

Left Atrial Calcification: Case Report and Review of Literature

Ajmer Singh*, Preeti, Ravina Mukati

Department of Cardiac Anaesthesia, Medanta the Medicity, Gurugram (Haryana)-122001, India.

***Corresponding Author:** Ajmer Singh, Department of Cardiac Anaesthesia, Medanta the Medicity, Gurugram (Haryana)-122001, India

Received Date: September 21, 2026 | **Accepted Date:** October 12, 2026 | **Published Date:** October 26, 2026

Citation: Ajmer Singh, Preeti, Ravina Mukati, (2026), Left Atrial Calcification: Case Report and Review of Literature, *J Clinical Cardiology and Cardiovascular Interventions*, 9(11); DOI:10.31579/2641-0419/597

Copyright: © 2026, Ajmer Singh. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract:

Left atrial calcification is a relatively uncommon sequela of rheumatic valvular heart disease. It has been described in cases of long-standing mitral stenosis and in patients with mitral valve replacement. Left atrial calcification is generally an incidental finding on chest radiographs with features of severe pulmonary hypertension and right heart failure in the presence of a normally functioning prosthetic mitral valve. Here we report a 60-year-old man who underwent mitral valve replacement about 16 years ago and now presents with severe aortic stenosis, aortic regurgitation, tricuspid regurgitation, and coronary artery disease. This report also discusses the available diagnostic and therapeutic options.

Key words: left atrial calcification; rheumatic heart disease; multivalvular disease; echocardiography; chest x-ray

Introduction

Left atrial calcification (LAC) is a relatively uncommon complication of long-standing rheumatic valvular heart disease. Complete LAC has been described as a 'coconut atrium' or 'porcelain atrium'. [1-2] Porcelain atrium involves the LA appendage, the free wall of the LA, and the mitral valve apparatus, except the interatrial septum, whereas coconut atrium involves all areas of the LA. Porcelain or coconut atrium is a rare disease entity; furthermore, massive LAC after mitral valve replacement (MVR) has been reported in very few cases. [3] LAC is primarily a radiologic diagnosis, which explains why its detection has become more frequent with increased use of chest or cardiac computed tomography (CT) scans. The most commonly cited etiology of LAC is sequelae of remote rheumatic heart disease. [4] We encountered a 60-year-old man with massive LAC after MVR. The patient now presented with severe aortic valve disease, severe tricuspid regurgitation, and coronary artery disease. Therefore, we report this case and review the literature.

Case Report

A 60-year-old man was admitted to our hospital with angina on exertion and congestive heart failure (New York Heart Association functional class IV). The patient had a history of MVR for rheumatic mitral stenosis in 2010 with no evidence of LAC at the time of surgery. An electrocardiogram showed atrial fibrillation and biventricular hypertrophy. A posteroanterior chest radiograph revealed cardiomegaly with a near-complete curvilinear density, a shadow outlining the LA

suggestive of mural calcification, and a mechanical valve in the mitral position (Figure. 1). A chest CT scan also revealed circumferential calcification confirming the radiographic findings (Fig. 2). A transthoracic echocardiogram showed good motion of the prosthetic mitral valve leaflets, a mean pressure gradient of about 3.69 mmHg, and mild valvular regurgitation. The leaflets of the aortic and tricuspid valves were thickened. There was severe aortic stenosis (aortic valve area 0.6 cm²), severe aortic regurgitation, a dilated tricuspid annulus (6.99 cm), severe tricuspid regurgitation, and an estimated systolic pulmonary artery pressure of 65 mmHg. Coronary angiogram showed double-vessel coronary artery disease. Informed consent was obtained for aortic valve replacement, tricuspid valve repair, and coronary artery bypass grafting. Using a standard anesthetic technique and cardiac monitoring, a transesophageal echocardiography (TEE) probe was inserted (Philips CX50 machine, X7-2t probe). Preoperative echocardiographic findings of the mitral and tricuspid valves were confirmed by intraoperative TEE (Figure. 2). However, the anatomy and function of the aortic valve could not be evaluated, possibly due to the presence of dense LAC. Despite all possible manipulations of the probe, including tilting, rotating, and angulating, the TEE image of the aortic valve was suboptimal. The surgical procedure consisted of replacement of the aortic valve with a mechanical prosthesis, tricuspid valve annuloplasty with an annular ring, and double-vessel coronary artery bypass grafting using cardiopulmonary bypass. The subsequent course of the patient in the hospital was unremarkable.

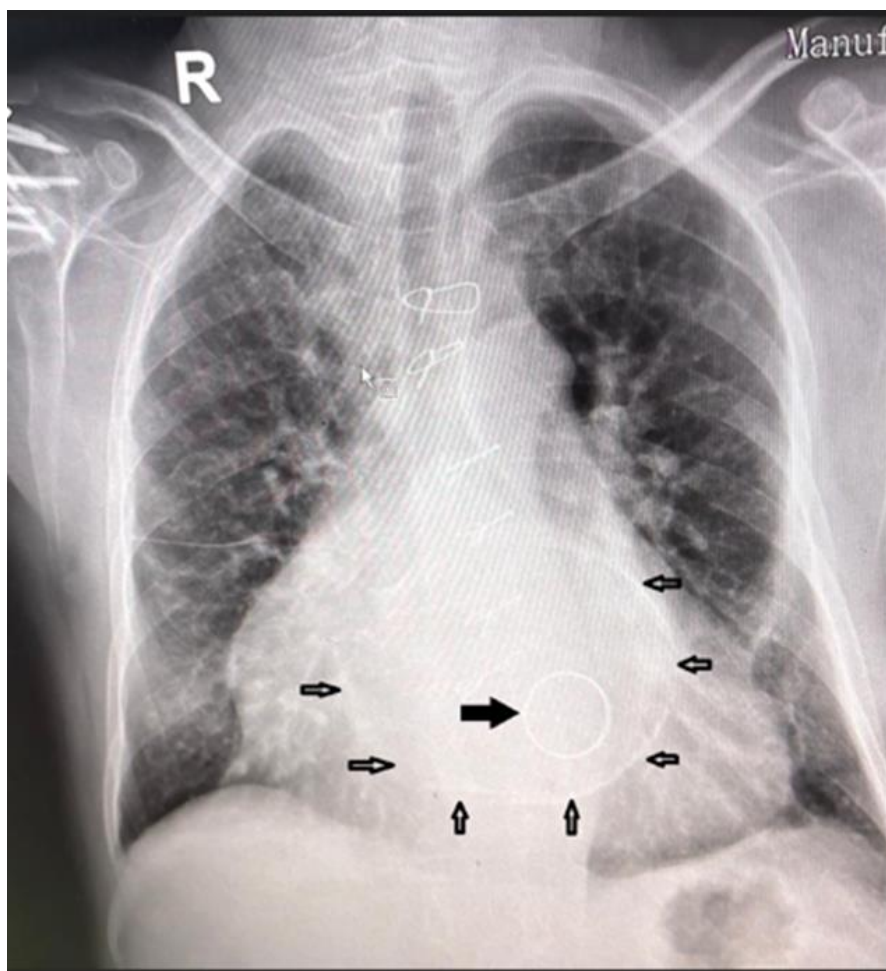


Figure 1: Chest radiograph depicting curvilinear density shadow outlining the left atrium (arrows). A prosthetic valve at the mitral position is also seen (dark black arrow).

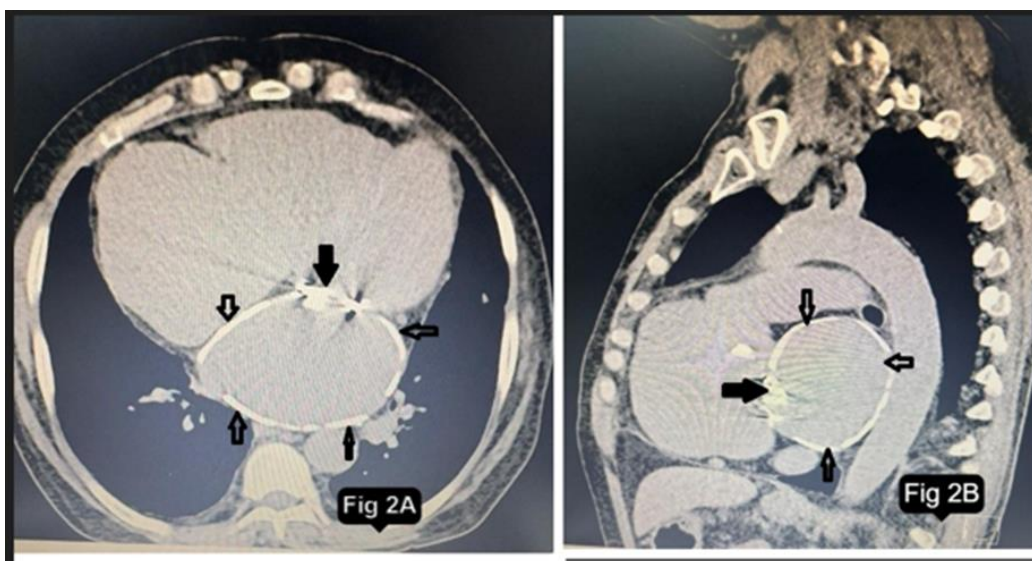


Figure 2: Computed tomography of the chest, coronal (Figure 2A) and sagittal views (Figure 2B) showing circumferential calcification of the left atrium (arrows) and mitral prosthesis (dark black arrow)

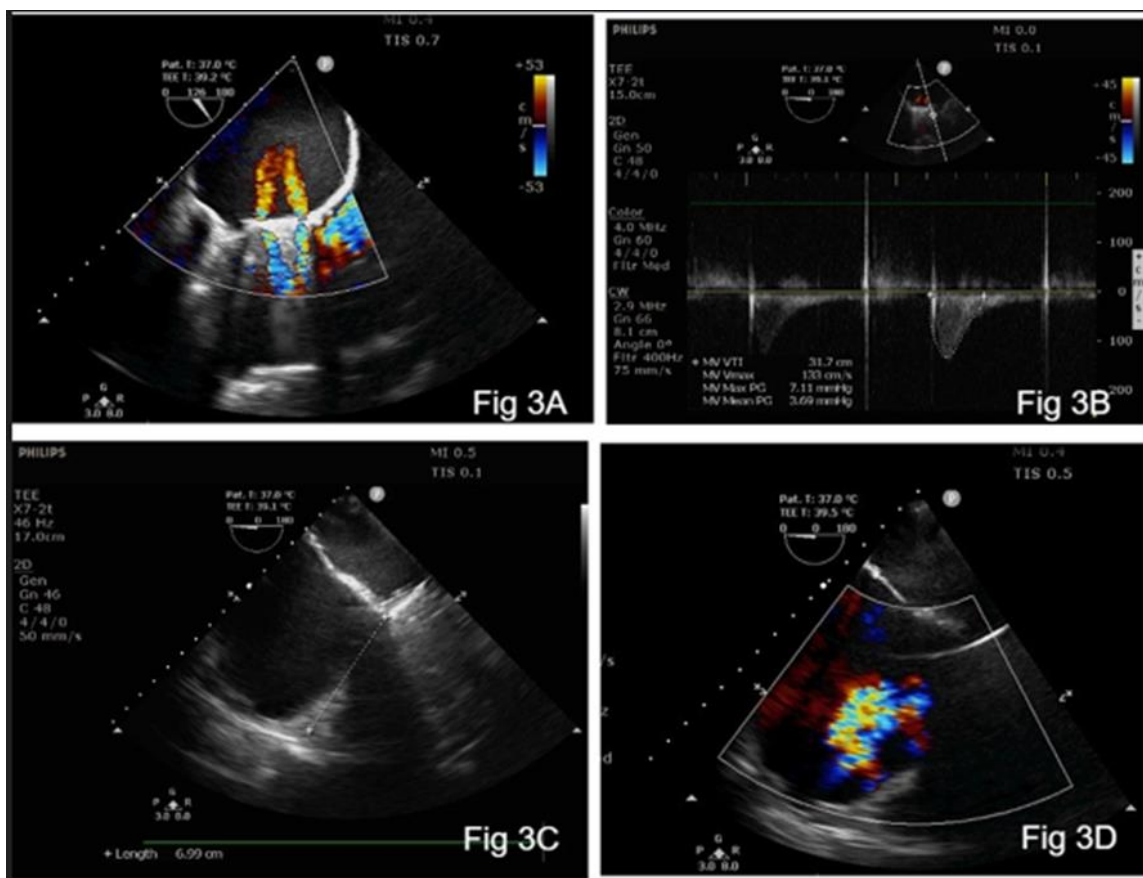


Figure 3: Transesophageal echocardiography views showing calcified left atrium, a normally functioning mitral prosthesis (Figure 3A, 3B), acoustic shadow at the aortic valve (Fig 3A), dilated tricuspid annulus (Figure 3C), and severe tricuspid regurgitation (Figure 3D)

Discussion

Left atrial calcification was first described by Oppenheimer in 1912, as an autopsy finding in a patient who died of congestive heart failure due to mitral stenosis.[5] The first X-ray of LAC, observed antemortem by Bedford, was published by Shanks and associates in 1938. [6] Leonard and co-workers reviewed 38 previous cases of LAC and added one of their own. [7] They stressed the frequency of multivalvular lesions and suggested this as a relative contraindication to surgery. Mahoney and O'Laughlin reviewed the significance of LAC and observed an average duration of disease of 29 years before the X-ray appearance of calcification, with an average duration of symptoms of 17 years. [8]

The exact etiology of LAC is not elucidated, but possible causes include mitral valve disease and MVR, end-stage renal disease, radiation therapy, and non-specific endocarditis. Atrial calcification is the end-result of extensive rheumatic auriculitis beginning with focal patches of calcification in areas of rheumatic endocardial ulceration and progressing to chronic fibrotic changes with plaques of subendocardial calcium of varying extent. LAC can predispose to or manifest cardiac conditions, including mitral valvular disease, bradyarrhythmia, and "stiff left atrial syndrome", a hemodynamic manifestation of atrial diastolic dysfunction. LAC and its associated conditions may have important clinical consequences, including sudden cardiac death, mortality after MVR, recurrent atrial fibrillation, and congestive heart failure. [9] Calcific deposits prevent the dilation of the LA, decrease its compliance, and cause the elevated LA pressure to be transmitted to the pulmonary vessels and the right ventricle, leading to tricuspid insufficiency.

The index cohort study reporting on LAC was published in 1966, in which 14 out of 15 patients had a history of rheumatic fever. [10] A recent study of MVR patients found that 24% (79 out of 327 cases in 7 years) of

patients had severe LAC, and correlated severity of calcification with incidents of major adverse cardiac events and all-cause mortality. [11] The authors also included hazard ratios for development of LAC and calculated the hazard for a history of rheumatic disease to be 3.06. Another review of the literature remarks that LAC cases are "almost always" caused by rheumatic fever at an incidence rate of 2-3%. [12] The estimated incidence of rheumatic fever in 1980 in the southeastern United States was 1-1.5 per 100,000 people annually, giving an estimated incidence of LAC (at 2-3%) of 0.03 per 100,000. [13]

Chest radiograph is the primary modality for the diagnosis of LAC. Mural calcification appears as a thin, curvilinear density tracing the outline of the LA in part or completely. In the frontal projection, a round or oval shell of calcium 8 to 10 cm in diameter is usually seen lying in the center of the cardiac silhouette. In lateral or oblique projections, a completely calcified wall appears as a C-shaped curvilinear density with the opening of the C lying anteriorly in the region of the mitral annulus. Occasionally, the calcification extends into the pulmonary veins. LAC must be differentiated from calcification occurring in the cardiac valves, pericardium, coronary arteries, ventricular aneurysms or infarctions, intracardiac tumors, mediastinal cysts, hilar nodes, and costal cartilages. [10] All of these have characteristic locations and motion, and present little difficulty to the experienced observer. Fluoroscopy is superior to plain radiographs for the detection of LA calcification, but inferior to CT chest. Multidetector CT may provide detailed three-dimensional visualization of the LA calcification with high resolution.

The suboptimal imaging of the aortic valve observed in this patient has not been described previously. It was assumed that the extensive calcification of the LA wall and the presence of a mechanical prosthesis at the mitral position made visualizing the aortic valve difficult. The acoustic density of the atrial wall can make visualization of the LA, LA

appendage, and the aorta difficult. TEE imaging is significantly impaired in the presence of calcified walls of the LA. Such patients may need a CT scan or magnetic resonance imaging for better visualization of intracardiac structures and thrombi. The Shaw classification divides these LACs into three groups. In type A, the calcifications are restricted to the LA appendage. In type B, all areas of the left atrium are affected (as observed in this patient). Type C corresponds to a MacCallum's patch, a very rare focal calcification on the posterior atrial wall caused by a regurgitation jet from a thickened mitral valve [14].

During mitral valve surgery, considerable difficulty may be encountered in entering the LA because of marked thickening and calcification of the LA wall. During closed mitral valvulotomy, entry into the LA can be accomplished by blunt dissection in the region of the LA appendage. Hemorrhage at the site of atriotomy may require a pericardial patch. For open mitral repair/replacement, the surgery can be approached through a right thoracotomy, atrial septum, or the posterior interatrial groove. The mitral valve can be approached through a noncalcified portion of the wall adjacent to the septum, which is generally free of calcification despite extensive involvement of the remainder of the chamber. The presence of associated valvular lesions will dictate which approach is preferable. The surgical approach to the patient with LAC must be individualized according to the overall clinical, hemodynamic, and radiologic situation. Surgical removal of the internal cast of calcium has been described but has an operative mortality of up to 25%, which is related to the difficulty of entering the LA, potential embolization, and impaired hemostasis. [15] A complete endoatriectomy of the LA wall and the interatrial septum followed by MVR has been reported in a patient with coconut LA. [16]

In conclusion, LAC is a rare but increasingly reported complication of long-standing rheumatic valvular heart disease. It should be suspected in patients with rheumatic heart disease with severe mitral stenosis or post-MVR who present with features of right heart failure. LAC constitutes a major complication and risk to mitral valve surgery due to difficulty in entering the LA, potential embolization, and impaired hemostasis. The condition can be diagnosed by a variety of radiologic investigations. Echocardiographic imaging may yield suboptimal results due to excessive calcification. Surgical approach through a right thoracotomy, posterior interatrial groove, or atrial septum must be tailored to the individual patient.

References

1. Del Campo C, Weinstein P, Kunnelis C, DiStefano P, Ebers GM (2000). Coconut atrium: transmural calcification of the entire left atrium. *Tex Heart Inst J* 27 (1):49-51
2. Edwards JM, Chisholm RJ. Porcelain atrium: rheumatic heart disease (2006). *Can J Cardiol* 22 (3):267
3. Lin YS, Tsai FC, Chu PH (2008). Massive left atrial and interatrial septal calcification after mitral valve replacement. *Chin Med J* 121 (15):1497-1499
4. Chamberlin JH, Smith CD, Gowani Z, et al. (2023). Left atrial calcification on chest CT: atrial ablation replaces rheumatic heart disease as the most identified etiology. *Pol J Radiol* 14; 88: e423-e429
5. Oppenheimer BS (1912). Calcification and osteogenic change of the left auricle in a case of auricular fibrillation. *Proc N Y Path Soc (N.S.)* 12: 213-215
6. Shanks SC, Kerley P, Twining EW (editors) 1938: *A Textbook of X-Ray Diagnosis* by British Authors, vol. 1. London, H. K. Lewis & Co., Ltd., pp 47
7. Leonard JJ, Katz S, Nelson D (1957): Calcification of the left atrium: Its anatomic location, diagnostic significance and roentgenologic demonstration. *New Eng J Med* 256: 629
8. Mahoney PS, O'Laughlin BJ (1976): Significance of calcification of the wall of the left atrium. *Radiology* 73: 402
9. Selleck C, Noel T, Hartlage G, et al. (2019). When hindsight is 20/20: stiff left atrium syndrome masquerading as mitral regurgitation. *JACC Case Rep* 1: 648-651
10. Harthorne JW, Seltzer RA, Austen WG (1966). Left atrial calcification. *Circulation* 34: 198-210
11. Choi JY, Suh YJ, Kim YJ, et al (2020). Characteristics and implications of left atrial calcium on cardiac computed tomography in patients with earlier mitral valve operation. *Am J Cardiol* 128: 60-66
12. Ferguson EC, Berkowitz EA (2010). Cardiac and pericardial calcifications on chest radiographs. *Clin Radiol* 65: 685-694
13. de Loizaga SR, Arthur L, Arya B, et al. (2021). Rheumatic heart disease in the United States: forgotten but not gone: results of a 10 year multicenter review. *J Am Heart Assoc* 10: e020992
14. Shaw DR, Chen JT, Lester RG (1976). X-ray appearance and clinical significance of left atrial wall calcification. *Invest Radiol* 11(6):501-517
15. Leung EC, Hirani N. Pulmonary hypertension caused by a coconut left atrium (2020). *Can J Cardiol* 36 (10): 1691.e1-1691.e2
16. Shrivastava S, Shrivastava S, Gopalamurugan AB, et al. (2026). Complete Endoatriectomy of Coconut Left Atrium with Mitral Valve Replacement and Tricuspid Repair. *Journal of the Heart Valve Society* 3 (1):57-60



This work is licensed under Creative Commons Attribution 4.0 License

To Submit Your Article Click Here:

Submit Manuscript

DOI: [10.31579/2641-0419/597](https://doi.org/10.31579/2641-0419/597)

Ready to submit your research? Choose Auctores and benefit from:

- fast, convenient online submission
- rigorous peer review by experienced research in your field
- rapid publication on acceptance
- authors retain copyrights
- unique DOI for all articles
- immediate, unrestricted online access

At Auctores, research is always in progress.

Learn more <https://auctoresonline.org/journals/clinical-cardiology-and-cardiovascular-interventions>