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Case Report

Obstructive Giant Left Atrial Myxoma Concurrent with Very High-Risk Acute Coronary Syndrome Presenting as Cardiogenic Shock: A Rare Dual Pathology

Muhammad Rizky Felani[1,2], Maria Lerista[3], Sandy Hasan[4], Aswin Nugraha[5], Andina Dewanty[6]

Abstract

Background: Primary cardiac tumors are exceptionally rare, with cardiac myxomas being the most common benign type, typically originating in the left atrium. Despite their benign histopathological nature, these tumors can lead to serious complications, including intracardiac obstruction, embolic events, and even sudden cardiac death. The coexistence of a Left Atrial (LA) myxoma and Coronary Artery Disease (CAD) is an unusual clinical finding. This report highlights the importance of recognizing cardiac myxoma as a potential contributor to high-risk acute coronary syndrome and hemodynamic instability.

Case Illustration: A 57-year-old man presented with recurrent chest pain and shortness of breath, consistent with very high-risk Non–ST Elevation Myocardial Infarction (NSTEMI) and cardiogenic shock. Transthoracic Echocardiography (TTE) revealed a large LA mass causing functional mitral valve obstruction, highly suggestive of a myxoma. Further evaluation with transesophageal echocardiography confirmed a giant, highly mobile mass intermittently occluding the mitral inflow. Coronary angiography revealed significant Two-Vessel Coronary Artery Disease (CAD2VD), including chronic total occlusion of the Left Circumflex Artery (LCx). The patient subsequently underwent urgent combined surgical excision of the tumor and Coronary Artery Bypass Grafting (CABG). Histopathological examination confirmed the diagnosis of cardiac myxoma. The postoperative course was uneventful, with marked clinical improvement, complete resolution of symptoms, and preserved ventricular function on follow-up echocardiography.

Conclusions: This case of very high-risk NSTEMI with cardiogenic shock was further complicated by a large LA myxoma causing dynamic mitral obstruction and diastolic inflow collapse. Early recognition using multimodal imaging and prompt surgical management are essential to prevent catastrophic hemodynamic deterioration and improve patient outcomes.

Keywords: Left Atrial Myxoma, Acute Coronary Syndrome, Echocardiography

[1]Cardiologist, Department of Cardiology and Vascular, Mohammad Hoesin General Hospital, Palembang, Indonesia.
[2]Cardiologist, Department of Cardiology and Vascular, Hermina Hospital, Palembang, Indonesia.
[3]Research Assistant, Department of Cardiology and Vascular, Mohammad Hoesin General Hospital, Palembang, Indonesia.
[4]General Practitioner, Hermina Hospital, Palembang, Indonesia.
[5]Cardiothoracic and Vascular Surgeon, Department of Cardiovascular, Thoracic and Vascular, Mohammad Hoesin General Hospital, Palembang, Indonesia.
[6]Cardiothoracic and Vascular Resident, Department of Cardiovascular, Thoracic and Vascular, Mohammad Hoesin General Hospital, Palembang, Indonesia.

Correspondence:
Muhammad Rizky Felani,

Cardiologist, Department of Cardiology and Vascular, Mohammad Hoesin General Hospital/Department of Cardiology and Vascular, Hermina Hospital, Palembang, Indonesia.

Email: rizky.kardiologiui@gmail.com

Introduction

Primary cardiac tumors are extremely rare, with reported incidence ranging from 0.0017–0.3% in autopsy studies. Among them, cardiac myxomas represent the most common benign tumors, accounting for approximately 50–85% of primary cardiac neoplasms. These tumors most commonly arise from the interatrial septum within the left atrium and typically appear as pedunculated masses. Despite their benign histological nature, cardiac myxomas may lead to severe clinical consequences, including intracardiac obstruction, systemic embolization, arrhythmias, and sudden cardiac death.[1-2]

Left Atrial (LA) myxomas are often termed "great mimickers" because their clinical manifestations can resemble a wide range of cardiovascular diseases, including valvular disease, heart failure, endocarditis, arrhythmias, and embolic syndromes.[3] Diagnosis is typically established by transthoracic or transesophageal echocardiography following clinical suspicion.[4] Symptomatology depends on tumor size, location, and mobility, commonly presenting as a triad of obstruction, embolization, and constitutional symptoms. In a French cohort of 112 patients, mitral obstruction was the predominant finding (67%), followed by embolic (29%) and systemic manifestations (34%). Systemic embolization occurs in up to 50% of cases, most frequently involving the central nervous system, while coronary embolism is rare but can lead to acute coronary syndrome.[2,5] When significant intracardiac obstruction occurs concurrently with acute coronary syndrome, the interaction between these two conditions may create a complex hemodynamic disturbance. This case illustrates the coexistence of dual pathology: an obstructive giant LA myxoma and very high-risk Non–ST Elevation Myocardial Infarction (NSTEMI), whose synergistic effects culminated in cardiogenic shock.

Case Illustration

A 57-year-old man with a prior diagnosis of NSTEMI was urgently transferred by ambulance to the emergency department of our tertiary cardiac center. The referral followed an echocardiographic finding of a giant LA myxoma, necessitating further assessment by coronary angiography, transesophageal echocardiography, and urgent cardiac surgery. During his preceding admission at a secondary care facility, he had reported intermittent left-sided chest pain accompanied by progressively worsening dyspnea over four days. At the time of admission to the first hospital, the patient was receiving Dual Antiplatelet Therapy (DAPT) with aspirin 80 mg once daily and ticagrelor 90 mg twice daily, therapeutic anticoagulation with subcutaneous enoxaparin 60 mg twice daily (1 mg/kg), and, due to worsening hemodynamic status, inotropic support with dobutamine at 5 μg/kg/min was also administered. Additional symptoms included nausea without vomiting, generalized weakness, or loss of consciousness. He was an active smoker with a history of recurrent dyspepsia but denied any other medication use or a family history of hereditary or metabolic disorders.

Upon presentation to the emergency department, the patient appeared dyspneic. His initial vital parameters were notable for a Blood Pressure (BP) of 91/60 mmHg, a regular heart rate of 82 beats per minute, a respiratory rate of 28 breaths per minute, oxygen saturation of 95% on room air, and a body temperature of 36.7°C, but then the BP progressively dropped to a hypotensive level with worsening pulmonary congestion. Chest auscultation revealed fine bilateral rales, while cardiac examination showed normal heart sounds without murmurs or gallops.

The initial Electrocardiogram (ECG) showed ST-segment elevation in lead aVR and widespread ST-segment depression in the precordial leads (V1–V6), consistent with global subendocardial ischemia (Figure 1). Chest radiography demonstrated marked cardiomegaly. Laboratory evaluation revealed elevated cardiac troponin-T, leukocytosis, and hyperglycemia. Initial Transthoracic Echocardiography (TTE) performed at the referring hospital identified a large, obstructive LA myxoma exhibiting hemodynamic features consistent with severe Mitral Stenosis (MS) physiology, accompanied by concentric Left Ventricular (LV) hypertrophy and preserved systolic function (Ejection Fraction [EF] 55% by Simpson's method).

While still on inotropic support, subsequent transesophageal echocardiography in our tertiary hospital had confirmed a mobile LA mass measuring approximately 4.2×2.3 cm, prolapsing into the mitral valve orifice during diastole and causing moderate functional MS (mean gradient 6-7 mmHg), with mild Tricuspid Regurgitation (TR) (with intermediate probability of pulmonary hypertension, TR Maximum Velocity [TR Vmax] 2.9 m/s), and global LV contractility remained normokinetic with EF of 57% (Figure 2) with sufficient stroke volume. Integrating the clinical presentation, physical

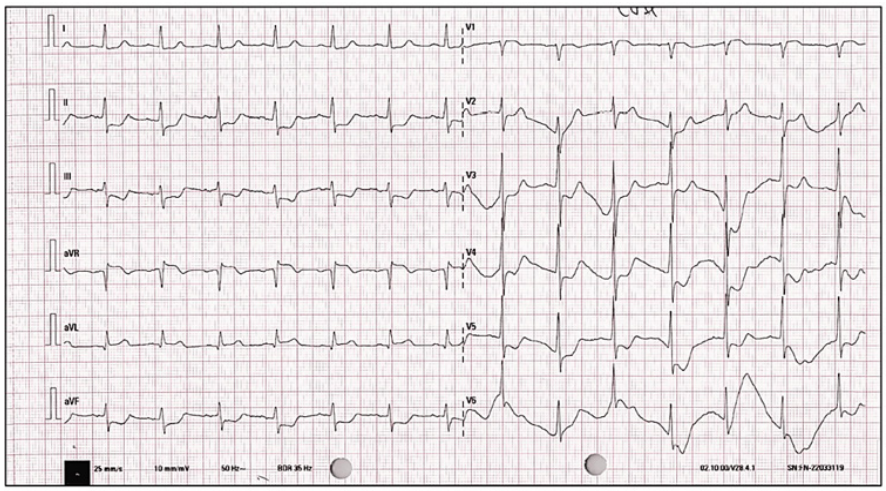


Figure 1. Patient's initial electrocardiography showed diffuse ischemia with ST-segment elevation in aVR.

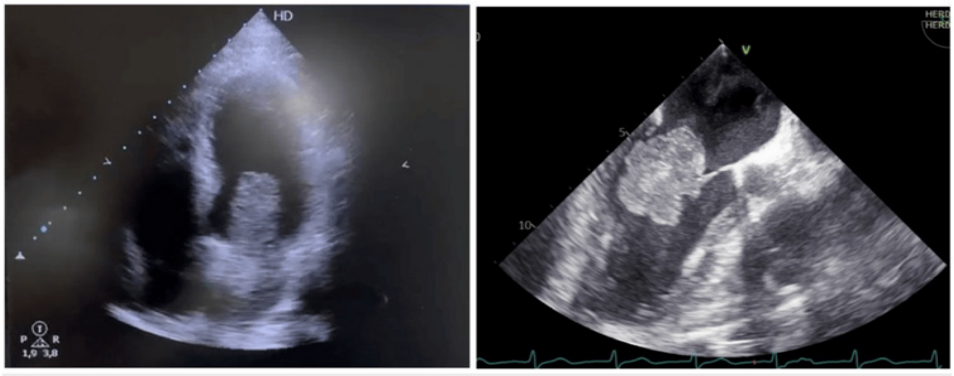


Figure 2. (Left) Initial echocardiography revealed giant LA myxoma with normal LV contractility; (Right) Transesophageal echocardiography (TEE) confirmed giant LA myxoma.



Figure 3. Coronary angiography revealed CAD2VD with left circumflex artery CTO.



Figure 4. (Left) Myxoma extirpated from left atrium; (Right) Post-operative echocardiography evaluation.

findings, and diagnostic investigations, the patient was diagnosed with cardiogenic shock due to very high-risk NSTEMI and giant LA myxoma with MS physiology.

The patient was admitted to the Cardiovascular Care Unit (CVCU) for intensive monitoring and management. During hospitalization, he received continuous dobutamine infusion along with anticoagulation and DAPT. Subsequent coronary angiography demonstrated Two-Vessel Coronary Artery Disease (CAD2VD) with chronic total occlusion of the Left Circumflex Artery (LCx) (Figure 3).

The patient underwent successful surgical excision of a 6 cm LA myxoma combined with Coronary Artery Bypass Grafting (CABG). He was subsequently transferred to the Cardiovascular Intensive Care Unit (CVICU) for post-operative monitoring. Histopathological examination of the excised masses, measuring 6×3×1 cm and 5×3×2 cm, revealed myxoid stroma consistent with cardiac myxoma. Following surgery, the patient's chest pain and dyspnea resolved, and post-operative TTE evaluation demonstrated good biventricular systolic function with a Left Ventricular Ejection Fraction (LVEF) of 56%, mild mitral regurgitation, and no residual LA mass (Figure 4). The patient showed excellent clinical recovery and was discharged home in stable condition after four days of postoperative care.

Discussion

This report describes a 57-year-old man with very high-risk non–ST-elevation myocardial infarction (NSTEMI) in whom a giant LA myxoma was incidentally identified. Cardiac myxomas, though rare, represent the most common primary cardiac tumors, with approximately 75% arising from the left atrium, typically at the fossa ovalis of the interatrial septum. They usually present between the third and sixth decades of life, showing a female predominance and sporadic occurrence, although 5–10% are familial. Clinical manifestations range from intracardiac obstruction and arrhythmias to embolic or constitutional symptoms, while up to half of cases are detected incidentally.[1-2] In this patient, the LA myxoma was discovered during evaluation for NSTEMI and progressive dyspnea. Although coronary embolization is a recognized but uncommon complication of atrial myxoma, the absence of angiographic evidence of acute embolic occlusion and the presence of chronic atherosclerotic lesions—supported by a history of tobacco use suggested that the ischemic event was unrelated to tumor embolization.

Cardiogenic shock is a life-threatening condition with reported mortality rates of 40–72%, most commonly secondary to myocardial infarction.[6] In this case, the patient presented with hypotension (91/60 mmHg) and was stabilized with carefully titrated dobutamine infusion. The shock was likely multifactorial, resulting from both myocardial ischemia and dynamic LV inflow obstruction caused by a large atrial myxoma. Even before the acute coronary syndrome occurred, the large LA myxoma had likely produced intermittent hemodynamic disturbance by partially obstructing the mitral valve orifice during diastole. Myxomas exceeding 5 cm in diameter can mimic severe MS, elevating LA and pulmonary venous pressures while cardiac output remained relatively preserved through compensatory mechanisms.[7] In this patient, transesophageal echocardiography demonstrated a prolapsing LA mass causing functional MS with a mean transmitral gradient of 6–7 mmHg,

findings consistent with combined cardiogenic and obstructive shock physiology. Large atrial myxomas may produce dynamic obstruction of the mitral valve during diastole, thereby limiting LV filling and reducing cardiac output, which can contribute to hemodynamic collapse.

The ECG showed ST-segment elevation in lead aVR with diffuse ST depression (V1–V6), a pattern indicative of left main or severe multivessel CAD. This was confirmed by coronary angiography, which demonstrated two-vessel disease with chronic total occlusion of the LCx.[8] Despite its embolic potential, the myxoma showed no evidence of acute coronary embolization, with angiographic findings consistent with chronic disease. In this patient, the giant LA myxoma produced dynamic obstruction of mitral inflow, mimicking severe MS and compounding the hemodynamic burden of concomitant NSTEMI.[9] The synergistic interaction between mechanical mitral inflow obstruction caused by prolapse of the LA myxoma and myocardial ischemia precipitated cardiogenic shock. Reduced LV filling due to dynamic mitral obstruction, together with impaired contractility, led to a marked decrease in cardiac output and potentially worsened myocardial ischemia. The uniqueness of this case lies in the coexistence of severe CAD and mechanical obstruction from a large atrial myxoma, both contributing synergistically to cardiogenic shock.[2]

In this case, echocardiography incidentally identified a giant LA myxoma obstructing mitral inflow. The patient underwent urgent surgical excision, and histopathological analysis of the resected masses (6×3×1 cm and 5×3×2 cm) confirmed the diagnosis of cardiac myxoma with myxoid stroma. Echocardiography remains the cornerstone of diagnosis, with transthoracic imaging offering ~95% sensitivity and transesophageal echocardiography approaching 100%.[10] While cardiac Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) provide complementary anatomic detail, their roles are largely supportive, whereas chest X-ray and ECG offer limited diagnostic utility. Definitive diagnosis relies on histopathological confirmation, and in familial cases, genetic testing helps identify hereditary associations.[2]

Conclusion

This case highlights the rare coexistence of a giant LA myxoma and very high-risk acute coronary syndrome presenting with cardiogenic shock. Dynamic mitral inflow obstruction caused by the myxoma may significantly exacerbate myocardial ischemia and hemodynamic instability. Multimodal cardiac imaging plays a crucial role in early diagnosis, and prompt surgical intervention remains the definitive treatment to prevent catastrophic outcomes.

List of Abbreviations

BP	Blood Pressure
CABG	Coronary Artery Bypass Grafting
CAD	Coronary Artery Disease
CAD2VD	Two-Vessel Coronary Artery Disease
CT	Computed Tomography
CVICU	Cardiovascular Intensive Care Unit
CVCU	Cardiovascular Care Unit
DAPT	Dual Antiplatelet Therapy
ECG	Electrocardiogram
EF	Ejection Fraction
LA	Left Atrial
LCx	Left Circumflex Artery
LV	Left Ventricular
LVEF	Left Ventricular Ejection Fraction
MRI	Magnetic Resonance Imaging
MS	Mitral Stenosis
NSTEMI	Non–ST Elevation Myocardial Infarction
Vmax	Maximum Velocity
TTE	Transthoracic Echocardiography

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

MRF: Conceptualization, Methodology, Supervision, Project administration, Writing – Review & Editing, and Final approval of the manuscript. ML: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – Original Draft, Writing – Review & Editing, and Final approval of the manuscript. SH: Investigation, Data curation, Formal analysis, Writing – Review & Editing, and Final approval of the manuscript. AN: Investigation, Data curation, Formal analysis, Writing – Review & Editing, and Final approval of the manuscript. AD: Investigation,

Data curation, Writing – Review & Editing, and Final approval of the manuscript. All authors contributed to the interpretation of the data, critically revised the manuscript for important intellectual content, approved the final version to be published, and agreed to be accountable for all aspects of the work.

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Conflict of Interest

The authors declare no conflicts of interest.

Availability of Data and Materials

Not applicable.

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Generative AI and AI-Assisted Technologies in the Writing Process

Authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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