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Giant Left Ventricular Intracavitary Mass: Diagnostic Challenges in Distinguishing Thrombus From Tumor

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Abstract

Giant left ventricular (LV) intracavitary masses are rare and pose significant diagnostic and therapeutic challenges due to their high risk of systemic embolization and sudden death. We report two acute cases illustrating contrasting presentations and outcomes. The first case involved a 70-year-old man with acute bilateral ischemic stroke and a large mobile LV mass in the setting of ischemic cardiomyopathy and concurrent gastric adenocarcinoma; the patient deteriorated rapidly and died before advanced imaging could clarify the nature of the mass. The second case concerned a 52-year-old man presenting with acute coronary syndrome complicated by severe LV dysfunction and an apical aneurysm; echocardiography revealed a large apical mass that resolved completely with anticoagulation, confirming an LV thrombus. These cases highlight the difficulty of distinguishing thrombi from cardiac tumors using transthoracic echocardiography (TTE) alone and underscore the value of cardiac magnetic resonance imaging when feasible. Integrating clinical context, imaging findings, and disease evolution is critical for early recognition and appropriate management of LV masses to reduce the risk of embolic complications.

Categories: Cardiology, Emergency Medicine
Keywords: acute coronary syndrome, cardioembolic stroke, case report, echocardiography, left ventricular intracavitary mass

Introduction

Giant left ventricular (LV) intracavitary masses encompass a broad differential diagnosis, including thrombus, primary or secondary cardiac tumors, valvular or mural vegetations in infective or non-bacterial thrombotic endocarditis, and intracardiac metastatic deposits [1]. Among these entities, distinguishing between a giant LV thrombus and a cardiac tumor is a critical diagnostic challenge, as management strategies and prognosis differ substantially [2]. While primary cardiac tumors are exceptionally rare, secondary metastatic cardiac lesions are 20 to 40 times more frequent [1,2]. Regardless of the underlying etiology, the presence of a large and mobile intracavitary mass carries a high risk of systemic embolization, ischemic stroke, and sudden cardiac death [3,4]. LV thrombus, the most common cause of such masses, remains a clinically significant complication, with TTE-based studies reporting incidences ranging from 0.4% to 2.7% in ST-elevation myocardial infarction (STEMI) patients overall and up to 9% in those with anterior STEMI, while contemporary cardiac magnetic resonance (CMR)-based meta-analyses report incidences of up to 6.3% in STEMI patients overall, rising to approximately 19% in those with anterior STEMI and reduced LV systolic function [5]. Transthoracic echocardiography (TTE) is the standard initial imaging modality for the evaluation of intracardiac masses; however, it remains a suboptimal screening tool for LV thrombi, with reported sensitivities as low as 21% to 47% [6]. This diagnostic performance is highly dependent on image quality, thrombus morphology, and sensitivity is notably lower for non-apical or mural thrombi compared with apical, protruding ones, and the timing of the examination, with detection rates substantially improved by repeated imaging or the use of contrast echocardiography [5,7]. Consequently, cardiac magnetic resonance imaging has emerged as the gold standard, providing superior tissue characterization that allows differentiation between avascular thrombi and perfused, contrast-enhancing tumors [8,9]. This article analyzes two acute clinical presentations to highlight the diagnostic challenges and clinical lessons learned from these LV intracavitary masses.

Case Presentation

Observation 1

Patient Information

A 70-year-old man with a history of hypertension and type 2 diabetes mellitus, treated with long-term amlodipine and metformin, presented to the intensive care unit with acute respiratory distress and impaired consciousness.

Clinical Findings

On admission, his Glasgow Coma Scale score was 8/15, oxygen saturation was 85% on room air, and respiratory rate was 35 breaths per minute. Neurological examination revealed a right-sided sensorimotor deficit, while pulmonary auscultation showed bilateral crackles in the lower lung fields, and cardiac examination was unremarkable. Electrocardiography showed a sinus rhythm with a complete left bundle branch block.

Timeline of Current Episode

The sequence of key events during the patient's hospitalization is summarized in Table *1*.

Day	Event
Day 0	Admission for respiratory distress and impaired consciousness; intubation required due to combined neurological and respiratory failure
Day 0	Cerebral and thoracic computed tomography: bilateral acute ischemic stroke and bilateral pneumonia
Day 0	Lab tests: severe anemia (Hb 8 g/dL), normal coagulation profile, elevated D-dimer (2700 ng/mL), negative troponin; ECG: sinus rhythm with complete LBBB
Day 1	heterogeneous, mobile LV apical mass measuring 33 mm × 46 mm, with preserved LV ejection fraction (53%) and isolated apical akinesia
Day 2	Upper gastrointestinal endoscopy: large ulcerative gastric mass; biopsy confirmed gastric adenocarcinoma
Day 3	Development of septic shock; patient died 72 hours after admission

TABLE 1: Timeline of key events during hospitalization of the first patient

TTE, transthoracic echocardiography; ECG, electrocardiogram; LBBB, left bundle branch block

Diagnostic Assessment

Initial cerebral and thoracic computed tomography demonstrated an acute bilateral ischemic stroke and bilateral aspiration pneumonia. TTE performed as part of the cardioembolic stroke workup revealed a large, mobile, heterogeneous LV apical mass measuring 33 mm × 46 mm in the context of ischemic cardiomyopathy, with a preserved LV ejection fraction of 53% and isolated apical akinesia. The mass adhered to the mid-segment of the lateral and inferoseptal walls (Figure *1*). Laboratory tests showed severe anemia (hemoglobin 8 g/dL), a normal coagulation profile, and markedly elevated D-dimer levels (2700 ng/mL), with negative cardiac troponins (Table *2*). Upper gastrointestinal endoscopy revealed a large ulcerative exophytic gastric mass, and histopathology confirmed gastric adenocarcinoma.

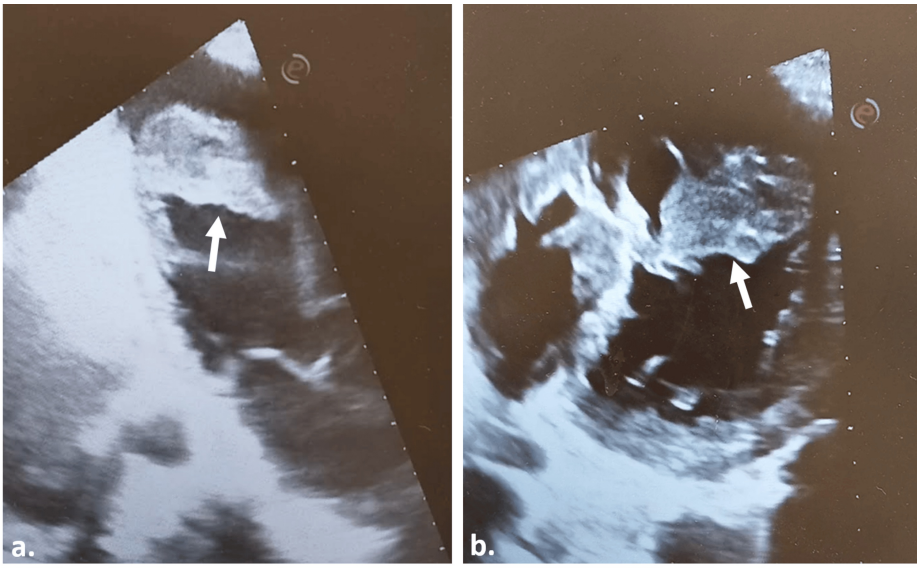


FIGURE 1: TTE showing a large, heterogeneous LV apical mass adherent to the mid-segment of the lateral and inferoseptal walls

(a) Apical three-chamber view showing the LV apical mass (arrow).

(b) Modified apical four-chamber view confirming the heterogeneous, mobile nature of the mass (arrow), adherent to the inferoseptal and lateral walls.

LV, left ventricular; TTE, transthoracic echocardiography

Laboratory parameter	Patient 1	Patient 2	Normal range
Hemoglobin (g/dL)	8.0 ↓	Normal	13.5-17.5 (men)
Cardiac troponin (hs-TnT, pg/mL)	Negative	57 ↑	<14
D-dimer (ng/mL)	2700 ↑	Not performed	<500
Coagulation profile (PT/aPTT)	PT: 82% aPTT: 0.9	PT: 92% aPTT: 1	PT 70%-100%, aPTT ratio 0.8-1.2

TABLE 2: Laboratory results for both patients with normal reference ranges

hs-TnT: high-sensitivity troponin T; PT: prothrombin time; aPTT: activated partial thromboplastin time; ↑: above normal range; ↓: below normal range

Diagnosis

Differentiating between an LV thrombus and a cardiac metastasis was challenging. Advanced cardiac imaging could not be performed due to the patient's rapid clinical deterioration, leaving the definitive nature of the mass undetermined. Tumor staging for the newly diagnosed gastric adenocarcinoma could also not be performed prior to the patient's death.

Therapeutic Interventions

Despite intensive care measures, including endotracheal intubation, mechanical ventilation, broad-spectrum antibiotics, and vasoactive support for septic shock, the patient's condition continued to worsen.

Follow-Up and Outcome of Interventions

The patient died 72 hours after admission. No follow-up was possible given the fatal outcome.

Patient Perspective

The patient's perspective was unavailable due to critical illness.

Informed Consent

Informed consent for publication was obtained from his next of kin.

Observation 2

Patient Information

A 52-year-old man, an active smoker with no other known cardiovascular risk factors or prior medical history, was brought to the emergency department after a syncopal episode during exertion. He reported typical angina-like chest pain 48 hours earlier, lasting approximately 30 minutes, which had spontaneously resolved.

Clinical Findings

On admission, he was hemodynamically, respiratorily, and neurologically stable, with a blood pressure of 100/60 mmHg, a heart rate of 97 beats per minute, and unremarkable cardiopulmonary and neurological examinations. Electrocardiography showed sinus rhythm with complete right bundle branch block, pathological Q waves in the inferior leads, and diffuse anterior T-wave inversions (Figure *2*).

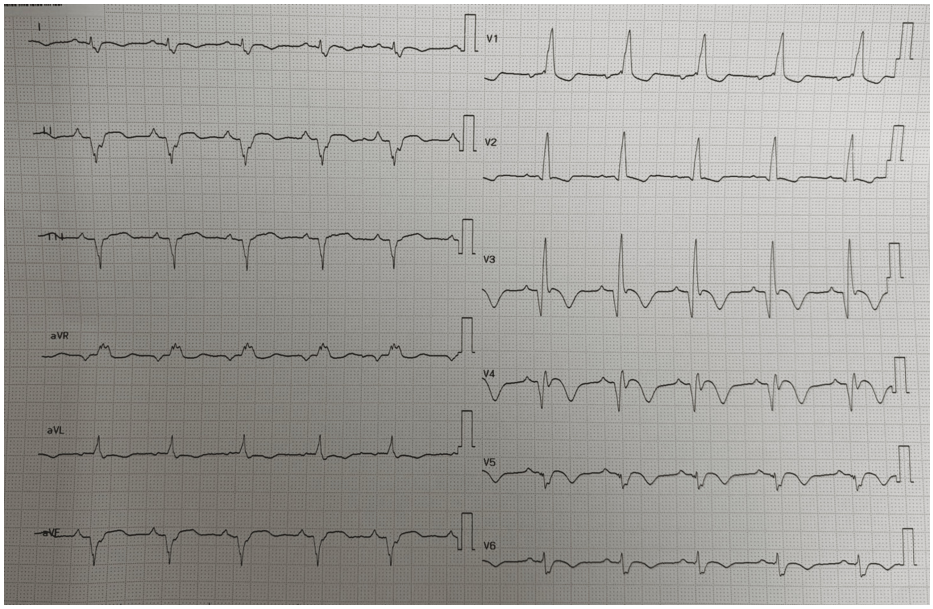


FIGURE 2: ECG showing sinus rhythm with complete right bundle branch block, pathological Q waves in the inferior leads, and diffuse anterior T-wave inversions

ECG, electrocardiogram

Timeline of Current Episode

Key events during hospitalization and follow-up are summarized in Table *3*.

Day	Event
0	Admission after syncope; ECG: right bundle branch block, Q waves, and T-wave inversions; laboratory tests: elevated troponin level
0	TTE: severe LV dysfunction, apical aneurysm, and an LV mass measuring 53 mm × 37 mm
1	Coronary angiography: RCA occlusion and left anterior descending-diagonal stenosis; PCI performed
1-2	Medical therapy initiated: dual antiplatelet therapy with curative-dose enoxaparin (1 mg/kg twice daily)
3	Acenocoumarol started with enoxaparin overlap; target INR 2.0-3.0
5-7	Two INR checks at 48-hour intervals, both within therapeutic range; enoxaparin discontinued
8	Discharged home; no arrhythmias documented during hospital stay
30	Final INR check confirming therapeutic levels; anticoagulation discontinued
90	Follow-up echocardiography: persistent LV dysfunction, unchanged aneurysm, and complete resolution of the mass (confirming LV thrombus); NYHA class II dyspnea, no angina; cardiac rehabilitation completed

TABLE 3: Timeline of key events during hospitalization of the second patient

TTE, transthoracic echocardiography; LV, left ventricular; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RCA, right coronary artery; INR: international normalized ratio

Diagnostic Assessment

Laboratory investigations were within normal limits except for moderately elevated cardiac troponin levels (Table *2*). TTE revealed severe LV systolic dysfunction (ejection fraction of 30%) with a large apical aneurysm extending to the mid-inferior and septal segments, and a large intracavitary mass measuring 53 mm × 37 mm occupying the base of the aneurysm, with small mobile components (Figure *3*). Coronary angiography revealed an acute occlusion of the mid right coronary artery (RCA) and severe proximal left anterior descending-diagonal bifurcation stenosis (Figure *4*).

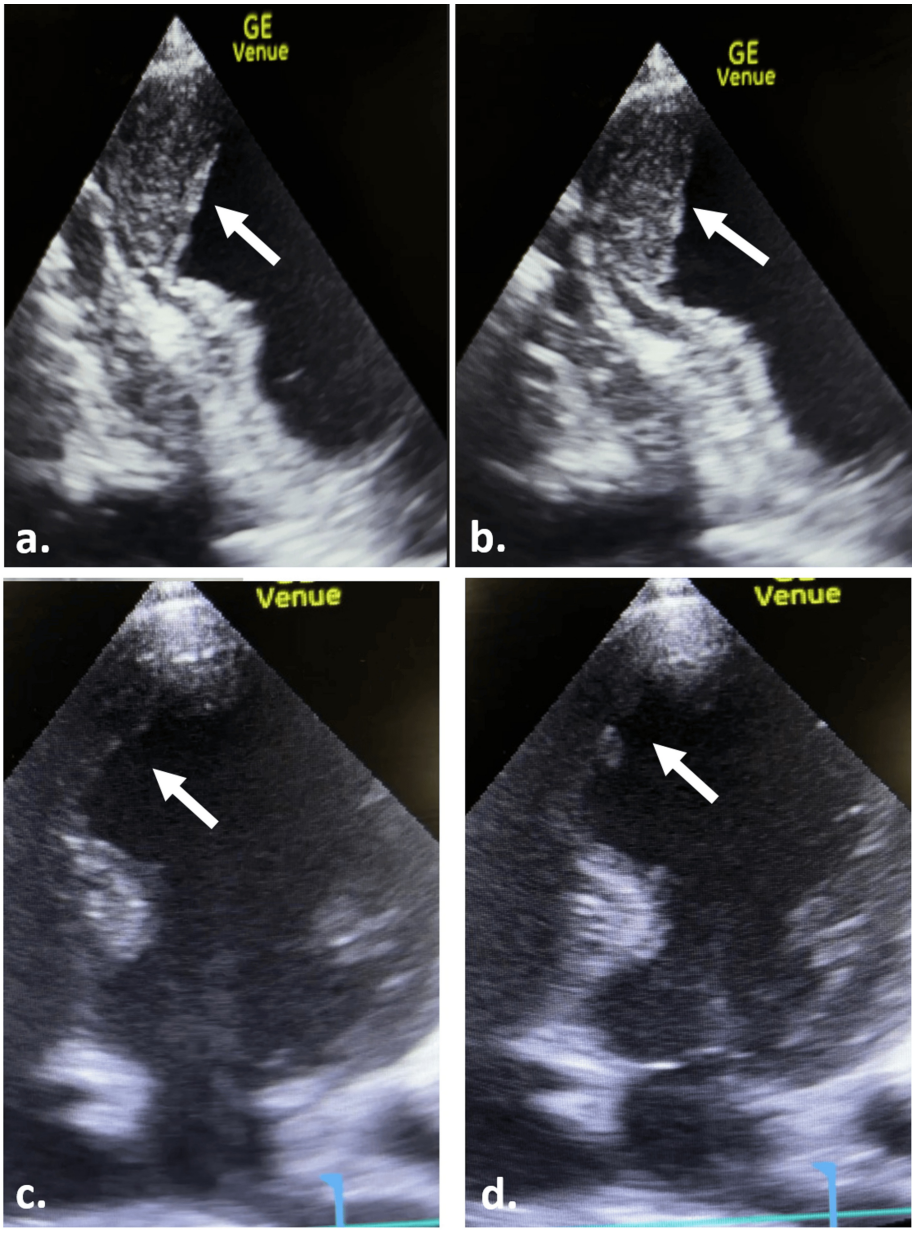


FIGURE 3: TTE images showing an apical two-chamber view in diastole and systole, demonstrating an apical aneurysm with a large intracavitary mass occupying the aneurysm base, and an apical four-chamber view in systole and diastole confirming the apical aneurysm

(a) Apical two-chamber view in diastole; (b) Apical two-chamber view in systole, showing the large intracavitary mass (arrows) occupying the base of the aneurysm; (c) Apical four-chamber view in diastole; (d) Apical four-chamber view in systole, confirming the extent of the apical aneurysm (arrows).

TTE, transthoracic echocardiography; GE, General Electric

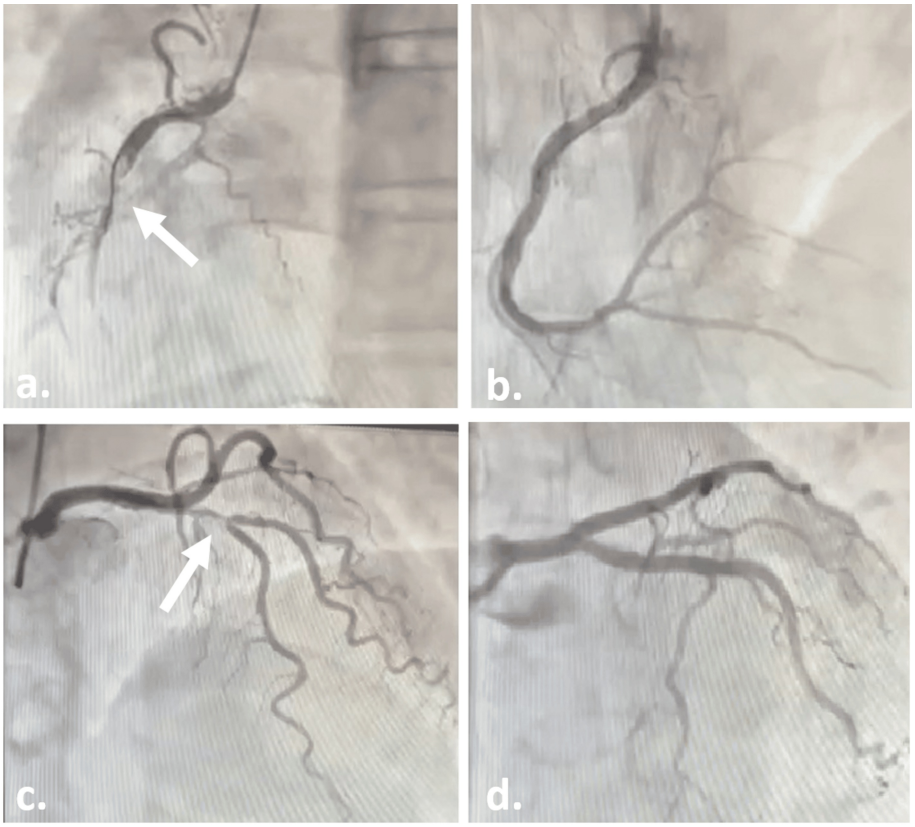


FIGURE 4: Coronary angiography images showing the RCA before and after angioplasty, and the LAD before and after angioplasty

(a) RCA before PCI, demonstrating acute mid-RCA occlusion (arrow).

(b) RCA after successful PCI with restored TIMI 3 flow.

(c) LAD before PCI, showing severe proximal LAD-diagonal bifurcation stenosis (arrow).

(d) LAD after successful PCI.

RCA, right coronary artery; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction; LAD, left anterior descending artery

Diagnosis

The working diagnosis was recent inferior myocardial infarction complicated by severe LV dysfunction, apical aneurysm, and probable LV thrombus. The syncopal episode was attributed to either a transient embolic event or a transient ventricular arrhythmia.

Therapeutic Interventions

Both coronary lesions were successfully treated with percutaneous coronary intervention (PCI) via the radial approach, with implantation of a single drug-eluting stent in the RCA and a single drug-eluting stent in the left anterior descending artery (LAD); no thrombus aspiration was performed, and the procedure was uncomplicated. Medical management included dual antiplatelet therapy (aspirin and clopidogrel), guideline-directed heart failure therapy (sacubitril/valsartan, bisoprolol, empagliflozin, and spironolactone), and anticoagulation with therapeutic-dose enoxaparin (1 mg/kg twice daily). Acenocoumarol was introduced on day 3 with enoxaparin overlap, targeting an INR of 2.0-3.0; two international normalized ratio (INR) measurements performed at 48-hour intervals confirmed therapeutic levels, after which enoxaparin was discontinued. Anticoagulation was maintained for a total of one month, with a final INR measurement prior to discontinuation confirming sustained therapeutic levels.

Follow-Up and Outcome of Interventions

The patient remained in the intensive care unit for three days and then spent five days in a cardiology ward with continuous telemetry, with no documented ventricular arrhythmias, and was discharged on day 8. At three-month follow-up, the patient reported persistent New York Heart Association (NYHA) class II dyspnea without recurrent chest pain or syncope and had completed a structured cardiac rehabilitation program. TTE showed persistent severe LV systolic dysfunction (ejection fraction of 32%) and an unchanged apical aneurysm, but the apical mass had completely resolved, confirming the diagnosis of LV thrombus (Figure 5). Anticoagulation was discontinued, and aspirin was reintroduced.

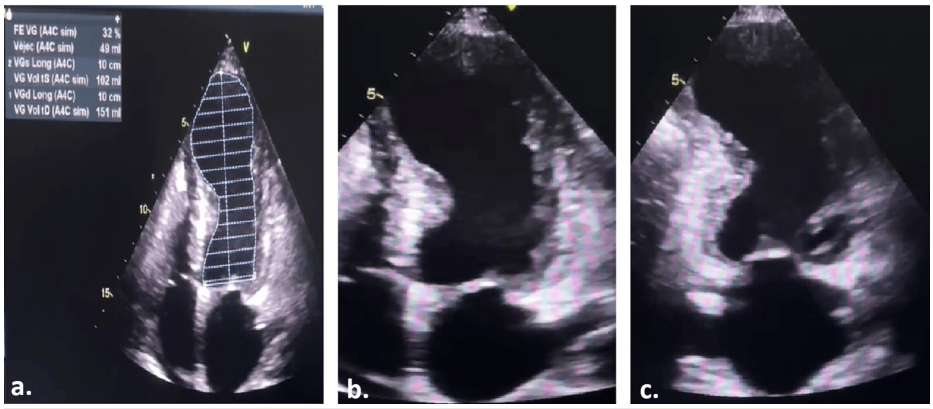


FIGURE 5: TTE at three-month follow-up demonstrating persistent severe LV systolic dysfunction, an unchanged apical aneurysm, and complete resolution of the previously documented apical mass, confirming the diagnosis of LV thrombus

(a) Apical four-chamber view with Simpson's biplane tracing showing preserved LV dimensions (EDV 151 mL, ESV 102 mL) and severely reduced ejection fraction (32%), with no residual intracavitary mass at the apex.

(b) Apical four-chamber view without tracing, confirming the dilated left ventricle with an apical aneurysm and complete absence of thrombus.

(c) Apical two-chamber view further confirming the aneurysmal apex with no residual mass, consistent with complete thrombus resolution following anticoagulation therapy.

LV, left ventricular; TTE, transthoracic echocardiography; EDV, end-diastolic volume; ESV, end-systolic volume

Patient Perspective

The patient expressed satisfaction with the care received.

Informed Consent

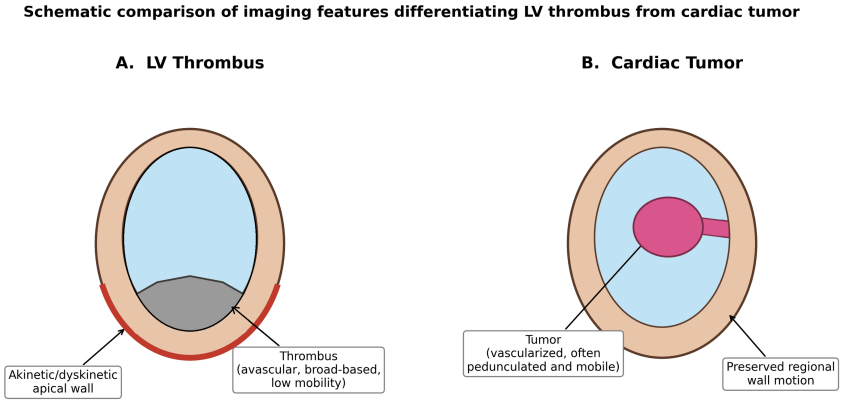
Informed consent for publication was obtained.

Discussion

LV intracavitary masses are uncommon findings and represent a significant diagnostic and therapeutic challenge, particularly in acute clinical settings. The differential diagnosis of a giant LV intracavitary mass primarily involves distinguishing between a thrombus and a tumor (either primary or metastatic), as the clinical prognosis and therapeutic approach for these entities vary substantially [4,8]. LV thrombi most commonly develop in the setting of myocardial infarction or severe LV systolic dysfunction, particularly when apical akinesia or aneurysm formation leads to blood stasis; they are usually contiguous with akinetic or dyskinetic myocardial segments [1,8]. In contrast, if the patient has a known history of malignancy, the likelihood of a secondary metastatic tumor is significantly higher, as cardiac metastases are 20 to 40 times more frequent than primary cardiac tumors [1,2]. These metastases can involve the myocardium or pericardium or, less frequently, present as intracavitary masses, sometimes mimicking thrombi on TTE [2,3].

While TTE is the most common first-line imaging modality due to its wide availability, it is a suboptimal screening tool for LV thrombi, with studies showing that it may miss roughly half of all cases and with reported sensitivities as low as 21% to 47% [1,6]. Cardiac magnetic resonance imaging has emerged as the gold standard imaging modality because it offers superior tissue characterization and high-resolution assessment of cardiac structures [1,9]. The most effective method for differentiating these masses using cardiac magnetic resonance imaging is late gadolinium enhancement; thrombi typically appear as non-

enhancing masses due to their avascular composition, whereas tumors generally show perfusion and enhancement [1,8]. Specifically, a pattern of hyper-isointensity with short inversion times and hypointensity with long inversion times on the TI scout has demonstrated a diagnostic accuracy of approximately 93% [8]. These and other key imaging and clinical features differentiating LV thrombus from cardiac tumor are summarized in Figure *6* [1,4,6].



Feature	Thrombus	Tumor
Late gadolinium enhancement (CMR)	Absent (avascular)	Present (vascularized)
Mobility	Usually low/absent	Often high (esp. myxoma)
Response to anticoagulation	Resolution/regression	No change
Typical clinical context	Recent MI, severe LV dysfunction, AF	Known malignancy or isolated finding

FIGURE 6: Schematic comparison of key imaging features differentiating LV thrombus from cardiac tumor. (A) LV thrombus typically appears as an avascular, broad-based mass with low mobility, located in continuity with an akinetic or dyskinetic myocardial segment. (B) Cardiac tumors are typically vascularized and may be pedunculated and mobile, without an associated regional wall motion abnormality

MI, myocardial infarction; LV, left ventricular; CMR, cardiac magnetic resonance; AF, atrial fibrillation

Image created using Microsoft Paint (Microsoft Corporation, Redmond, WA, USA) and Microsoft PowerPoint (Microsoft Corporation, Redmond, WA, USA).

In the first case, a giant LV intracavitary mass was identified during the etiological workup of acute bilateral ischemic stroke. The embolic pattern of cerebral infarction strongly suggested a cardioembolic mechanism. However, the patient's history of gastric adenocarcinoma raised the possibility of cardiac metastasis. TTE alone was insufficient to definitively differentiate between thrombus and tumor, as both entities may appear as mobile, heterogeneous intracavitary masses. Primary cardiac tumors, such as myxoma, were considered less likely given the mass's location within the LV apex in a region of ischemic myocardium, an atypical site for myxoma, which arises almost exclusively from the interatrial septum, narrowing the differential diagnosis primarily to thrombus versus metastatic involvement. Cardiac magnetic resonance imaging, which could have provided tissue characterization to resolve this distinction, could not be performed due to the patient's rapid clinical deterioration and death, and no autopsy was carried out, leaving the definitive nature of the mass undetermined; furthermore, systemic embolization to other organs, such as the spleen or kidneys, could not be excluded, as no additional imaging was performed. This case illustrates the diagnostic uncertainty frequently encountered in critically ill patients, where clinical severity may preclude comprehensive etiological assessment, and underscores the importance of pursuing CMR early in the diagnostic workup of LV masses, together with additional imaging, such as abdominal CT, or autopsy when feasible, to clarify the nature of the mass and screen for silent systemic emboli.

In contrast, the second case represents a typical and confirmed presentation of LV thrombus in the setting of acute coronary syndrome complicated by severe LV dysfunction and apical aneurysm. The presence of a large, immobile intracavitary mass in a region of akinesia, combined with its complete resolution under anticoagulation on follow-up TTE, allowed a definitive diagnosis of LV thrombus to be established. Primary cardiac tumors, such as myxoma, were also considered unlikely given their rarity in the LV, where they typically arise from the atrial septum rather than the ventricular apex. Although tissue confirmation or CMR

with late gadolinium enhancement was not performed, complete resolution under anticoagulation is widely accepted as a reliable diagnostic criterion for LV thrombus, as no other intracavitary mass would be expected to respond in this manner; when CMR is unavailable or unfeasible, contrast-enhanced echocardiography represents a valuable alternative, improving thrombus detection sensitivity compared to standard TTE [7]. This case highlights the importance of systematic echocardiographic evaluation in patients with myocardial infarction and reduced LV function, as early detection of LV thrombus may allow timely initiation of anticoagulation and prevention of embolic complications. A further limitation of this case is the relatively short follow-up period of three months, which does not allow assessment of long-term thrombus recurrence or late embolic events; extended follow-up of at least six to 12 months would be desirable in future similar cases.

The management of a giant LV intracavitary mass is highly dependent on its underlying etiology; however, the immediate priority across all presentations is the prevention of life-threatening systemic embolization, such as stroke, myocardial infarction, or mesenteric ischemia. Whether the mass is ultimately determined to be a thrombus or a tumor, the presence of a large and mobile intracavitary mass is frequently considered a primary indication for surgical removal to mitigate the high risk of sudden death or massive embolism [3,4]. For masses confirmed to be thrombi, the cornerstone of treatment is anticoagulation therapy. A significant reduction in mass size following the initiation of anticoagulation can retrospectively confirm the diagnosis of a thrombus [1,8]. Several reports have demonstrated that even large LV thrombi can resolve completely with appropriate anticoagulation, as observed in our second patient [4]. The management of cardiac tumors is primarily dictated by whether they are primary or secondary, although surgery remains a key consideration for any large, mobile mass to prevent life-threatening embolism or sudden death [3,4]. For primary cardiac tumors, prompt surgical resection is usually indicated upon presumptive diagnosis [1]. In cases of metastatic cardiac tumors, management is more complex and must be individualized, with treatment primarily directed toward the underlying malignancy. Because these masses carry a high risk of systemic embolization, prophylactic anticoagulation is often recommended for eligible patients [1,2].

When diagnosed and treated promptly, LV thrombi generally carry a favorable prognosis, with complete resolution achievable under appropriate anticoagulation therapy and low recurrence rates when the underlying cause is adequately managed [4]. Ultimately, the prognosis of secondary cardiac tumors is largely determined by the stage and biological behavior of the primary cancer, and management may shift toward palliative or supportive care in advanced disease [2]. Taken together, these two cases illustrate the heterogeneous presentation of giant LV intracavitary masses and emphasize the importance of integrating clinical context, imaging findings, and disease evolution when establishing a diagnosis. They also highlight the real-world limitations encountered in acute and critical care settings, where diagnostic certainty may not always be achievable. Early recognition of LV masses and awareness of their potential etiologies remain essential to guide management and improve patient outcomes.

Conclusions

These two cases of giant LV intracavitary masses, one remaining diagnostically unresolved due to rapid clinical deterioration in the setting of concurrent gastric adenocarcinoma, and one diagnosed as LV thrombus based on its complete resolution under anticoagulation, illustrate the profound diagnostic challenge these lesions pose in acute clinical settings. Differentiating between thrombus and cardiac tumor is crucial, as management strategies and prognosis differ markedly, and TTE alone is frequently insufficient to achieve diagnostic certainty. The key lesson from these cases is that integrating clinical context, echocardiographic findings, and disease evolution is essential, and that cardiac magnetic resonance imaging should be pursued whenever feasible to guide appropriate and timely management. As these observations are derived from two individual cases, they should be interpreted as illustrative rather than generalizable, and further studies are needed to refine diagnostic algorithms for giant LV intracavitary masses.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Amal Baicha, Khaoula Aboubakr, Najat Mouine, Zouhair Lakhal, Aatif Benyass

Acquisition, analysis, or interpretation of data: Amal Baicha, Khaoula Aboubakr

Drafting of the manuscript: Amal Baicha, Khaoula Aboubakr

Critical review of the manuscript for important intellectual content: Amal Baicha, Najat Mouine, Zouhair Lakhal, Aatif Benyass

Supervision: Najat Mouine, Zouhair Lakhal, Aatif Benyass

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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