

Approach to left atrial appendage aneurysm in asymptomatic individuals

Description

The presented case describes a 48-year-old woman with persistent, asymptomatic Atrial Fibrillation (AF) in whom an incidental finding of a Large Left Atrial Appendage Aneurysm (LAAA) was discovered. The report contributes meaningfully to the limited body of literature on LAAA, particularly in asymptomatic adults, and raises key considerations regarding diagnosis, pathophysiology, and management strategies.

Clinical presentation and diagnostic approach

The index finding in this young patient was asymptomatic AF of several years' duration, which, in the absence of comorbidities, represents an uncommon clinical scenario. While AF is generally associated with aging, hypertension, or structural heart disease, the lack of these predisposing factors appropriately prompted further imaging. This underscores the importance of the recommendation that younger patients with early forms of AF should undergo a thorough assessment, including detailed family history and anatomical and functional evaluation to exclude congenital or structural causes [1,2].

According to current literature the condition has been reported across all age's groups. Most diagnoses are established during the third decade of life, when symptom onset or thromboembolic events lead to thoracic imaging studies [3,4]. No significant differences in the prevalence of LAAA have been observed between males and females; either in adults, or in pediatric populations [5].

Symptoms are present in up to 75% of patients, with palpitations and dyspnea being the most common [6]. However, as in this patient, LAAA could also be asymptomatic. It is often difficult to determine whether symptoms are attributable to the aneurysm itself or to coexisting cardiac disorder such as atrial arrhythmia. Moreover, more than 50% of pediatric patients also appear to be symptomatic.

Since its first description by Dimond et al. in 1960, X-ray (typically showing enlargement of the left heart border [7]), angiography, and surgical exploration were the only available diagnostic modalities prior to the advent of echocardiography. Currently, Transthoracic Echocardiography (TTE) plays a key role in the diagnosis of this entity. In the present case, Doppler imaging confirmed flow within the structure, supporting its vascular nature and ruling out pericardial cysts. However, case series have reported a diagnostic accuracy of 45%-65% for TTE. Transesophageal echocardiography could increase accuracy up to 90%, with high sensitivity for thrombus detection.

The use of multimodal imaging is consistent with best practice. Most patients undergo

Javier Cantalapiedra-Romero*

Department of Cardiology, University Hospital Arnau Vilanova, Lleida, Spain

*Author for correspondence:
Javier Cantalapiedra-Romero, Department of Cardiology,
University Hospital Arnau Vilanova, Lleida, Spain, E-mail:
jcantalapiedraromero@gmail.com

Received date: 27-Oct-2025, Manuscript No. FMIC-25-172394;
Editor assigned: 29-Oct-2025, PreQC No. FMIC-25-172394 (PQ);
Reviewed date: 12-Nov-2025, QC No. FMIC-25-172394;
Revised date: 19-Nov-2025, Manuscript No. FMIC-25-172394 (R);
Published date: 26-Nov-2025, DOI: 10.37532/1755-5310.2025.17(4).1024

cardiac Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) not only to confirm the diagnosis but also to delineate the aneurysm's relationship with surrounding structures information that is critical for surgical planning when indicated. A strong positive correlation has been observed between aneurysm measurements obtained by CT/MRI and intraoperative findings. In addition, CT imaging can provide valuable information on coronary anatomy, as in this case, where the subsequent CT confirmed aneurysm's dimensions (8 × 5 cm) and excluded coronary lesions.

Pathophysiology and etiology

As the authors note, approximately 90% of LAAA cases are congenital, attributed to dysplasia or weakness of the pectinate muscles leading to progressive dilatation under hemodynamic stress. Other congenital anomalies may coexist in about 12% of patients, including atrial septal defect, ventricular septal defect and Noonan syndrome; as previously described. The remaining cases are acquired and are typically associated with elevated left atrial pressures or structural alterations secondary to mitral valve disease or pericardial defects. In the present case, the absence of mitral pathology supports a congenital origin, with no additional abnormalities identified.

From a mechanistic standpoint, the enlarged and dyskinetic left atrial appendage predisposes to blood flow stasis, explaining the frequent coexistence of atrial arrhythmias (such as atrial fibrillation) and the increased risk of thromboembolism. However, aneurysm size has not been shown to predict thromboembolic events on multivariate analysis. In a multivariate analysis published by Daralammouri, et al. [4], presence of arrhythmia was significantly associated with older age, male sex, and the presence of thrombus within LAAA. In this patient, none of these factors were present, yet the absence of other atrial abnormalities on imaging suggests a causal relationship, supporting the hypothesis that the LAAA contributed to arrhythmogenesis.

Therapeutic considerations

After exclusion of intracardiac thrombi, electrical cardioversion was attempted but was unsuccessful, consistent with reports indicating that rhythm control is often ineffective in patients with significant atrial structural abnormalities. In cases of atrial flutter, catheter ablation may be considered [8]. However, in this patient, the long-standing nature of atrial fibrillation and the absence of symptoms precluded more aggressive interventions. Consequently, a rate-control strategy was adopted, with no pharmacological therapy required to maintain adequate ventricular response. This approach aligns with current AF management paradigms but raises the broader issue of optimal treatment in asymptomatic patients with LAAA.

In this context, the conventional CHA2DS2-VA score was deemed inappropriate given the presence of structural heart disease. The occurrence of arrhythmias in patients with LAAA may indicate a more advanced disease phenotype and increased risk of thromboembolic complications. Therefore, oral anticoagulation with dabigatran was initiated to mitigate the medium-to-long-term thromboembolic risk.

Surgical resection remains the standard of care in most centers particularly in symptomatic patients or those with compressing manifestations, although successful procedures have also been reported in asymptomatic individuals [9]. Aneurysmectomy effectively prevents potential complications, and published series suggest that surgical outcomes are excellent, with low perioperative morbidity. Depending on LAAA size and anatomy, resection may be performed *via* median sternotomy, left thoracotomy or minimally invasive thoracoscopic approaches. In selected cases, concomitant surgical AF ablation may also be undertaken. Catheter-based occlusion has also been described as an alternative in patients deemed inoperable [10].

However, as the authors correctly note, no comparative studies have evaluated surgical versus conservative management in asymptomatic individuals, and no data are available regarding the prevalence of spontaneous rupture. Prior autopsy series predominantly described male patients with acquired forms of the disease and intraluminal thrombi, but no evidence of rupture [11].

For these reasons, in asymptomatic patients with isolated congenital LAAA, conservative management with anticoagulation as chosen in this case represents a reasonable and patient-centered approach. Regular imaging follow-up is recommended to monitor aneurysm size and detect potential complications, although optimal surveillance intervals remain undefined due to the rarity of this condition.

Future directions

Several gaps in knowledge remain. Given the rarity of this entity, there is currently no consensus on the definition or diagnostic criteria for LAAA. Furthermore, longitudinal data are lacking, and risk stratification of asymptomatic patients has yet to be systematically evaluated.

References

1. Van Gelder IC, Kotecha D, Rienstra M, et al. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J.* 45(36):3314-414 (2024).
2. Cuenca Castillo J. Left atrial appendage aneurysm: Knowledge and gaps. *JACC Case Reports* [Internet]. 3(18):1930-1931 (2021).
3. Ayala Torres JD, Sepulveda Gallego JA, Gonzalez Gonzalez M. Left atrial appendage aneurysm: A case report and literature review. *Cureus.* 16(3) (2024).

Short Communication

4. Daralammouri Y, Odeh A, Abuzahra S, et al. Left atrial appendage aneurysm: A descriptive systematic review of 177 cases. *BMC Cardiovasc Disord.* 24(1):633 (2024).
5. Norozi K, Subasri M, Díaz LA, et al. Left atrial appendage aneurysm in pediatrics: Case study and literature review. *Front Cardiovasc Med* [Internet].10:1-5 (2023).
6. Aryal MR, Hakim FA, Ghimire S, et al. Left atrial appendage aneurysm: A systematic review of 82 cases. *Echocardiography.* 31(10):1312-1318 (2014).
7. Gray R, Magdy J, Cheruvu C, et al. Multimodality imaging and surgical management of a left atrial appendage aneurysm. *Eur Heart J Cardiovasc Imaging* [Internet]. 23(10):E468 (2022).
8. Aksu T, Mutluer FO, Cabbar AT, et al. A giant left atrial appendage aneurysm with left atrial flutter: feasibility of catheter ablation strategy. *J Interv Card Electrophysiol* [Internet]. 66(8):1765-1768 (2023).
9. Chowdhury UK, Seth S, Govindappa R, et al. Congenital left atrial appendage aneurysm: A case report and brief review of literature. *Hear Lung Circ.* 18(6):412-416 (2009).
10. Kothandam S, Ramasamy R. Planning and execution of catheter closure of a giant left atrial appendage aneurysm causing recurrent cardioembolism. *Ann Pediatr Cardiol.* 13(4):353-356 (2020).
11. Fnon NF, Sharif AF, Sobh ZK. The lethal fifth cardiac chamber: A rare autopsy case report of left atrial appendage aneurysm and review of literature. *Forensic Sci Med Pathol.* 1299-1306 (2025).