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Case Reports

Large Left Ventricular Pseudoaneurysm Presenting as an Embolic Stroke After a "Silent" Myocardial Infarction

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Abstract

A 72-year-old woman with no history of coronary artery disease presented with an acute left middle cerebral artery stroke and was found to have a large left ventricular pseudoaneurysm measuring 8.7 × 7.6 cm and 2 large left ventricular thrombi, the source of her systemic embolization. Despite initial medical management, she developed refractory New York Heart Association functional class III heart failure, uncontrolled atrial fibrillation, and further enlargement of her pseudoaneurysm to 5.5 × 10.6 × 9.2 cm. She underwent urgent aneurysmectomy. Left ventricular pseudoaneurysms are rare and most commonly occur following an acute myocardial infarction when a ventricular free-wall rupture is contained by pericardium or thrombi. Historically, left ventricular angiography displaying a lack of an overlying coronary artery was the gold standard for diagnosis. Now, noninvasive imaging such as computed tomography, magnetic resonance imaging, and echocardiogram with ultrasound-enhancing agent, are reliable diagnostic tools. They can distinguish a pseudoaneurysm from a true left ventricular aneurysm using characteristic findings such as a narrow aneurysm neck, bidirectional doppler flow between the pseudoaneurysm and the left ventricle, and abrupt changes in the cardiac wall structures. Progressive dilation, wall thinning, and dyskinesis can result in refractory heart failure, arrhythmias, and thrombi formation from venous stasis. Pseudoaneurysms have a 30% to 45% risk of rupture and can be treated with left ventricular aneurysmectomy.

Keywords: Pseudoaneurysm; embolic stroke; myocardial infarction

Introduction

Left ventricular (LV) free-wall rupture is a rare and often fatal complication following an acute myocardial infarction (MI).[1,2] An LV pseudoaneurysm is formed when the free-wall rupture is contained by the pericardium, thrombotic material, or scar tissue.[1,3] The most common locations of pseudoaneurysms within the left ventricle are posterior (43%), lateral (28%), and apical (24%), with a median diameter of 6.0 cm.[3] Because of the increased radius of the pseudoaneurysm and progressive wall thinning, there is a high risk (30%-45%) of rupture.[3,4] In this report, the authors present a unique case of "silent" MI in a 72-year-old woman whose initial presentation was that of an embolic stroke as a result of thrombus that was caused by stasis from a large LV pseudoaneurysm. This pseudoaneurysm rapidly expanded, reaching 10.6 cm in diameter within 5 months, and contributed to the onset of heart failure symptoms that align with New York Heart Association (NYHA) functional class III. The patient underwent emergent aneurysmectomy and coronary artery bypass grafting.

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Case Report

A 72-year-old woman with a history of insulin-dependent, type 2 diabetes mellitus, hypertension, and hyperlipidemia was admitted to another hospital with aphasia and right upper-extremity weakness. A computed tomography (CT) scan of the head was performed and showed a large left middle cerebral artery territory ischemic infarction. She underwent a successful thrombectomy with no postoperative neurologic deficits. On further evaluation for the etiology of her stroke, she was found to have new-onset atrial fibrillation (AF) with a CHA_2DS_2-VASc (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, stroke or transient ischemic attack, vascular disease, age 65 to 74 years, sex category) score of 8. A transthoracic echocardiogram (TTE) showed mildly reduced LV systolic function with an LV ejection fraction (LVEF) of 48%, a dyskinetic apex, and a large apical pseudoaneurysm measuring 8.7 × 7.6 cm. Within the LV apex, there were 2 large thrombi measuring 2.7 × 1.3 cm and 1.25 × 0.85 cm that were visualized using DEFINITY, an ultrasound-enhancing agent (UEA; Lantheus; Fig. 1 and Fig. 2). Cardiology and cardiothoracic surgery documentation from the hospitalization was unavailable for review, but it was understood that she did not undergo a coronary angiogram at that time. At discharge, she was prescribed carvedilol, amiodarone, atorvastatin, furosemide, and apixaban. Based on this information, the authors of this report presumed that the patient experienced a "silent" and "missed" MI.

In the following 5 months after discharge, the patient was admitted to outside hospitals with 3 episodes of AF with a rapid ventricular response and acute-on-chronic systolic heart failure exacerbations with NYHA functional class III symptomatology, which required intravenous diuresis.

Abbreviations and Acronyms

ACC	American College of Cardiology
AHA	American Heart Association
AF	atrial fibrillation
CT	computed tomography
LV	left ventricular
LVEF	LV ejection fraction
MI	myocardial infarction
NYHA	New York Heart Association
RCA	right coronary artery
TTE	transthoracic echocardiogram
UEA	ultrasound-enhancing agent

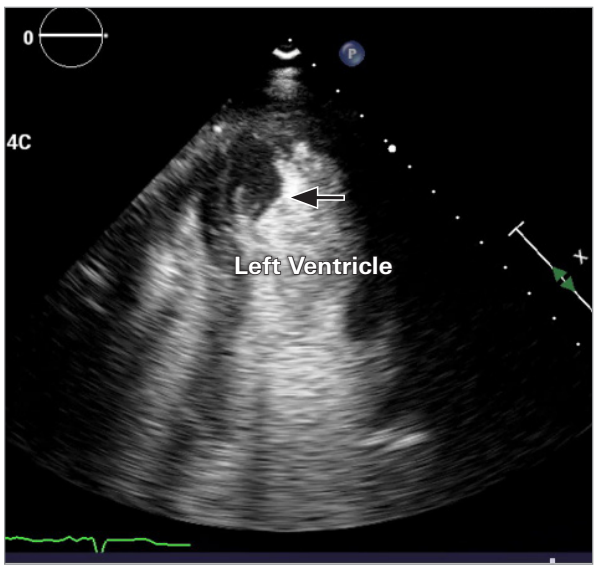

Fig. 1 Initial apical 2-chamber transthoracic echocardiogram with an ultrasound-enhancing agent shows a thrombus measuring 2.7 × 1.3 cm (arrow) within the apex of the left ventricle. Not visualized is an additional 1.25 × 0.85 cm thrombus and the pseudoaneurysm measuring 8.7 × 7.6 cm.

Supplemental motion image is available for Figure 1.

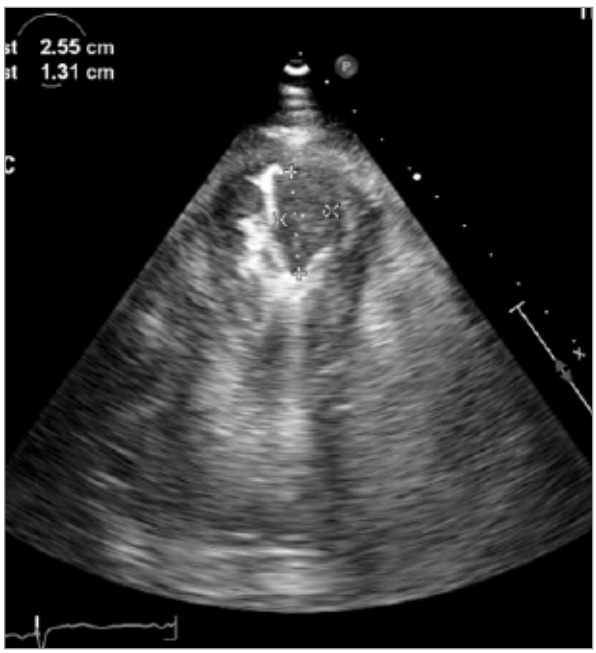

Fig. 2 Initial apical 2-chamber transthoracic echocardiogram with an ultrasound-enhancing agent shows both thrombi, measuring 1.25 × 0.85 cm (left) and 2.7 × 1.3 cm (right), within the apex of the left ventricle.

The patient was readmitted 5 months after the index admission to the Cleveland Clinic with chest pain and palpitations. Her presenting rhythm was AF but converted to normal sinus rhythm with atrioventricular nodal blocking agents. She was hemodynamically stable and euvolemic on physical examination. Cardiac enzymes were mildly elevated with a troponin T level of 0.148 ng/mL (reference, 0.0-0.029 ng/mL) and a level of 0.138 ng/mL measured 6 hours later, as well as an N-terminal pro b-type natriuretic peptide level of 9,379 pg/mL (reference, <125 pg/mL). An electrocardiogram showed no new repolarization abnormalities. Physical examination was not notable for any murmurs, gallops, or friction rubs. Repeat TTE with a UEA showed an LVEF of 40% and a large apical pseudoaneurysm (Fig. 3, Fig. 4, and Fig. 5), with a ratio of 0.36 comparing the neck diameter and the maximal internal diameter of the pseudoaneurysm. The LV was dilated with an LV end-diastolic diameter of 5.6 cm, mild (grade 1) mitral regurgitation secondary to leaflet tethering, and normal right ventricular systolic function with estimated right ventricular systolic pressure of 27 mm Hg. The prior thrombi had resolved with anticoagulation. Coronary

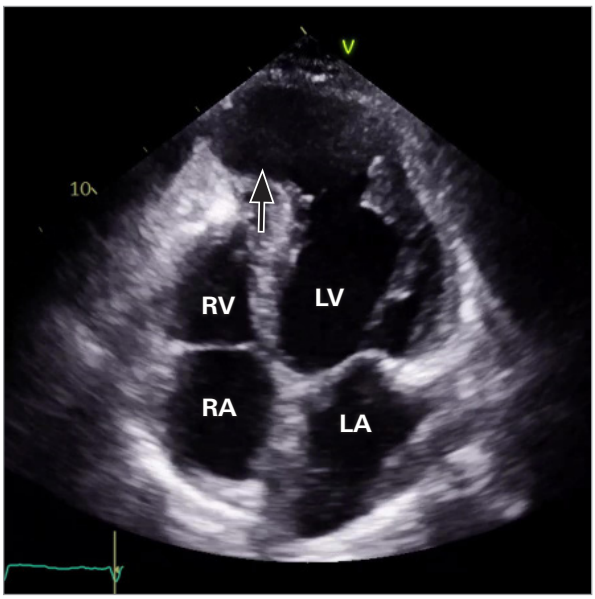

Fig. 3 Repeat apical 4-chamber transthoracic echocardiogram without UEA shows a large apical pseudoaneurysm (arrow) that measured 10.6 × 9.2 cm, 5 months after the index admission. The 2 prior thrombi had resolved but were also not visualized using UEA (thus not shown).

Supplemental motion image is available for Figure 3.

LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; UEA, ultrasound-enhancing agent.

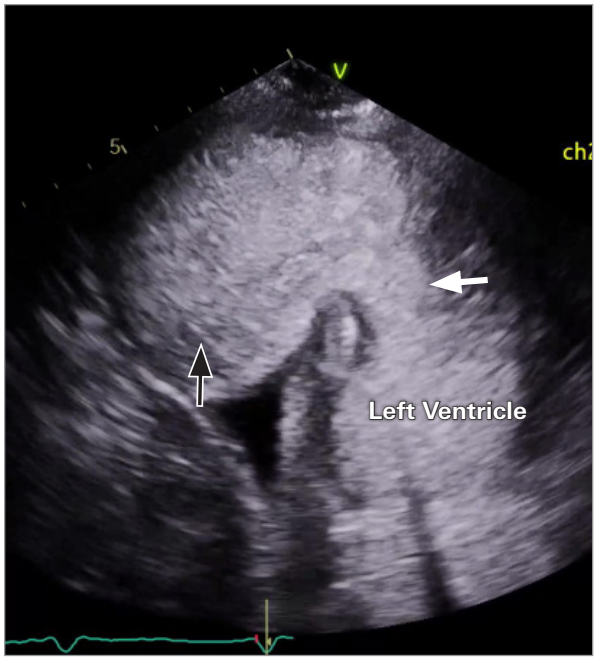

Fig. 4 Repeat apical 2-chamber transthoracic echocardiogram with an ultrasound-enhancing agent shows a large apical pseudoaneurysm (black arrow; 10.6 × 9.2 cm) with a narrow neck (white arrow), 5 months after the index admission.

Supplemental motion image is available for Figure 4.

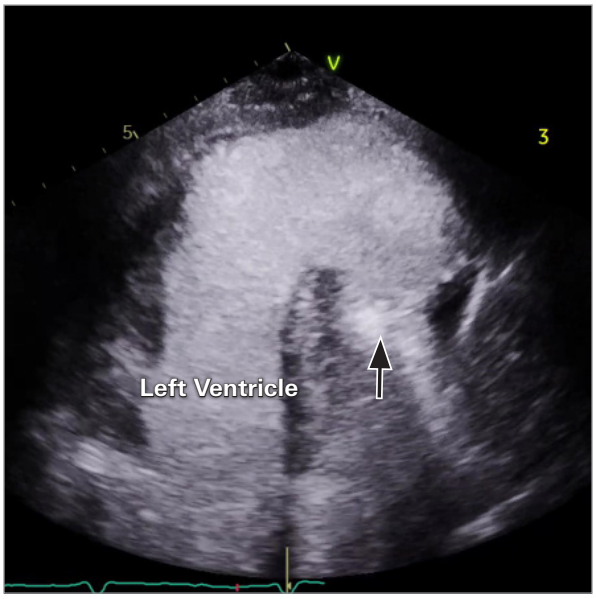

Fig. 5 Repeat apical long-axis transthoracic echocardiogram with an ultrasound-enhancing agent shows a large apical pseudoaneurysm (arrow; 10.6 × 9.2 cm), 5 months after the index admission.

Supplemental motion image is available for Figure 5.

angiography showed triple-vessel coronary artery disease with a focal 70% lesion in the proximal left anterior descending artery, a 30% focal stenosis of the mid left circumflex artery, and an eccentric tubular 50% stenosis in the mid-right coronary artery (RCA). Left ventricular angiography redemonstrated the large LV pseudoaneurysm, which was 5.5×10.6×9.2 cm, as measured by a nongated chest CT scan.

The patient was urgently evaluated by a cardiothoracic surgeon and underwent surgery within 24 hours. The patient underwent an LV pseudoaneurysm resection and repair using a Gore-Tex patch (Gore Medical), a 2-vessel coronary artery bypass surgery (left internal mammary artery graft to left anterior descending artery and saphenous vein graft to the distal RCA), transseptal mitral valve repair (#29 Duran band; Medtronic), cryoablation of the left and right atria, and implantation of a left atrial appendage closure device (an #45 AtriClip; AtriCure). Intraoperatively, she was found to have an anomalous course of the RCA through the right atrium, which was unroofed before bypass grafting. Intraoperative pathology from the LV pseudoaneurysm sac showed granulation tissue and organizing thrombus. She was extubated the following day and weaned from catecholamine support by postoperative day 7. A repeat TTE on postoperative day 3 showed the LV repair with an LVEF of 33% and an LV end-diastolic diameter of 6.7 cm (Fig. 6 and Fig. 7).

Discussion

Rupture of the myocardium during an MI can result in the formation of a true aneurysm or, less commonly, a false pseudoaneurysm.[1] Higher systolic blood pressures predispose the left ventricle to rupture more frequently than the right ventricle or interventricular septum.[5] A true aneurysm is an "outpouching" that involves the entire ventricular wall and includes all layers of cardiac tissue, including the endocardium, a thinned myocardium, and pericardium.[1,2] In contrast, a pseudoaneurysm is the result of a ventricular free-wall rupture that is contained by either overlying pericardium, thrombotic material, or scar tissue.[1] Pseudoaneurysms, therefore, lack any endocardium or myocardium, which results in an abrupt decrease in ventricular wall thickness and a lack of overlying coronary arteries.[6,7] Pseudoaneurysms are most frequently located in the inferior or posterior lateral wall following MI.[8] Between 40% and 65% of LV aneurysms develop a thrombus because of venous stasis with aneurysm.[9,10]

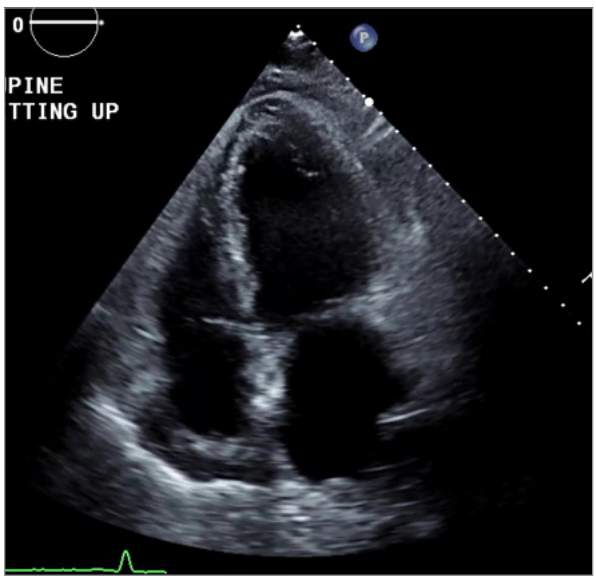

Fig. 6 Postoperative day 3 apical 4-chamber transthoracic echocardiogram without an ultrasound-enhancing agent following aneurysmectomy, with an estimated left ventricular ejection fraction of 33% and an left ventricular end-diastolic diameter of 6.7 cm.

Supplemental motion image is available for Figure 6.

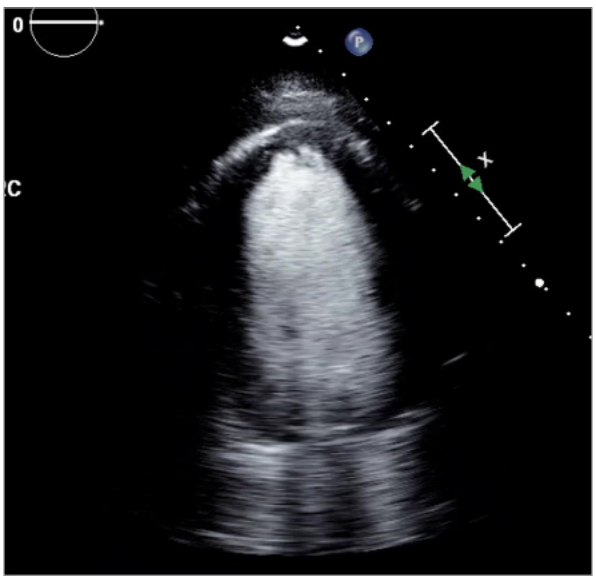

Fig. 7 Postoperative day 3 apical 2-chamber transthoracic echocardiogram with ultrasound-enhancing agent following aneurysmectomy, with estimated left ventricular ejection fraction of 33% and an left ventricular end-diastolic diameter of 6.7 cm.

Supplemental motion image is available for Figure 7.

Because of an increased radius and decreased wall thickness caused by the aneurysm or pseudoaneurysm, tension results in progressive thinning following the law of Laplace.[2] True aneurysms have a higher rate of rupture early in the course but stabilize with ongoing fibrosis and scarring of the ventricular wall.[5] Pseudoaneurysms have a 30% to 45% risk of rupture even following the later fibrous stages.[3,4,11] An aneurysm is noncontractile and can result in systolic heart failure, ventricular arrhythmias, or predisposition to arterial thrombi from stasis.[3,8] A minority of patients, up to 12%, with LV pseudoaneurysms are asymptomatic.[3]

The first historical diagnostic tests for the evaluation of LV aneurysms were angiography and ventriculography, which showed the aneurysmal avascular wall.[7] Echocardiography, initially M-mode then 2-dimensional, allowed for noninvasive evaluation of aneurysms via visualization of abrupt changes in the endocardial border and the saccular dilation of the aneurysm.[12,13] Classically, LV pseudoaneurysms have a narrow neck connecting the larger pseudoaneurysm to the ventricle, whereas true aneurysms have a wider neck.[1] The ratio between the neck diameter and the maximal internal diameter of the pseudoaneurysm is often less than 0.5, whereas true aneurysms generally have a ratio of 0.9 to 1.0.[3-5,12] Use of Doppler and color-flow ultrasound allows the visualization of bidirectional flow between the neck of the aneurysm and the LV cavity, from the LV to the pseudoaneurysm during systole, and from the pseudoaneurysm to the LV during diastole.[3-5] A UEA, such as Lantheus' DEFINITY, can aid in the visualization of the apex, estimation of the aneurysm size, and identification of a thrombus.[14] Computed tomography similarly shows the anatomic boundaries of the pseudoaneurysm as a loss of enhancement in the endocardial wall at the base of the pseudoaneurysm.[5,8] Magnetic resonance imaging allows for precise identification of the components of the ventricular wall and can differentiate between pericardium, myocardium, and thrombus, wherein a pseudoaneurysm is only contained by the pericardium or thrombus.[5,8] Marked delayed gadolinium enhancement of the pseudoaneurysm pericardium can further distinguish a false aneurysm and can evaluate the size of the infarction.[6,15] The 2021 American Heart Association (AHA) scientific statement on mechanical complications of acute MI suggests the use of CT or echocardiography for initial diagnosis of a pseudoaneurysm.[16]

Differentiation between LV aneurysms and pseudoaneurysms is critical because pseudoaneurysms have a high propensity to rupture without surgical intervention.[3,4] Following the introduction of fibrinolysis, rates of post–ST-segment elevation MI LV aneurysms significantly decreased from approximately 18.8% to 7.2%, owing to the reduction of myocardial necrosis.[17,18] The incidence of mechanical complications with modern revascularization techniques, including primary percutaneous coronary intervention, is significantly lower, occurring in approximately 0.27% to 0.91% of patients.[18] The incidence of LV pseudoaneurysms is not well delineated. The 2004 American College of Cardiology (ACC) and AHA practice guideline for the management of ST-segment elevation MI cites a class IIa recommendation (level B evidence) for LV aneurysmectomy and coronary artery bypass graft surgery in patients with LV aneurysm and concomitant heart failure or ventricular tachyarrhythmias that are not responsive to medical therapy.[17] The 2013 ACC/AHA update does not give any formal surgical recommendations but notes that surgery may be considered in patients who remain refractory to medical management, including recurrent thromboembolism.[19] Alternative aneurysm closure options include patches (such as Dacron, Gore-Tex, or pericardium) or pledgeted sutures.[16] Appropriate timing of the aneurysmectomy is unknown, although Prêtre et al[20] recommended urgent surgical repair in patients with acute pseudoaneurysms and elective surgical repair in those who develop symptoms or have expansions greater than 3 cm during surveillance. In-hospital mortality following aneurysmectomy ranges from 2% to 19%, and risk is increased in those with severe LV dysfunction before surgery.[2] Following LV reconstruction with the Dor procedure (an endocardial patch plasty), 5-year transplant-free survival was 82.1%, with preoperative LVEF, LV volumes, and NYHA functional class being independent predictors for survival.[21] Similarly, the Coronary Artery Surgery Study registry found that the increased mortality in patients with an LV aneurysm was related to the degree of LV dysfunction rather than the presence of an LV aneurysm.[22]

Conclusion

Left ventricular pseudoaneurysms are rare complications of MIs that have high morbidity and mortality owing to the risk of rupture from increased wall stress. Venous stasis can predispose to thrombus formation and subsequent systemic embolization. Distinguishing LV pseudoaneurysms from true LV aneurysms using angiography or echocardiography is important for prognostication because of the high risks of late rupture in

pseudoaneurysms. Surgical aneurysmectomy with coronary artery bypass grafting is an ACC and AHA class IIa recommendation and has favorable outcomes with 5-year survival outcomes with improvement in NYHA functional class and LVEF.

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