A giant and double-walled left atrial ball thrombus complicating a mitral stenosis: case report—a truck tire into the heart

Adelina Selimi 1,2†, Umberto Ianni 3, Michela Molisana 3, and Vito Maurizio Parato 3,2*†

1Cardiology and Arrhythmology Clinic, University Hospital 'Umberto I-Lancisi-Salesi', Via Conca 71, 60126 Ancona, Italy; 2Politecnica delle Marche University, School of Medicine, Via Tronto 10, 60126 Ancona, Italy; and 3Cardiology and Cardiac Rehabilitation Unit, Madonna del Soccorso Hospital, Via Luciano Manara 8, 63074 San Benedetto del Tronto, Italy

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Background
High thromboembolic risk associated with rheumatic mitral stenosis has been well established, especially in concomitant atrial fibrillation; however, the presence of left atrium ball thrombus is an uncommon finding.

Case Summary
A 75-year-old woman with a history of mild rheumatic mitral stenosis was admitted to Emergency Department with cardiogenic shock and high ventricular rate atrial fibrillation. Emergency electrical cardioversion was performed—before trans-oesophageal echocardiography (TOE)—due to haemodynamic deterioration which restored sinus rhythm.

Transthoracic echocardiography (TTE) revealed severe reduction of left ventricular ejection fraction (LVEF: 15%), severe rheumatic mitral stenosis and a large, perfectly rounded mass, situated at the ostium of left upper pulmonary vein. Due to the persistence of haemodynamic instability and acute pulmonary oedema the patient was intubated and mechanically ventilated and treated with intravenous administration of inotropes and high doses of diuretics. During the stay in Intensive Care Unit, a TOE confirmed a spherical and double-walled mass suggesting a working diagnosis of left atrial ball thrombus (LABT). The case was discussed in Heart Team and considering the poor haemodynamic status in the contest of refractory cardiogenic shock with evidence of multi-organ failure, emergency surgical thrombectomy and mitral valve replacement was deemed prohibitive. Patient developed cardiac arrest and emergency TTE showed left atrial mass engaged into the mitral valve totally obstructing the left ventricle inflow tract. The autopsy and histologic examination confirmed the thrombotic nature of the mass.

Discussion
A free-floating ball thrombus in the left atrium is an unusual occurrence in rheumatic mitral stenosis and it may cause fatal systemic embolization or acute left ventricular inflow obstruction, resulting in syncope, pulmonary congestion, and sudden cardiac death. When possible, emergency surgical thrombectomy and mitral valve replacement can be life-saving.

Keywords
Left atrial ball thrombus • cardiogenic shock • mitral stenosis • case report

ESC Curriculum
4.4 Mitral stenosis • 5.3 Atrial fibrillation • 6.2 Heart failure with reduced ejection fraction • 2.2 Echocardiography
### Learning points
- Free-floating Left atrial Ball Thrombus (LABT) is a rare condition, which can present variegated onset from asymptomatically, incidental occurrence to critically unwell and cardiogenic shock requiring resuscitation.
- When feasible, surgery is best management. However, if perioperative or operative risk is deemed to outweigh the potential benefits, a bridging treatment with veno-arterial extracorporeal membrane oxygenator (VA-ECMO) should be considered in case of haemodynamic instability.
- Anticoagulation therapy is the cornerstone of LABT treatment but thrombolysis should be considered in selected cases, when small dimensions and morphological characteristics predicting thrombus stability suggest a low probability of fragmentation and systemic arterial embolization.
- Differential diagnosis of LABT must include benign, malignant or metastatic masses and, for these reasons, transthoracic, and transoesophageal 2D–3D echocardiography should be integrated with computed tomography and cardiac magnetic resonance in a multimodality imaging approach.

### Introduction
In rheumatic mitral stenosis (MS), the presence of a left atrial free-floating ball thrombus is an uncommon finding. By definition, it is larger than the mitral orifice, it has a smooth surface and no signs of attachment to the atrial wall.\(^1,^2\) Related symptoms can be syncope, pulmonary congestion or sudden cardiac death depending on the grade of occlusion of mitral valve orifice (occasional, incomplete, or complete respectively). Lastly, it may cause peripheral embolization due to superficial fragmentation.\(^1,^2\)

### Case presentation
A 75-year-old woman, with a history of mild rheumatic MS, was taken to the emergency department (ED) for near-syncope followed by acute dysnea. She had not been followed up for her MS for 20 years, and was not on any anticoagulant therapy. Shortly after admission to the ED the patient experienced rapid deterioration with haemodynamic instability resulting in cardiogenic shock with a cold and wet profile [SCAI (Society for Cardiovascular Angiography and Interventions) classification—stage C] and pulmonary oedema confirmed on chest X-ray. An ECG showed atrial fibrillation (AF) with rapid ventricular response. In accordance with the latest European Society of Cardiology (ESC) guidelines for the management of atrial fibrillation (AF),\(^1\) given the haemodynamic instability, an immediate electrical cardioversion was performed in the ED with restoration of sinus rhythm. Arterial blood gas analysis showed severe metabolic acidosis (pH: 7.04) with a very high lactate (16 mg/dl) and marked hypoxemia (pO\(_2\): 50 mmHg). Due to the persistence of haemodynamic instability associated with severe pulmonary oedema despite intravenous diuretic administration, she was intubated and mechanically ventilated. Immediate infusion of noradrenaline (0.5 mcg/kg/min) and dobutamine (0.5 mcg/kg/min) was started. Urgent transthoracic echocardiography (TTE) was performed in the ED, which revealed a severe reduction of left ventricular ejection fraction (LVEF: 15%) due to a diffuse left ventricular hypokinesia. The right ventricle was normal sized with severely reduced systolic function, along with the presence of severe tricuspid regurgitation and high estimated pulmonary systolic pressure (50 mmHg). Mitral leaflets appeared severely calcified and thickened with reduced systolic motion and doming appearance during diastole (Figure 1). The mean gradient to the diastolic filling across mitral valve was initially 5 mmHg (Figure 2A), raising to 16 mmHg (Figure 2B) when inotropic drugs infusion was started. A clear picture of severe rheumatic MS was evident. The left atrium (LA) was severely dilated and a large rounded mass was immediately appreciable. 2D–3D trans-oesophageal echocardiography (TOE) confirmed the severe MS (Figure 3A and Supplementary material online, Video S1) and the severe biventricular systolic dysfunction. The left atrial mass appeared perfectly spherical (diameter of 40 mm x 40 mm) and double-walled, largely immobile and leaning to the lateral atrial wall with no clear

### Timeline

| Admission Day 1 | Patient admitted with pulmonary oedema, cardiogenic shock, and atrial fibrillation. |
|-----------------|----------------------------------------------------------------------------------|
| Actions:       | 1. Electrical/DC cardioversion performed due to haemodynamic instability. |
|                | 2. 2D–3D TTE and TOE performed which showed severely reduced LVEF (15%), severe mitral stenosis, and evidence of large, spherical, and double-walled ball thrombus in left atrium. |
|                | 3. Patient intubated and mechanically ventilated. |
|                | 4. Intravenous infusion of inotropes and vasopressors started. |
|                | 5. Start of systemic anticoagulation with intravenous unfractioned heparin (UFH). |
|                | 6. Contrast-enhanced chest computed tomography (CT) performed which showed spherical mass contiguous to the lateral wall of the left atrium, and no signs of acute pulmonary embolism or systemic arterial embolization. |
|                | 7. Brain CT performed which showed: no signs of intracranial haemorrhage or recent ischaemia. |
| Day 2          | Ongoing cardiogenic shock refractory to dobutamine and norepinephrine. |
| Actions taken: | 1. Epinephrine infusion (0.1 mcg/kg/min) was started. |
|                | 2. The case was discussed in Heart Team, but given the persistent and severe haemodynamic instability associated with multi-organ failure, the patient was considered unfit for emergency surgery. |
| Day 3          | Refractory cardiac arrest due to incarceration of ball thrombus in the mitral orifice (hole-in-one thrombus). |

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1. Selimi A, et al. Persistent and severe haemodynamic instability associated with multi-organ failure, the patient was considered unfit for emergency surgery.
A truck tire into the heart

signs of attachment. The imaging was not suggestive of atrial myxoma (Figure 3B). The external contour appeared very echo-dense with an external fibrous cap almost 4 mm thick and an internal thinner layer. The central core of the mass appeared friable. The final appearance was of a ‘truck tire’ (Figure 3B and Supplementary material online, Videos S2 and S3). The mass appeared to be a ball-valve thrombus. A dense echocochetric filled the entire LA with a mobile clot inside the left atrial appendage (LAA) (Supplementary material online, Video S4). 3D-TEE images were helpful in confirming the mass composition (Supplementary material online, Videos S5 and S6). The patient underwent contrast-enhanced chest and abdomen computed tomography (CT) with iodine contrast to discriminate diagnosis between thrombus, left atrial myxoma, or primary malignant tumours as well as cardiac metastases from an extracardiac origin. CT confirmed the presence of the rounded mass contiguous to the lateral wall of the LA with no post-contrast perfusion confirming its avascular nature. Therefore, we excluded acute pulmonary embolism or systemic arterial embolization or extracardiac tumours (Figure 4). Brain CT showed no signs of intra-cranial haemorrhage or recent ischaemia. The levels of troponin T (TnT) (peak level of 147 ng/mL with an upper reference limit, <14 ng/mL) and N-terminal pro-brain natriuretic peptide (NT-proBNP) (17,000 pg/mL with an upper reference limit, <20 pg/mL) were elevated. Patient continued to be monitored in the intensive care unit and systemic anticoagulation therapy with intravenous unfractioned heparin (UFH) was commenced according to the ESC guidelines for the management of AF.3 On day 2, because of persistence of cardiogenic shock (SCAI stage D), refractory to dobutamine and norepinephrine, epinephrine infusion (0,1 mcg/kg/min) was added. The case was discussed in Heart Team and considering the haemodynamic instability in the context of refractory cardiogenic shock with evidence of multi-organ failure, an emergency surgical procedure was deemed inappropriate. The feasibility of mechanical circulatory support such as VA-ECMO was considered and deemed not feasible due to the evidence of critical peripheral vascular disease on the CT scan prohibiting arterial vascular access. The patient developed cardiac arrest due to ventricular fibrillation and immediate cardiopulmonary resuscitation was performed. Unfortunately, defibrillation was followed by irreversible pulseless electrical activity. Emergency TTE showed left atrial mass engaged into the mitral valve and totally obstructing the left ventricle inflow via a ball-valve effect, so-called ‘hole-in-one thrombus’,4 and the patient died shortly after this. The autopsy findings confirmed the presence of the mass entrapped into the mitral valve (Figure 5A, arrow) and histologic examination revealed its thrombotic nature. The mass section revealed its constitution of multiple layers (Figure 5B).

Discussion

Differential diagnosis of cardiac masses includes neoplastic lesions (benign and malignant tumours) and tumour-like conditions (thrombus, Lambl’s excrescences, vegetations, caseous and calcific lesions, intracardiac cysts, etc.). The use of multimodality imaging (including 2D–3D TTE and TOE, cardiac CT, cardiac magnetic resonance [CMR]) allows a more accurate identification of the mass nature, analyzing its extension, extracardiac involvement and tissue characterization.5 Cardiac

Figure 1 TTE, parasternal long axis (PLAX) showing severely dilated atrium with a giant rounded mass and doming of thickened mitral leaflets during diastole.

Figure 2 Continuous wave (CW) Doppler across mitral inflow: the mean gradient to the diastolic filling across mitral valve was initially 5 mmHg (panel A), raising to 16 mmHg (panel B) when inotropic drugs infusion was started.
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thrombus is the most common intracardiac mass but an unattached, freely moving thrombus within the LA, called ‘Left Atrial Ball Thrombus’ (LABT), is a rare entity with peculiar morphology related to a specific etiopathogenesis.

**Etiopathogenesis and morphology**
LABT usually arises in a context of atrio-ventricular low-flow status, due to rheumatic MS or different pathology. It usually originates from the atrial septum (uncommonly from the LAA) as a small mural thrombus and it progressively increases its size by thrombotic neo-apposition until the pedicle becomes too thin and the mass finally completely detaches. Thereafter, it moves freely in the atrium with a rotary movement around its own axis with fresh clot being layered out concentrically as it spins around the atrium having randomly oriented collisions with left atrial walls and mitral valve apparatus. In this way, it acquires the characteristic smooth, spherical appearance and a size which is usually bigger than the mitral valve orifice. In our patient,

**Figure 3** Panel A: 2D-TOE, apical five chamber (ASC) view. Severely calcified and thickened mitral leaflets with reduced diastolic motion and doming appearance during diastole. Double-walled, spherical mass in LA. Panel B: 2D-Trans-Oesophageal Echocardiography (TOE), ASC view focused on left atrium, showing a perfectly spherical mass (diameter of 40 mm x 40 mm) with a double-walled appearance, not very mobile and leaning to the lateral atrial wall with no clear signs of attachment. The external contour appeared very echo-dense and 4 mm thick, while the internal and concentric wall was thinner. Between the two walls and in the central core of the mass a friable material was evident.
LABT had a double-walled shape with a ‘truck tire’ appearance. This shape may be due to a double calcification layer associated with hemolysis areas between the two calcified layers and in the central core of the mass. For this reason, the LABT here reported is characterized by an alternance of calcified layers and friable substance. These findings were confirmed by the post-mortem specimen sections and histologic analysis.

### Complications

The main complications derive from the free movements inside the LA, especially toward mitral valve. It could cause three types of clinical events: (i) syncope (if mitral valve is occasionally occluded for a short time); (ii) an acute pulmonary oedema (if mitral valve is incompletely obstructed); (iii) sudden death, if mitral valve is completely and permanently occluded. Arterial peripheral embolization due to superficial mass fragmentation could represent a rare complication maybe accountable to ageless endothelization of long-lasting mass surface that may prevent platelet aggregation and subsequent fragmentation.

As previously described in the literature, we speculate in the acute setting our patient experienced acute diastolic heart failure, severe pulmonary oedema and near-syncope due to transient incomplete obstruction of the left ventricular inflow tract (LVIT) above the mitral orifice, even though at the moment of cardiologic evaluation in the ED there was no evidence of diastolic mechanical inflow obstruction and the mass appeared adhering to left atrial wall. More likely, rapid ventricular response rate during AF led to inadequate diastolic filling time contributing to haemodynamic instability. Concurrent with loss of atrial contribution to ventricular filling, these conditions exacerbate haemodynamic failure, leading to a standstill low-flow status and once more thrombus adherent to the left atrial free wall. Another concern was the presence of severe left ventricular systolic dysfunction and refractory cardiogenic shock poorly responsive to high-dose inotropic infusion therapy (SCAI stage D). LVEF is generally preserved in severe

![Figure 4](image1.png) **Figure 4** Contrast-enhanced chest computed tomography (CT). Spherical mass contiguous to the lateral wall of the left atrium.

![Figure 5](image2.png) **Figure 5** Panel A: post-mortem gross examination of the heart with the presence of the mass entrapped into the mitral valve (arrow). Panel B: gross examination of the post-mortem specimen, cross section of the clot with roughly concentric lamellae with interposed areas of semi-liquid material.
MS and cardiogenic shock is usually due to inadequate preload-dependent state. We postulated different hypotheses regarding the etiology of severe reduction of LVEF in this patient: (i) acute mechanical diastolic dysfunction could lead a transient global reduction of preload with consequent reduction of coronary blood flow causing global myocardial ischaemia; (ii) acute coronary syndrome caused by coronary embolization could result in regional wall motion abnormalities, but the territory of potential one vessel ischaemia did not match the severe diffuse left ventricle hypokinesia shown during acute presentation; (iii) moreover, lack of diastolic left ventricular filling yielded to a chronic left ventricular low-flow status resulting in left ventricular remodeling with pathological end-systolic volume development, due to inability to generate an adequate anterograde stroke volume; (iv) lastly, as our patient has never been followed up for her valvular disease, we have no clear history of a new-onset AF or permanent AF, with potential role of a chronic tachy-cardiomyopathy reducing left ventricular systolic function.

Treatment

Because of the relative infrequency of this condition, there is a lack of consensus on best management. In previously published case reports, several authors state that, once the diagnosis of a LABT is established, prompt surgical thrombectomy with mitral valve replacement is recommended in order to prevent the catastrophic complications arising from the unstable nature of such masses. Anticoagulation and thrombolytic therapy seems not to be included in the acute management of LABT because of the risk of superficial fragmentation and embolization. We promptly started systemic anticoagulation according to current guidelines for the management of AF for several reasons: (i) according to guidelines effective anticoagulation is strongly recommended in AF; (ii) we obtained clear evidence of atrial thrombi after TOE, (iii) to reduce further stratification on the LABT surface and also to reduce its diameter in order to prevent incarceration into the mitral orifice. However, we decided not to start fibrinolysis because of: (i) lack of available evidence in the setting of LABT; (ii) the high risk of catastrophic cerebral and systemic arterial embolization due to the fragmentation of such a large thrombus. Although one successful case with anticoagulation therapy alone has been described, it accounts for small size mass, while most successful cases were treated surgically. The outcome of untreated ball thrombus is unlikely to be favourable. Current guidelines on management of valvular heart disease recommend percutaneous mitral commissurotomy (PMC) in symptomatic patients with clinically significant rheumatic MS without unfavorable characteristics for PMC and in the absence of contraindications, including left atrial thrombus. Otherwise, mitral valve surgery is recommended. Furthermore, there are no dedicated guidelines on the management of cardiogenic shock in MS, and medical management strategy is relatively limited in this clinical setting. Only two studies discuss the efficacy of emergency PMC in a cardiogenic shock setting, where mortality rate was described to be up to 35%. Evident left atrial thrombus in our case excluded a PMC. Unfortunately, given the persistent and severe refractory cardiogenic shock associated with multi-organ failure despite inotropes, an emergency surgical thrombectomy with mitral valve replacement was deemed infeasible.

Conclusion

LABT represent an uncommon finding in cases of severe rheumatic mitral stenosis and an unusual cause of cardiogenic shock. The presence of left atrial mass contradicts any percutaneous approach which could be life-saving in the emergency setting where a classic surgical approach is deemed infeasible. Medical therapy only has a marginal role in severe mitral stenosis due to preload dependent state and poor response to classical inotropic therapy. Direct thrombectomy with mitral valve replacement appear the only resolutive approach in these selected cases but is often contraindicated due to preoperative instability and adverse surgical risk, even more so in the acute context of refractory cardiogenic shock.

Lead author biography

Vito Maurizio Parato, MD, is a professor of Cardiology at Politecnica delle Marche University, Ancona, Italy. He is an FESC. His fields of interest are: (i) Non-invasive cardiovascular imaging; (ii) Valvular heart diseases; and (iii) Cardiac rehabilitation.

Supplementary material

Supplementary material is available at European Heart Journal – Case Reports.

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

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient’s sister, in line with COPE guidance.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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