluated with CMRI. The absence of contrast filling of the inferior wall PSA confirmed its successful closure. However, a contrast-filled peri-mitral valve neck on left ventricular basal anterior segment was noted, communicating it with the PSA located at the anterior wall, and a structure suggestive of small thrombus (*Figure 4*), (Supplementary material online, *Video S2*). Due to multiple previous interventions, a conservative approach was chosen and patient was discharged without any further complications.

The patient is being followed-up since hospital discharge. On echocardiographic assessment, 5 months after surgery, despite an improvement in left ventricular function with EF of 51% and normal right ventricular function, we identified a moderate paravalvular leak from the mitral prosthesis on anterior topography, which might be related to mitral annulus instability from both procedures. His last outpatient appointment happened 1 year and 2 months after surgery: he remains asymptomatic without signs of heart failure and is being managed conservatively.

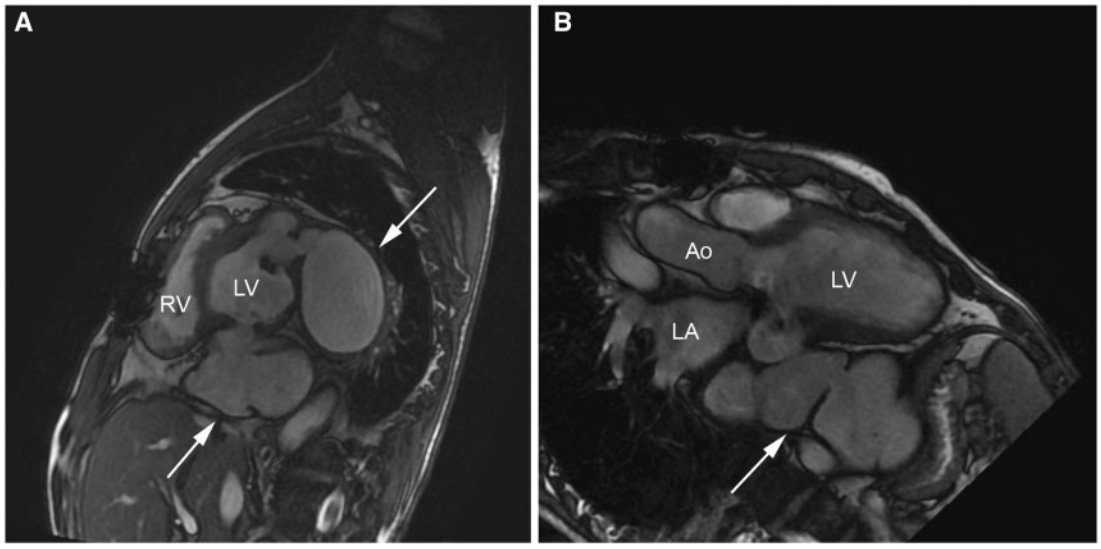

Figure 3 Cine cardiovascular magnetic resonance imaging. (*A*) Short axis. (*B*) Long axis. White arrows: pseudoaneurysms.

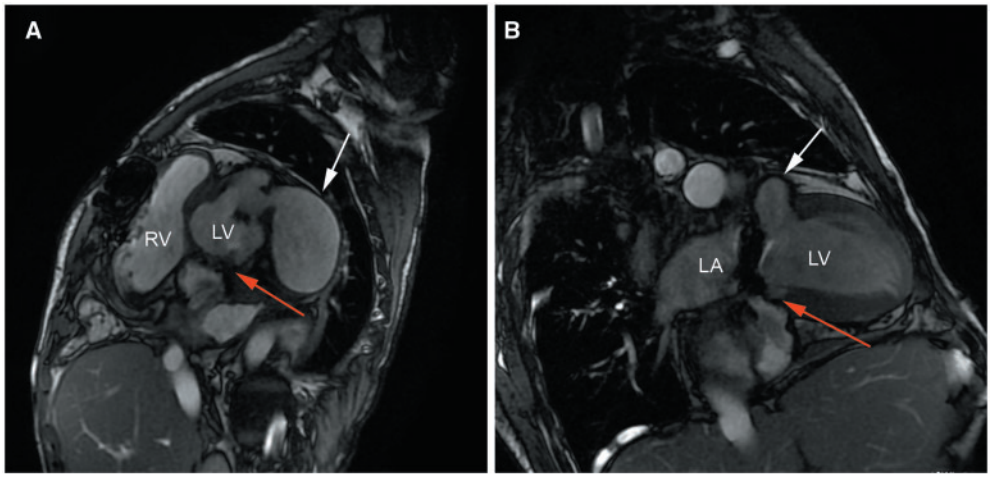

Figure 4 Post-operative cine cardiac magnetic resonance imaging. (*A*) Short axis. (*B*) Long axis. Red arrow: note the absence of contrast-filled neck communicating inferior pseudoaneurysm and left ventricle. White arrow: superior pseudoaneurysm which remained connected to the left ventricle.

Discussion

Atrioventricular groove disruption is the most common and complex type of cardiac rupture after mitral valve replacement.[5] The posterior mitral annulus tends to be more fragile than the anterior due to the lack of a well-formed fibrous cord.[8,9] Frequently, it is also more calcified.[8,9] The pathogenesis of this type of lesion involves mitral annulus injury during surgery and an underlying weakened myocardium.[1] Several predisposing conditions have been described: (i) excessive leaflet tissue resection, (ii) debridement of heavily calcified valves, (iii) forceful traction and inadvertent incision of the annulus, (iv) insertion of an oversized prosthesis, (v) deeply placed and excessively tight sutures, (vi) female sex, (vii) advanced age, and (viii) re-operations.[1,9,10] In addition , rheumatic aetiology, particularly with mitral stenosis, seems to be an important risk factor.[10] These patients usually have relatively small left ventricles, densely calcified valves and,occasionally, a weakened myocardium secondary to the pathological changes produced by rheumatic disease itself.[1,10] In the case herein described, although there was no description of cardiac rupture in the initial procedure, the surgeons reported thickly calcified mitral valve, partial posterior leaflet resection, and pericardial adherences probably secondary to previous rheumatic carditis. Moreover, the

circumflex occlusion identified might be explained either by PSA compression or by previous injury, which is frequently concomitant with posterior annular trauma due to anatomic proximity.[1,10]

Interestingly, post-operative PSAs can happen after the repair of an earlier rupture or develop *de novo* after an original procedure without a recognizable myocardial tear, as in the case we describe.[1] Most patients remain asymptomatic until development of complications, and many of them are diagnosed incidentally.[6,11]

Infective endocarditis causing suture dehiscence also plays a major role in the development of cardiac PSAs.[12,13] In this particular case, the patient had no previous history of endocarditis and, despite his presentation with fever and elevated inflammatory markers, blood cultures were negative and surgical *in situ* analysis of the prosthetic valve revealed no signs of endocarditis.

Conclusion

This case underscores PSAs' potential to grow for a long period before causing symptoms, demonstrating the importance of continuous post-operative surveillance with imaging and physical examination.[13] It also indicates the complexity and risks of chronic AVG disruption correction, stressing the importance of preventive measures in mitral valve surgery, such as proper valve sizing, carefully annular manipulation and debridement, posterior leaflet preservation and proper suture techniques with avoidance of mechanical injury.[1,9,10]

Lead author biography

Dr Ciro Mancilha Murad received his medical degree from Federal University of Minas Gerais (UFMG) in 2014 and completed general cardiology residency in 2020 at Hospital das Clínicas/UFMG. He is currently a fellow of Advanced Heart Failure and Cardiac Transplantation at Heart Institute/University of São Paulo. His areas of interest are advanced heart failure, acute cardiovascular care, and postoperative care following cardiac surgery.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Acknowledgements

We are grateful to Dr Gustavo Brandão for echocardiogram assessment.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

References

1. Karlson KJ, Ashraf MM, Berger RL. Rupture of the left ventricle following mitral valve replacement. *Ann Thorac Surg* 1988;**46**:590–597.
2. Kasahara H, Beran G, Mohl W. Left ventricular pseudoaneurysm following mitral valve repair. *Gen Thorac Cardiovasc Surg* 2009;**57**:221–223.
3. Treasure RL, Rainer WG, Strevey TE, Sadler TR. Intraoperative left ventricular rupture associated with mitral valve replacement. *Chest* 1974;**66**:511–514.
4. Miller DW, Johnson DD, Ivey TD. Does preservation of the posterior chordae tendineae enhances survival during mitral valve replacement? *Ann Thorac Surg* 1979;**28**:22–27.
5. Nagamine H, Date Y, Takagi T, Kawase Y. To repair or not to repair: a case report of atrioventricular groove heatoma during mitral valve surgery. *J Cardiothorac Surg* 2019;**14**:8–12.
6. Şahan E. Left ventricular pseudoaneurysm after mitral valve replacement: review of pseudoaneurysms late after mitral valve replacement. *Herz* 2015;**40**:778–782.
7. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol* 1998;**32**:557–561.
8. Ho SY. Anatomy of the mitral valve. *Heart* 2002;**88**:iv5–iv10.
9. Deniz H, Sokullu O, Sanioglu S, Sargin M, Ozay B, Ayoglu U, Aka A, Bilgen F. Risk factors for posterior ventricular rupture after mitral valve replacement: result of 2560 patients. *Eur J Cardiothorac Surg* 2008;**34**:780–784.
10. Zhang HJ, Ma WG, Xu JP, Hu SS, Zhu XD. Left ventricular rupture after mitral valve replacement: a report of 13 cases. *Asian Cardiovasc Thorac Ann* 2006;**14**:26–29.
11. Yeo TC, Malouf JF, Jae K, Seward JB. Clinical profile and outcome in 52 patients with cardiac pseudoaneurysm. *Ann Intern Med* 1998;**128**:299–305.
12. Chenier M, Krishna KP, Svensson LG, Navia J, Sabik JF, Rodriguez L, Grimm RA, Griffin BP, Desai MY. Characteristics and outcomes of patients with postoperative cardiovascular pseudoaneurysms. *J Thorac Cardiovasc Surg* 2017;**153**:43–50.
13. Tirone ED. False aneurysms after heart valve surgery. *J Thorac Cardiovasc Surg* 2017;**153**:51–52.