

Cardiac Arrest after a Transatlantic Flight in a Patient with a Large Left Atrial Myxoma



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INTRODUCTION

Primary cardiac tumors are extremely rare, with an incidence, according to autopsy series, of <0.1%.¹ About 75% of all primary cardiac tumors are benign. Cardiac myxomas, a neoplasm arising from endocardial mesenchymal stem cells, represent the most common benign cardiac tumor in adults.² Cardiac myxomas can occur in all age groups but are most common in middle-aged patients,² with a female predominance of approximately 2:1.³ Cardiac myxomas typically originate from the interatrial septum, and the left atrium is the most common location of the mass, housing up to 80% of all myxomas.² Cardiac myxomas are often discovered accidentally, typically using transthoracic echocardiography (TTE) or other imaging modalities such as transesophageal echocardiography (TEE) or computed tomography.⁴ The condition can be asymptomatic, and the clinical manifestations are generally determined by the size and anatomic location of the tumor.⁵ Common symptoms include dyspnea, arrhythmias, fatigue, malaise, and low-grade fever.⁶ Although cardiac myxomas are considered benign, they can cause acute life-threatening situations related to systemic embolization or intracardiac obstruction and constitute an indication for prompt surgical intervention.⁷

We report a case of cardiac arrest in a patient with a large left atrial myxoma, after a transatlantic flight, in which TEE played a crucial role in decision-making.

CASE PRESENTATION

A 68-year-old woman presented to the emergency department in severe respiratory distress. Earlier that day she had traveled on a 6-hour, nonstop transatlantic flight. After landing, she suddenly developed dyspnea and was urgently transferred by ambulance to the emergency department. Medical history was notable for hypertension, osteoarthritis, obesity, and 50 pack-years of smoking. The patient had exercise-induced dyspnea for 6 months before the event, which was interpreted as asthma.

On presentation, airways were clear, but the patient experienced significant tachypnea, with poor oxygen saturation of 55% to 70%

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Drs. Wedin and Kristófi contributed equally to this work.

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VIDEO HIGHLIGHTS

Video 1: Midesophageal four-chamber view showing a large left atrial myxoma prolapsing into the mitral valve during diastole, partially occluding the inflow of blood to the left ventricle.

Video 2: Color Doppler midesophageal four-chamber revealed a turbulent inflow into the left ventricle, suggesting a hemodynamically significant obstruction.

Video 3: Color Doppler midesophageal four-chamber view also showed a concomitant mitral regurgitation.

Video 4: Color Doppler midesophageal long-axis view of the mitral valve demonstrated a mitral valve regurgitation jet hitting the left atrial myxoma, making echocardiographic assessment of the severity complicated. During perioperative water leakage test, mitral valve regurgitation was determined to be severe.

Video 5: Zoomed midesophageal four-chamber view after resection of the left atrial myxoma and subsequent mitral valve repair showed only mild residual mitral regurgitation.

Video 6: Zoomed midesophageal long-axis view revealed mild central mitral regurgitation after removal of the left atrial myxoma and subsequent mitral valve repair.

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despite supplemental oxygen delivery at 15 L/min. This was confirmed by arterial blood gas analysis, further demonstrating lactic acidosis with a pH of 6.9. Initially, the patient was hemodynamically stable with a blood pressure of 160/115 mm Hg and a heart rate of 100 beats/min. Electrocardiography demonstrated incomplete right bundle branch block and signs of left atrial enlargement (Figure 1). Physical examination revealed bilateral diffuse crackles and a holosystolic murmur. There were no signs of peripheral edema or jugular venous distention. The patient had no neurologic deficits, except for exhaustion, and no signs of ongoing infection or asymmetric swelling of the lower limbs.

Laboratory results showed elevated D-dimer of 3.8 mg/L (normal value <0.5 mg/L) as well as elevated cardiac troponin I of 37 ng/L (normal value <16 ng/L). Point-of-care echocardiography raised suspicion of pulmonary embolism, as the right ventricle and right atrium appeared dilated. Pulmonary embolism was ruled out with computed tomographic angiography, which instead revealed a large mass (approximately 60 × 40 mm) in the left atrium with the appearance of a myxoma (Figure 2). However, there was a slight delay in reporting of the radiologic findings, and the mechanism of respiratory failure remained unknown when the patient was transferred to the thoracic intensive care unit for stabilization. After arrival to the thoracic intensive care unit, the patient became hemodynamically unstable and

developed ventricular fibrillation. Cardiopulmonary resuscitation was initiated with return of spontaneous circulation after 10 min. TEE demonstrated a mobile left atrial mass, originating from the interatrial septum. It moved in synchrony with the atrial contractions back and forth through the mitral valve, partially obstructing the filling of the left ventricle (Figure 3, Videos 1 and 2). TEE thus revealed the mechanism of pulmonary congestion, from which a final decision to undertake urgent surgery was made. Concomitant mitral regurgitation was also diagnosed on TEE (Videos 3 and 4).

The patient underwent an acute median sternotomy, and full cardiopulmonary bypass was initiated through separate cannulation of the venae cavae and the ascending aorta. The atrial mass was resected through incision of the left atrium; it measured 70 × 50 × 35 mm (Figure 4), and histologic analysis later confirmed the diagnosis of cardiac myxoma. Intraoperative testing of the mitral valve confirmed prolapse of both the P2 and A2 segments of the posterior and anterior leaflets, respectively. Mitral valve repair was performed with a 30-mm Carpentier-Edwards Physio II ring, and separate artificial

chordae tendineae were sutured to the respective leaflet segments. Intraoperative TEE showed complete removal of the left atrial myxoma and a well-functioning mitral valve repair with only mild residual regurgitation (Videos 5 and 6).

During weaning from cardiopulmonary bypass, the patient developed respiratory failure, necessitating initiation of venoarterial extracorporeal membrane oxygenation (ECMO) through the femoral artery and vein. After 2 days, lung function had recovered, and the patient was weaned off ECMO support. The subsequent postoperative course was uneventful, and the patient was discharged from the ward after a total stay of 10 days. At 1-month follow-up, the patient had recovered well, with only minor problems from the ECMO cannulation site in the right leg.

DISCUSSION

This is the first report of cardiac arrest after a commercial flight in a patient with a left atrial myxoma. The atrial myxoma likely caused

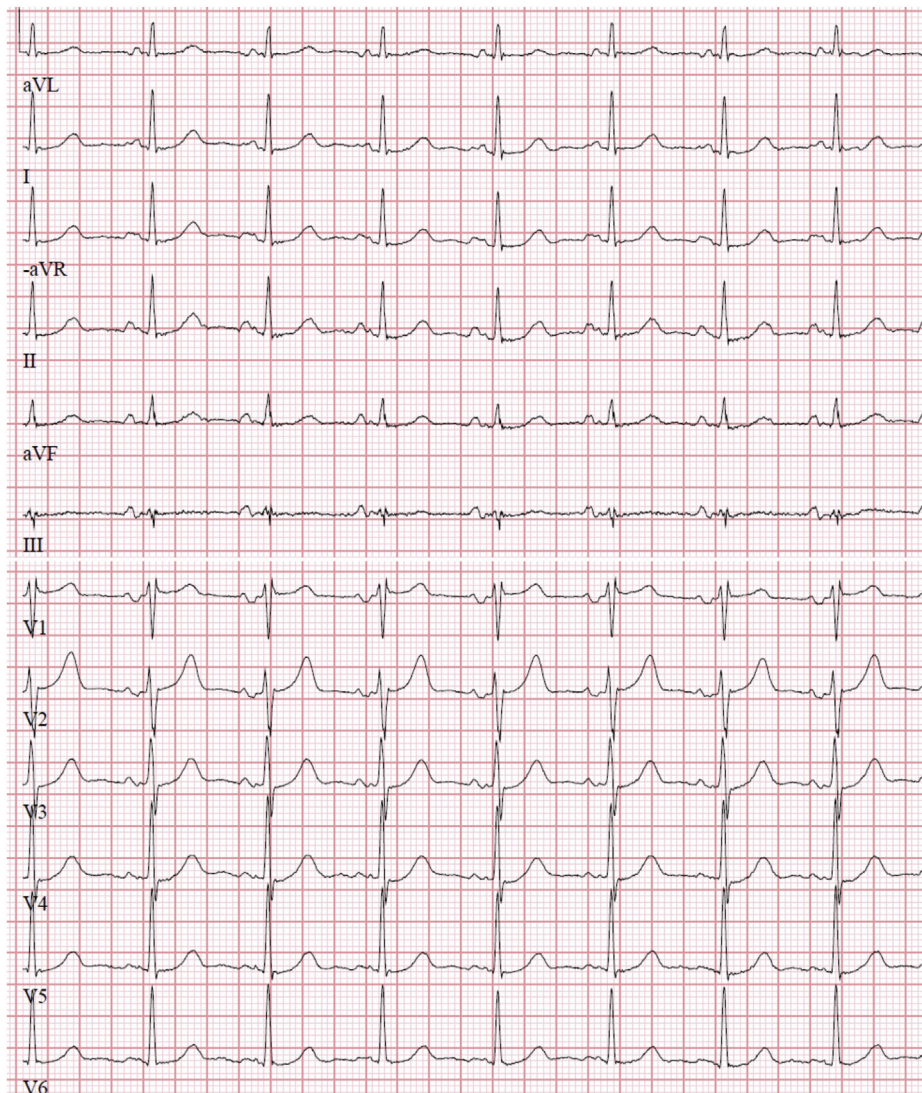


Figure 1 Preoperative electrocardiography at the emergency department showed tachycardia, incomplete right bundle branch block, and signs of left atrial enlargement.

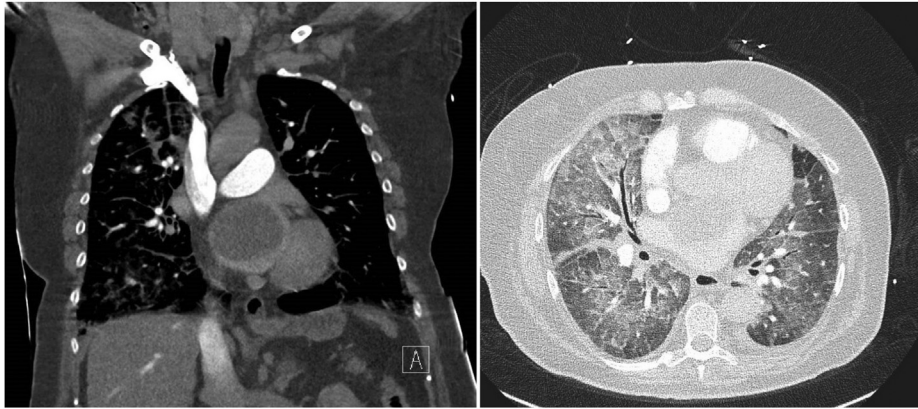


Figure 2 Computed tomography (CT) ruled out pulmonary embolism as a cause of respiratory failure. Instead, a large left atrial mass was found. CT also showed pronounced bilateral pulmonary edema.

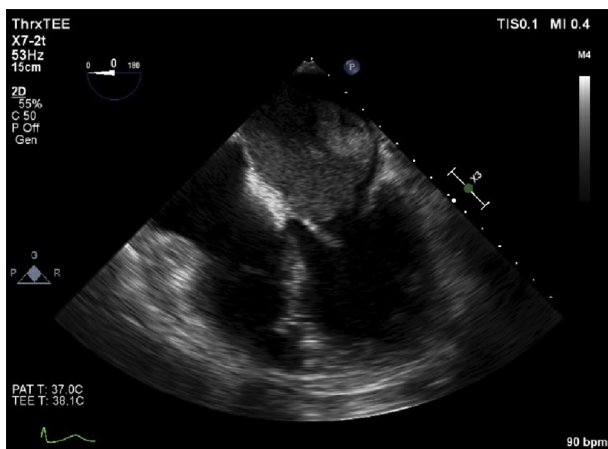


Figure 3 TEE was used to diagnose the cause of hemodynamic instability. It revealed a large atrial mass with the appearance of a cardiac myxoma, and a decision to undertake urgent surgery was made. This midesophageal four-chamber view shows how the left atrial myxoma prolapses into the mitral valve during diastole.

obstruction of the pulmonary venous return to the heart, with increased pulmonary artery pressure and pulmonary edema as a result. A combination of hypoxia and reduced filling of the left ventricle probably induced ventricular fibrillation. The patient's aggravated respiratory failure after the transatlantic flight is probably explained by several physiologic cardiovascular alterations induced by air travel. The sympathetic nervous system is stimulated during a flight, which leads to an increased heart rate and contractility. Dehydration is also common during air travel, leading to a further compensatory increase in heart rate to maintain cardiac output.⁸ There is increased pulmonary capillary permeability as well as pulmonary vasoconstriction with leakage of blood plasma through the capillary bed as a result.⁹ All these physiologic effects are tolerated by healthy individuals but can predispose to cardiac decompensation in people with heart conditions.⁸ Hypobaric hypoxia is another adverse effect of air travel. The cabin pressure in commercial flights usually corresponds to that of an altitude of 1,500 to 2,700 m above

sea level.¹⁰ Although the subatmospheric pressure is well tolerated by healthy individuals, it can cause respiratory distress in patients with heart problems as they experience reduced baseline oxygen saturation.⁹ Moreover, the respiratory distress becomes more severe with increasing flight time.¹¹ Furthermore, our patient had a large left atrial myxoma that partially occluded mitral inflow, mimicking a mitral valve stenosis. Sudden mobilization of venous return, after 6 hours of immobilization, led to a sudden increase in preload, probably causing pulmonary venous stasis and subsequent pulmonary edema when the patient returned to sea level.

The use of point-of-care TTE in the emergency department may allow rapid diagnosis of life-threatening situations like cardiac tamponade.¹² It may indeed be lifesaving. However, there are several drawbacks with this modality when used by nonspecialist practitioners. The scope of practice is restricted to the skill that the imager possesses. Emergency care physicians have less training in image acquisition and interpretation compared with physicians and sonographers that are trained in echocardiography. The imager might also lack knowledge about rare conditions such as cardiac myxoma. In our patient, the myxoma was overlooked in the initial evaluation with point-of-care TTE.

It is well known that large myxomas occasionally obstruct filling of the left ventricle, and temporary complete obstruction has been reported to cause syncope or sudden death.¹³ The uniqueness of this case is that the symptoms of the myxoma were aggravated after a long-distance flight (i.e., sudden onset of refractory respiratory failure and subsequent hypoxic cardiac arrest). We hypothesize that the aforementioned physiologic responses to air travel induced cardiopulmonary decompensation in our patient. This has been described in several heart conditions, but it has never been reported for cardiac myxomas. In addition, the case demonstrates the potential advantages of TEE in emergency situations. The high sensitivity of TEE has been confirmed previously.¹⁴ TEE can be performed during cardiopulmonary resuscitation without the need to stop compressions, and image quality is superior to that of TTE.¹⁵ TEE in the thoracic intensive care unit revealed the mechanism of acute pulmonary edema and cardiac arrest, on the basis of which the adequate decision of immediate life-saving surgery could be made. It also demonstrated severe mitral regurgitation, which led to the decision to perform concomitant mitral valve repair. The reason the patient experienced postoperative respiratory failure, necessitating venoarterial ECMO, was probably a

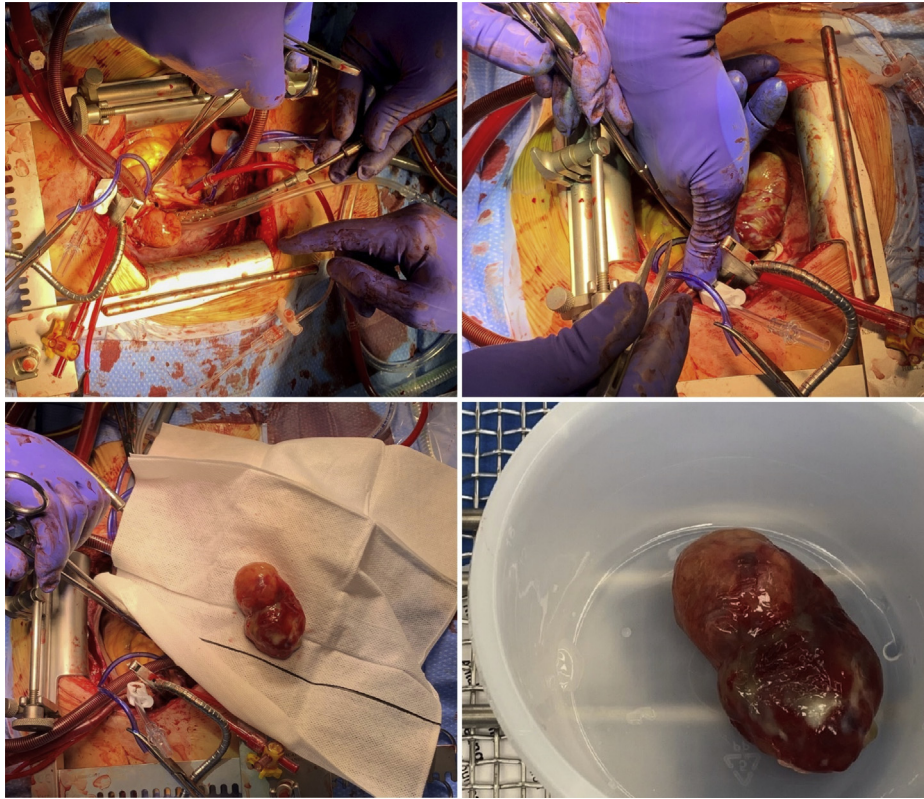


Figure 4 The patient was transferred to the operating room for urgent surgery. A median sternotomy was performed, and through subsequent opening of the left atrium, the cardiac myxoma was excised. It originated from the interatrial septum and measured 70 × 50 × 35 mm.

combination of the severe pulmonary edema and contusions related to cardiopulmonary resuscitation.

CONCLUSION

In conclusion, myxomas are the most frequent primary cardiac tumors. Regardless of the clinical presentation of cardiac myxomas, resection via open heart surgery is considered the gold-standard treatment, and the results are excellent.⁷ It is important to include cardiac myxoma in the differential diagnosis of respiratory failure and acute pulmonary edema in the emergency department, as early consultation with a cardiothoracic surgeon may be lifesaving. This case also demonstrated the usefulness of TEE as a diagnostic tool during cardiopulmonary resuscitation and its role in decision-making.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2019.10.003>.

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