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Surgical Septal Myectomy and Atrial Myxoma Resection: Two Diseases, One Heart, and a Case Report

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 38-year-old
Final Diagnosis: Cardiac myxoma
Symptoms: Dispnea
Clinical Procedure: Cardiac surgical procedures
Specialty: Cardiac surgery

Objective: Rare coexistence of disease or pathology

Background: The coexistence of cardiac myxoma and hypertrophic cardiomyopathy is exceedingly rare and poses diagnostic and therapeutic challenges. While atrial myxomas may cause acute hemodynamic compromise, concomitant left ventricular outflow tract obstruction (LVOTO) due to hypertrophic cardiomyopathy can remain clinically underestimated, raising uncertainty regarding the optimal timing of septal reduction therapy.

Case Report: A 38-year-old man was admitted with acute dyspnea and hypoxemia. Imaging revealed pulmonary congestion and a large left atrial mass causing functional mitral stenosis. Transthoracic echocardiography demonstrated asymmetric septal hypertrophy (maximum thickness 25 mm) with dynamic LVOTO and a mobile left atrial mass consistent with myxoma. The patient underwent surgical resection of the tumor with concomitant septal myectomy. Histopathology confirmed atrial myxoma and myocardial hyperplasia. Postoperatively, complete atrioventricular block required permanent dual-chamber pacemaker implantation. At short-term follow-up, the patient was asymptomatic with mild residual LVOTO.

Conclusions: This case underscores the importance of comprehensive structural and functional assessment in patients with intracardiac tumors. In young patients with favorable prognostic features and significant left atrial dilatation, early septal myectomy performed concomitantly with tumor resection may be justified, even when resting LVOT gradients are below conventional thresholds. This strategy aligns with the latest American and European guidelines on cardiomyopathies and may prevent delayed intervention and disease progression. The case also highlights atrioventricular block as a relevant complication of surgical myectomy, reinforcing the need for careful perioperative planning. Overall, this report provides an instructive example of individualized surgical decision-making in complex cardiomyopathy presentations.

Keywords: Cardiology • Case Reports • Hypertrophic Cardiomyopathy • Myxoma
Abbreviations: LVOTO, left ventricular outflow tract obstruction; LV, left ventricle
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953036

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Introduction

Primary cardiac tumors are rare neoplasms. The prevalence of primary cardiac tumors in autopsy studies is approximately 0.02%, with benign tumors representing 75% of cases. The majority of patients are asymptomatic, and tumors are found incidentally through imaging. Atrial myxoma is the most common benign primary cardiac tumor and is recognized as a potentially life-threatening condition due to its embolic and obstructive complications [1].

In turn, hypertrophic cardiomyopathy with left ventricular outflow tract obstruction (LVOTO) is a genetically determined myocardial disease addressed in dedicated guidelines [2,3] because of its association with heart failure, arrhythmias, and sudden cardiac death. Treatment strategies differ depending on the finding of LVOTO, defined by peak LVOT gradient 30 mmHg or greater. Gradients of 50 mmHg or greater are usually considered capable of causing symptoms [3] and, therefore, are the threshold for considering advanced pharmacological or invasive therapies if refractory symptoms occur.

The coexistence of both diseases is exceptionally rare, with few case reports in the literature published between 1981 and 2021. This association can lead to diagnostic uncertainty and therapeutic challenges, as both conditions can contribute to dynamic obstruction and hemodynamic compromise. This case illustrates the importance of comprehensive imaging and guideline-based decision-making in complex structural heart disease.

Case Report

A 38-year-old man was admitted to the emergency department with sudden dyspnea that worsened in the supine position, accompanied by cough and oxygen desaturation requiring supplemental oxygen. He denied fever in the previous few days, other comorbidities, smoking, alcohol, other drug abuse, or regular use of medications. He had tachypnea and was in respiratory distress, without cyanosis. His heart rate was 130 beats/min, arterial blood pressure 110/80 mmHg, and temperature 36.6°C. Pulse oximetry was 94% with a nasal oxygen catheter at 4 L/min. Lungs presented bilateral basal rales, and cardiac auscultation was normal. He had mild edema in both legs with no signs of deep vein thrombosis.

Arterial blood gas showed pH of 7.39, PaO_2 of 62.5 mmHg, $PaCO_2$ of 36.1 mmHg, HCO_3^- of 21.4 mmol/L, and oxygen saturation of 91%. Other labs included Na^+ 140 mmol/L, K^+ 3.9 mmol/L, Cl^- 106 mmol/L, and glucose 133 mg/dL. D-dimer was 1782 ng/dL (upper limit was 550 ng/dL). There were no other significant laboratory test alterations. Chest X-ray revealed signs of pulmonary congestion and left atrium enlargement.

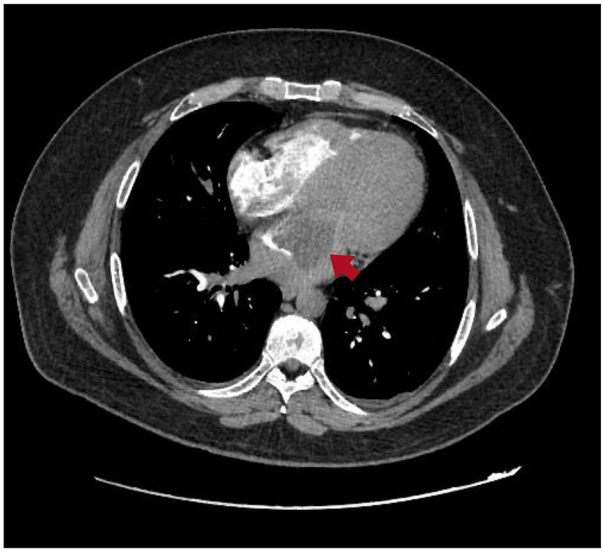

Figure 1. Chest tomography showing an ovoid and regular mass measuring 56×42 mm with no contrast enhancement in the left atrium in contact with the mitral valve.

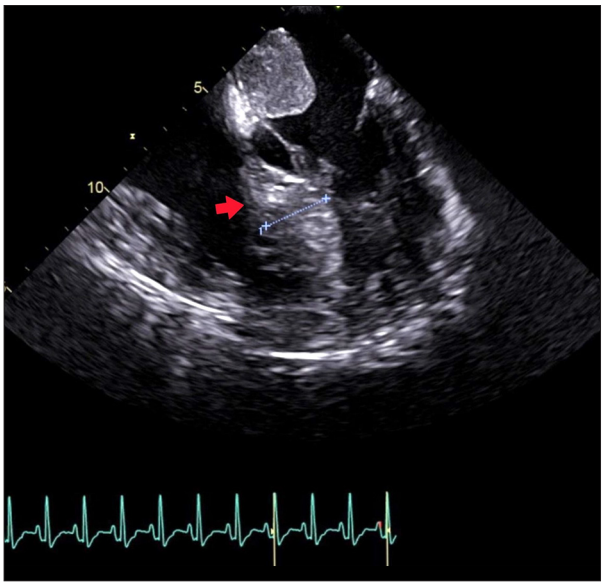

Figure 2. Echocardiogram with asymmetrical left ventricle concentric hypertrophy of the interventricular septum (25 mm).

The electrocardiogram showed sinus rhythm, right bundle branch block, and no signs of ischemia.

Although the Wells criteria were low for pulmonary embolism, chest tomography (CT) was performed, which showed no evidence of pulmonary embolism. An ovoid and regular mass measuring 56×42 mm with no contrast enhancement was observed in the left atrium in contact with the mitral valve (**Figure 1**). Pulmonary edema was diagnosed, attributed to a left atrial tumor causing functional mitral valve obstruction.

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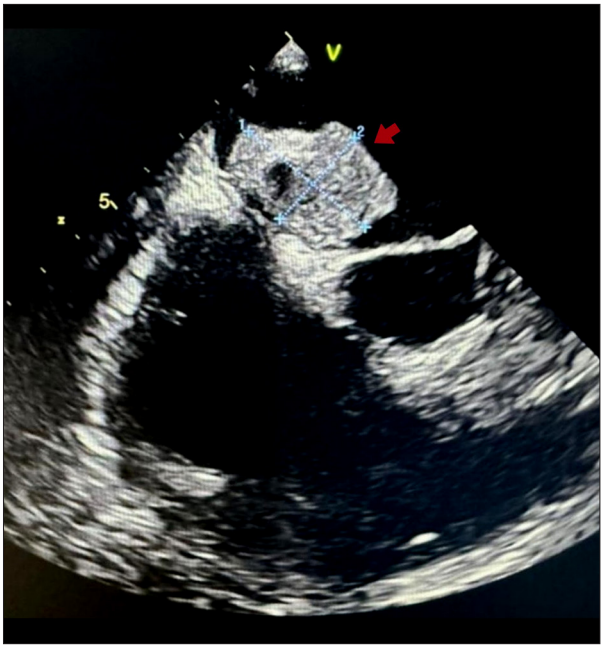

Figure 3. Echocardiogram with intracardiac mass, measuring 35×28 mm, protruding toward the mitral valve.

Transthoracic echocardiogram showed enlarged left atrium diameter (52 mm), and asymmetrical left ventricle (LV) concentric hypertrophy, predominantly affecting the interventricular septum, with a thickness of 25 mm (**Figure 2**). Dynamic LV outflow tract obstruction was observed, with a maximum gradient of 21 mmHg at rest and 39 mmHg after the Valsalva maneuver. A mobile mass was observed in the left atrium, measuring 35×28 mm, which protruded toward the mitral valve. The mass significantly compromised blood flow, resulting in functional mitral stenosis, with a maximum left atrial to LV diastolic gradient of 23 mmHg and an average gradient of 8 mmHg (**Figure 3**).

The patient was transferred to the Cardiology Unit. Symptoms improved with intravenous diuretic therapy. Surgical treatment to remove the intracardiac mass, probably a myxoma, was proposed. Another concomitant diagnosis was hypertrophic cardiomyopathy. There were concerns about the need for simultaneous septal myectomy, as the maximum resting gradient was less than 50 mmHg, symptoms were attributed to myxoma, and the procedure, although adding low morbidity and mortality, is not free from complications. The patient's preoperative mortality risk was estimated as 1.21% through the European System for Cardiac Operative Risk Evaluation (EuroSCORE) II, considering isolated myxoma resection, and 2.05% considering concomitant septal myectomy.

Surgical treatment involved median sternotomy and establishment of cardiopulmonary bypass. We used a transatrial septal approach to resect a violaceous and friable atrial tumor

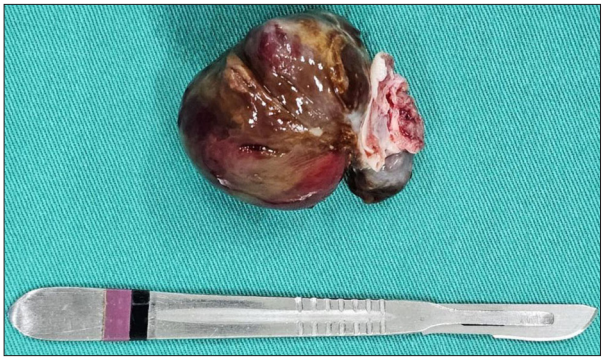

Figure 4. Tumor after resection, measuring 58×45×20 mm and weighing 44.3 g.

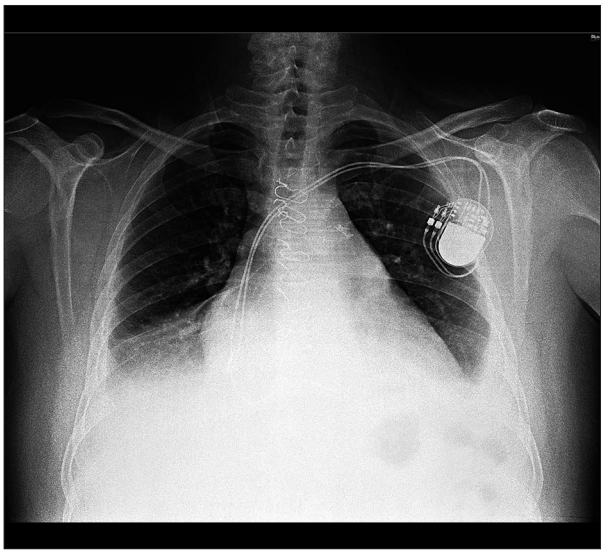

Figure 5. Chest X-ray after dual-chamber pacemaker implantation.

(**Figure 4**). We then performed an aortotomy and extended a rectangular septal myectomy inferior to the implantation of the right coronary cusp. After releasing the aortic clamp and de-airing the heart chambers, the patient presented a 2: 1 atrioventricular block rhythm. Two temporary epicardial pacemaker wires were placed. He was weaned from cardiopulmonary bypass and transferred to the Intensive Care Unit.

During the postoperative period, the patient had complete atrioventricular block and needed a temporary epicardial pacemaker. After carefully reviewing the most recent guidelines on cardiac pacing [4,5], considering the low likelihood of sinus rhythm recovery due to the proximity of the myectomy site to the cardiac conduction system, and noting the persistence of complete atrioventricular block in the postoperative period, we opted for implantation of a permanent dual-chamber pacemaker on postoperative day 6 (**Figure 5**), following loss of capture of the temporary epicardial pacing system.

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Transthoracic echocardiogram performed on postoperative day 13 showed moderate left atrium dilatation, asymmetric septal hypertrophy with an interventricular septum thickness of 18 mm, mild LV outflow tract stenosis (peak gradient of 25 mmHg), and a peak diastolic intraventricular gradient of 12 mmHg, without interventricular septal defect, and with normal aortic valve function.

Histopathology confirmed the diagnosis of a myxoma measuring 58×45×20 mm, weighing 44.3 g. Deep surgical margins were free from neoplasia. The portion of septal myectomy revealed a tissue with moderate myocardial hyperplasia. The patient was discharged on day 18 after surgery and returned for clinical evaluation on postoperative day 30 with no symptoms and taking enalapril and spironolactone. Pacemaker function was normal.

Over 1 year after surgical treatment, the patient remains asymptomatic, with a transthoracic echocardiogram showing preserved left ventricular ejection fraction, moderate left atrium dilation, and no signs of LV outflow tract stenosis. The patient remains dependent on a pacemaker, currently on ventricular pacing and sensing mode due to permanent atrial fibrillation, for which rivaroxaban has been prescribed.

Discussion

In adults, hypertrophic cardiomyopathy is defined by a LV wall thickness of 15 mm or greater in 1 or more myocardial segments, in the absence of abnormal loading conditions [3]. The coexistence of hypertrophic cardiomyopathy and atrial myxoma is exceedingly rare and presents unique diagnostic and therapeutic challenges. During the initial Heart Team discussion, the indication for surgical septal myectomy was considered debatable, as the reported LVOT gradients were not severe. Moreover, hemodynamic assessment was likely confounded by a myxoma-related, mitral stenosis–like physiology, leading to impaired LV filling and reduced preload, potentially underestimating the true degree of dynamic LVOT obstruction.

The observed clinical improvement with diuretic therapy further supported the hypothesis that pulmonary congestion secondary to inflow obstruction, rather than fixed or dynamic LVOTO, was the predominant mechanism of symptoms at presentation. Nevertheless, reliable assessment of LVOTO under these conditions is inherently limited. Given the patient's young age and the unequivocal indication for surgical resection of the atrial myxoma, the operative setting provided a unique opportunity to address a potentially progressive LVOTO at an early stage. It is relevant to emphasize that the transeptal approach was selected due to the myxoma's septal attachment, allowing complete excision of the tumor and its implantation site. Residual septal tumor is a recognized risk factor for recurrence.

Both the American and European guidelines emphasize that surgical septal reduction therapy should be considered in patients with hypertrophic cardiomyopathy undergoing cardiac surgery for other indications [2,3], particularly when LVOTO cannot be confidently excluded and when delaying intervention may be associated with worse long-term outcomes. Furthermore, guideline statements highlight that earlier intervention may be reasonable in the presence of left atrial enlargement and symptoms attributable, at least in part, to diastolic dysfunction and elevated filling pressures. In this context, to improve long-term functional status, quality of life, and prognosis, the Heart Team deemed concomitant septal myectomy appropriate rather than a staged approach, which might require reoperation for septal reduction after initial myxoma resection.

Transaortic septal myectomy in asymmetric septal hypertrophic cardiomyopathy with LVOTO allows reduction of left ventricular gradient, with a mortality rate of less than 1% and success rate of 90% to 95% [2]. Performing this procedure adds little risk to patients undergoing cardiac surgery for other reasons, and reducing the LVOT gradient may reduce postoperative hemodynamic instability. Electric cardiac disturbances are common after septal myectomy. Left bundle branch block is reported in up to 40% to 60% of patients. Total atrioventricular block requiring permanent pacemaker implantation is less common and reported in 9% of cases [6].

Importantly, our patient exhibited several favorable prognostic factors associated with excellent surgical outcomes, including age less than 50 years, absence of atrial fibrillation, modest left atrial size, and male sex [3]. Taken together, these considerations supported the decision to perform concomitant surgical septal myectomy, resulting in a comprehensive and guideline-consistent surgical strategy.

Associations between hypertrophic cardiomyopathy and cardiac myxoma are rare, with sporadic case reports in the literature. In 2019, Gi et al published a literature review that identified a total of 6 reports [7]. More recently, in 2021, Brzozowski et al [8] published a report on 2 patients with this association who underwent surgical treatment for myxoma resection and septal myectomy. In the report from Brzozowski et al, the first patient had a LVOT gradient of 60 mmHg, and septal myectomy was performed via the transaortic approach. The second patient in this report had an LVOT gradient of 80 mmHg and also required mitral valve replacement. Other existing case reports showed an LVOT gradient less than 30 mmHg or an unavailable gradient, and patients were submitted to surgical treatment for isolated myxoma resection.

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Conclusions

The coexistence of atrial myxoma and hypertrophic cardiomyopathy represents a rare but clinically relevant scenario that demands meticulous anatomical and functional assessment. This case demonstrates that, in selected young patients with favorable prognostic features and significant left atrial remodeling, concomitant septal myectomy at the time of tumor resection may be a rational and guideline-consistent strategy, even in the presence of subthreshold resting LVOT gradients. While effective in preventing delayed intervention, this approach requires careful perioperative planning. Overall, the case underscores the importance of individualized surgical decision-making in complex structural heart disease.

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Informed Consent

Patient consent was obtained.

Declaration of Figures' Authenticity

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