

Caught in the flow: a case report of large left atrial thrombus obstructing the mitral valve in atrial fibrillation

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Abstract

Background: Large, mobile left atrial thrombi can intermittently occlude the mitral orifice and precipitate rapid hemodynamic compromise in atrial fibrillation. Timely echocardiography is pivotal to diagnosis and management.

Case presentation: A 71-year-old woman with previously undocumented atrial fibrillation presented with dyspnea, rapid ventricular response, and borderline blood pressure (100/50 mmHg). Transthoracic echocardiography showed a large, mobile left atrial mass prolapsing through the mitral valve; transesophageal echocardiography confirmed a 3.3×2.8 cm thrombus with spontaneous echo contrast and 2 smaller septal thrombi. Mitral leaflets were thickened and calcified with commissural fusion, consistent with rheumatic disease; the transmitral gradient was mild, but biatrial enlargement suggested chronic pathology. Given thrombus size and mobility, anticoagulation and thrombolysis were deferred. The patient underwent emergency surgical thrombectomy, bioprosthetic mitral valve replacement, and left atrial appendage exclusion. Recovery was uneventful, with normal prosthetic function and no residual thrombus on follow-up imaging.

Conclusion: In atrial fibrillation with suspected mechanical obstruction, bedside transthoracic echocardiography followed by transesophageal echocardiography enables rapid confirmation of a ball-valve left atrial thrombus. In this patient, rapid echocardiographic characterization and multidisciplinary assessment supported urgent surgery because of intermittent mechanical obstruction, marked thrombus mobility, associated mitral-valve disease, and the need for definitive treatment. Management should be individualized; this single case does not establish the superiority of surgery over anticoagulation for all large left atrial thrombi.

Keywords: Atrial fibrillation, Case report, Echocardiography, Left atrial thrombus, Mitral stenosis

Introduction

Atrial fibrillation (AF) is one of the most common cardiac arrhythmias, particularly in the elderly, and significantly increases the risk of thromboembolic events due to left atrial thrombus formation.^[1] Several factors, including atrial enlargement, mitral stenosis, and heart failure, further contribute to this risk.^[2] While stroke and systemic embolization are the most frequent complications, a less common but serious consequence is the development of a large, mobile thrombus in the left atrium (LA), which can mechanically obstruct the mitral valve.^[3]

The datasets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Thrombus formation in AF is primarily driven by impaired atrial contraction and blood stasis, with the left atrial appendage being the most typical site of thrombus development. However, in rare cases, thrombi may form in the LA body, as seen in the present case, where atrial enlargement due to mitral stenosis and AF created a hemodynamic environment favorable for thrombus formation outside the usual location.

This phenomenon, sometimes referred to as a “ball-valve” thrombus,^[4] can severely compromise hemodynamics by obstructing blood flow from the LA to the left ventricle.^[5] Dynamic obstruction of the mitral orifice by such thrombi can result in abrupt hemodynamic deterioration, pulmonary edema, or cardiogenic shock. In the emergency department, rapid identification of this condition is crucial, as inappropriate initial management (eg, anticoagulation or thrombolysis) may precipitate systemic embolization or sudden circulatory collapse.

Point-of-care echocardiography, particularly transesophageal echocardiography (TEE), is a key diagnostic tool in this context, providing real-time assessment of thrombus morphology, mobility, and hemodynamic impact. Early collaboration between emergency physicians, intensivists, cardiologists, and cardiac surgeons is essential to facilitate life-saving surgical intervention to prevent fatal outcomes.^[6]

Case report

A 71-year-old obese woman presented to the emergency department with a 48-hour history of progressively worsening dyspnea. She reported increasing difficulty breathing, particularly

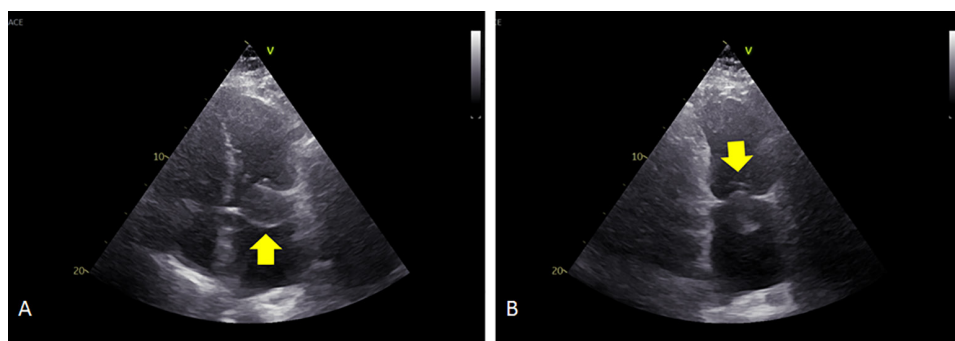


Figure 1. Transthoracic echocardiography in 4-chamber apical view (A) and 2-chamber view (B) showing a thrombus occupying the mitral orifice (arrow).

on exertion, but denied chest pain or syncope. Her medical history included hypertension and type 2 diabetes mellitus, with no prior documented episodes of arrhythmia or other significant cardiac conditions. On arrival, she was in moderate respiratory distress. Her vital signs included a blood pressure of 100/50 mm Hg, a heart rate of 170 beats per minute, a respiratory rate of 24 breaths per minute, a body temperature of 36 °C, and an oxygen saturation of 90% on room air. Physical examination revealed an irregular heart rhythm and bilateral lower extremity edema, though lung auscultation was unremarkable.

An initial 12-lead electrocardiogram showed AF with a rapid ventricular response, with a mean heart rate of 168 beats per minute but no signs of acute ischemia. Given her symptoms and hemodynamic status, transthoracic echocardiography (TTE) was performed to identify potential causes. The study revealed a preserved left ventricular ejection fraction of 60% along with significant left and right atrial enlargement. TTE revealed thickened and calcified mitral valve leaflets with commissural fusion, consistent with rheumatic mitral valve disease. Because the examination was performed during atrial fibrillation with rapid ventricular response and intermittent occupation of the mitral orifice by the mobile thrombus, reliable quantitative grading of the valve lesion was not possible. Intraoperative inspection demonstrated extensive fibrotic and calcific leaflet involvement with commissural fusion. Because the valve was considered unsuitable for durable repair and urgent surgical thrombectomy was required, mitral valve replacement was performed. The most striking finding was a large, mobile, oval-shaped hypoechoic mass measuring 3.1×2.7 cm in the LA, intermittently obstructing the mitral valve (Fig. 1). To further characterize the mass, a TEE was performed, confirming the presence of a highly mobile 3.3×2.8 cm mass continuously occupying the mitral valve orifice (Fig. 2A),

raising immediate concern for mechanical mitral valve obstruction. Additionally, 2 smaller masses, measuring 1.5×1.08 cm and 1.0×1.3 cm, were observed attached to the interatrial septum, while spontaneous echo contrast was noted in the LA (Fig. 2). A small, consolidated mass was also identified in the left atrial appendage.

Atrial myxoma and infective endocarditis were considered in the differential diagnosis due to the presence of an intracardiac mass and the patient's clinical presentation. However, atrial myxomas typically have a gelatinous, heterogeneous appearance and are most often attached to the interatrial septum rather than freely mobile in the mitral orifice. Infective endocarditis was deemed unlikely given the absence of fever, negative inflammatory markers, and echocardiographic findings that lacked typical vegetations or valve destruction.

On admission, the patient's rapid ventricular response was contributing to her worsening dyspnea, and initial management focused on stabilizing her heart rate and hemodynamic status. Intravenous metoprolol 5 mg was given under continuous blood-pressure and rhythm monitoring to control the rapid ventricular response. Heart rate decreased from 170 to 115 beats/min without adverse hemodynamic response. Because of the large, highly mobile left atrial thrombus intermittently occluding the mitral valve, anticoagulation and thrombolysis were contraindicated due to the imminent embolic risk. Therefore, the patient underwent emergency surgical thrombectomy and mitral valve replacement with a bioprosthesis.

She underwent urgent mitral valve replacement with a biological prosthesis, along with complete removal of the large atrial thrombus and the smaller masses attached to the interatrial septum (Fig. 3). Preoperative laboratory data, including platelet count, prothrombin time/international normalized ratio,

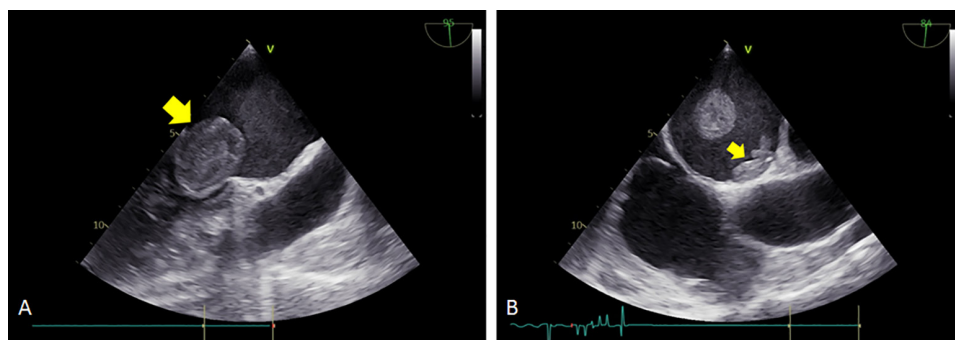


Figure 2. Transesophageal echocardiography showing a large thrombus occupying the (A) mitral orifice and (B) smaller thrombi.

activated partial thromboplastin time, fibrinogen, and D-dimer, were all within normal limits, and there was no personal or family history of thrombophilia. To further reduce the risk of future thromboembolic events, left atrial appendage closure was performed, given its role as a common site for thrombus formation in AF. The decision to proceed with valve replacement rather than repair was based on the severity of mitral valve calcification and the patient’s age. A biological prosthesis was chosen to minimize thromboembolic risk while ensuring durable valve function. The Maze procedure, which could have been considered for rhythm control, was not performed due to the patient’s hemodynamic instability and the need for rapid intervention. The surgery proceeded without complications, resulting in the complete removal of the thrombi and the successful implantation of the mitral prosthesis. Histopathology of the excised mass showed layered fibrin, red cells, and platelets without myxomatous cells or vascular cores, confirming thrombus rather than tumor even if the absence of a stalk on TEE and the echolucent, hypoechoic homogeneous appearance further argued against myxoma.

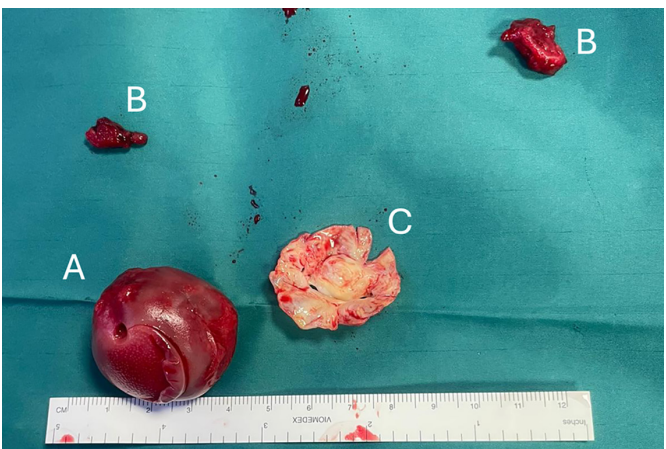


Figure 3. Surgical specimen. (A) Large thrombus. (B) Small thrombi adhered to the inferior interatrial septum. (C) Heavily fibrotic and stenosed mitral valve.

Intraoperatively, the mitral valve showed thickened fibrotic leaflets with commissural fusion and dense calcification of the posterior leaflet consistent with chronic rheumatic mitral stenosis. The pathology report confirmed fibrous pannus formation and focal calcification without inflammatory infiltrates or vegetations.

The postoperative course was uneventful. The patient was extubated 8 hours after surgery and remained hemodynamically stable in the intensive care unit, where intravenous medications were gradually discontinued. A TTE performed on postoperative day 7 showed no residual intracardiac masses and satisfactory function of the bioprosthesis. At 3-month follow-up, TEE confirmed complete exclusion of the LAA with no residual stump thrombus. She was transitioned from intravenous to oral therapy, including anticoagulation, to mitigate the risk of future thrombus formation. Her recovery was smooth, with no recurrence of arrhythmias or complications during hospitalization, and her respiratory symptoms significantly improved.

Postoperative anticoagulation consisted of rivaroxaban 20 mg once daily, initiated on postoperative day 8 after adequate surgical hemostasis had been confirmed. Renal function was preserved, with a creatinine clearance above 50 mL/min, and no major postoperative bleeding occurred. Although a vitamin K antagonist was considered, rivaroxaban was selected after multidisciplinary assessment because the native stenotic valve had been replaced with a bioprosthesis, the patient had an acceptable bleeding risk, and reliable INR monitoring was expected to be difficult. Anticoagulation was planned indefinitely because of persistent atrial fibrillation, the previous large left atrial thrombus, and a CHA₂DS₂-VASc score of 4. Her long-term follow-up plan involved regular visits with the cardiology and cardiothoracic surgery teams to monitor her prosthetic mitral valve function and overall cardiac health. At the 3-month follow-up, she reported significant improvement in breathing and functional capacity, with no thromboembolic events. A 12-lead electrocardiogram documented persistent atrial fibrillation with a controlled ventricular response. TEE showed satisfactory prosthetic function, complete left atrial appendage exclusion, and no residual thrombus (Table 1). She remained on anticoagulation therapy without complications

Table 1.

Time	Clinical event and documented findings
48 hours before admission	Progressive exertional dyspnea
Admission, day 0	Blood pressure 100/50 mmHg; 12-lead ECG showed atrial fibrillation with rapid ventricular response, approximately 170 beats/min
Day 0	TTE demonstrated rheumatic mitral-valve morphology and a large mobile left atrial mass intermittently obstructing the mitral orifice
Day 0	TEE confirmed a 3.3 × 2.8 cm mobile thrombus, two smaller septal thrombi, spontaneous echo contrast, and a small LAA thrombus
Day 0	Intravenous rate-control treatment was administered under continuous blood-pressure and rhythm monitoring
Day 0	Multidisciplinary assessment followed by urgent thrombectomy, bioprosthetic mitral valve replacement, and surgical LAA exclusion
Postoperative period	Histopathology confirmed thrombotic material and rheumatic fibrotic-calcific mitral-valve disease
Postoperative day 7	TTE showed satisfactory bioprosthetic valve function and no residual intracardiac masses; telemetry showed rate-controlled AF
Postoperative day 8	Discharged on rivaroxaban and beta-blocker therapy; 12-lead ECG documented AF with a controlled ventricular response
Three months	Clinical assessment and 12-lead ECG documented rate-controlled AF; TEE showed satisfactory prosthetic function, complete LAA exclusion, and no residual thrombus

and continued to be free from major adverse events or hospital readmissions following surgery.

Discussion

This case illustrates several key considerations in the emergency management of large LA thrombi causing mechanical mitral obstruction. The patient's acute presentation with dyspnea, along with the finding of a significant left atrial mass on echocardiography, underscores the importance of rapid diagnosis and intervention in such cases.

AF is characterized by chaotic electrical activity in the atria, impairing effective atrial contraction and promoting thrombus formation, particularly in the left atrial appendage and body.^[7] The large mobile thrombus intermittently obstructed the mitral orifice and was associated with worsening dyspnea, rapid ventricular response, and borderline hemodynamics. Both TTE and TEE were critical in identifying the thrombus and assessing its mobility and impact on mitral valve function. TEE, in particular, offers superior imaging of the LA and appendage,^[8] providing a more accurate assessment of thrombus size, location, and potential impact on cardiac function.

This case illustrates several unique features. First, the patient had no prior history of diagnosed AF or overt rheumatic heart disease. The episode of previously undocumented AF with rapid ventricular response likely unmasked long-standing but compensated rheumatic mitral stenosis, creating hemodynamic conditions conducive to rapid thrombus formation. This underscores that even a first recognized episode of AF in the setting of subclinical mitral valve disease can precipitate severe, life-threatening complications.

Second, the location and morphology of the thrombus were unusual. Although the LAA is the most common site of thrombus formation in AF, in this case, the main body of the LA harbored the largest thrombus, with only a small, consolidated mass in the appendage. This likely reflects complex intra-atrial flow dynamics caused by severe atrial dilation and mitral stenosis.^[9] Severe atrial dilation creates zones of stasis, while a high-velocity jet through the stenotic mitral valve induces turbulence and endothelial injury. This combination likely promoted thrombogenesis in the atrial body. In such cases, the atrial body may become a zone of relative stasis, while preserved flow within the LAA may paradoxically protect it from large thrombus formation.

It is worth noting that mitral stenosis plays a significant role in left atrial thrombus formation in patients with AF. The narrowing of the mitral valve restricts blood flow from the LA to the left ventricle, increasing atrial pressure and leading to enlargement. This dilation promotes blood stasis in the LA, particularly in the appendage, a common site for thrombus formation. AF exacerbates this process by causing disorganized atrial contraction, reducing effective blood flow and enhancing clot formation. The combination of mitral stenosis and AF creates a hemodynamic environment conducive to thrombus development, increasing the risk of thromboembolic events such as stroke and systemic embolism.

Although the LAA is the predominant site for thrombus in AF, local flow dynamics can favor thrombus formation in the atrial body. In our patient, the coexistence of a markedly dilated LA, generating zones of profound flow stasis, and a high-velocity jet across the stenotic mitral valve likely produced recirculation vortices and localized endothelial injury. This synergistic milieu may

favor thrombus formation within the atrial body rather than the appendage.^[10, 11]

Third, our case illustrates the critical limitations of anticoagulation and thrombolysis in this setting. Although anticoagulation is the cornerstone of stroke prevention in AF, the large, highly mobile thrombus posed an immediate embolic risk if subjected to anticoagulation or lysis. Because of the thrombus size, marked mobility, intermittent mechanical obstruction, concomitant mitral-valve disease, and immediate surgical availability, the multidisciplinary team selected urgent surgery rather than initial anticoagulation or thrombolysis. This case-specific decision should not be interpreted as a general contraindication to anticoagulation for left atrial thrombus. Thus, urgent surgery was deemed the safest intervention. Percutaneous thrombectomy has been explored in selected cases, but its role remains investigational and was not deemed safe or feasible in this patient given the large thrombus burden, its mobility, and the need for simultaneous mitral valve replacement. Thus, urgent surgical thrombectomy remained the definitive and safest intervention.

The presence of a large, mobile thrombus posed a significant risk for systemic embolization, making anticoagulation or thrombolytic therapy extremely hazardous. Urgent surgical intervention—mitral valve replacement and thrombectomy—was essential to prevent embolic events and resolve the mechanical obstruction. Surgery not only addressed the obstruction but also removed the thrombus and additional masses that could have led to further complications.

The choice of a biological prosthesis for mitral valve replacement was appropriate, considering the patient's age and the need for a valve that would minimize thrombosis risk and provide durable function. The successful removal of the thrombus and additional masses during surgery alleviated the immediate obstruction and relieved the patient's symptoms.

Postoperative care focused on monitoring for complications, managing anticoagulation therapy, and ensuring proper valve function. Given the bioprosthetic valve and excluded left atrial appendage, catheter ablation may be considered in the future to achieve rhythm control, reduce AF burden, and potentially lower long-term thromboembolic risk. Long-term follow-up is essential to maintain anticoagulation, monitor for AF recurrence, and assess prosthetic valve function.

Conclusion

This case illustrates a rare and life-threatening manifestation of atrial thrombosis, where a large, mobile thrombus caused acute mechanical mitral obstruction. Timely echocardiographic evaluation and coordinated multidisciplinary management were crucial for diagnosis and prompt surgical treatment. This case highlights the value of prompt TTE and TEE in characterizing a large mobile left atrial thrombus with intermittent mitral obstruction. Urgent surgical thrombectomy with valve surgery was successful in this patient, but treatment selection should be individualized according to thrombus morphology, embolic and obstructive risk, valve pathology, hemodynamic status, and local expertise. It also highlights the importance of rapid recognition and individualized decision-making to prevent catastrophic embolic complications.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Cacciapuoti F conceived the study and drafted the manuscript. Cacciapuoti F, Gottilla R, Crispo S, Caso I, and Pirozzi G collected and interpreted clinical data. Torella M and Santo LD performed surgical intervention and contributed to manuscript revision. All authors approved the final version.

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Ethical approval of studies and informed consent

The study followed the principles of the Declaration of Helsinki as revised in 2013. According to the guidelines of the Ethics Committee of Antonio Cardarelli Hospital, publication of single case reports is exempt from formal ethics approval. Written informed consent for publication was obtained from the patient.

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