Geriatric Presentation of Idiopathic Left Ventricular Aneurysm

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INTRODUCTION

Left ventricular aneurysm is a known complication of transmural myocardial infarction. However, less common causes include hypertrophic cardiomyopathy, inflammatory conditions (like myocarditis or sarcoidosis), or a postsurgical complication. Idiopathic left ventricular aneurysms are very uncommonly described in the literature, especially in the geriatric population. The management and treatment options for idiopathic left ventricular aneurysms are not well described due to the rarity of the condition, especially in a geriatric population.

We describe a case of a 74-year-old woman who initially presented with atypical chest pain and exertional palpitations and was subsequently diagnosed with an idiopathic left ventricular aneurysm. Given the lack of symptoms and lack of progression in size, she was managed conservatively with a good outcome.

CASE PRESENTATION

A 74-year-old female presented to our hospital for evaluation of atypical chest pain and palpitations for several months. Her palpitations were exertional in nature. Her past medical history was significant for hypertension and hyperlipidemia. She denied any symptoms of heart failure or syncope. She did not have any history of coronary artery disease, chest trauma, autoimmune conditions, or recent infections.

On physical examination, her vital signs were within normal limits. No cardiac murmurs were appreciated, and her lungs were clear to auscultation. There was no evidence of lower extremity edema. An electrocardiogram revealed normal sinus rhythm with no significant abnormalities.

To further evaluate her palpitations, a transthoracic echocardiogram (TTE) was performed that demonstrated a focal dyskinetic outpouching of the apical anterior wall. The other left ventricular wall segments had normal contractility, and the left ventricular ejection fraction was normal at 60% (Figure 1 and Video 1). Upon administration of a microbubble echo contrast agent, no communication between the myocardial and pericardial space was noted (Figure 2 and Video 2). The remainder of the TTE exam was unremarkable.

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Given her symptoms of exertional palpitations, an exercise echocardiogram was performed. She exercised for six minutes (which was expected for her age and gender) and attained 128% of the age-predicted maximal heart rate. Although she did not experience any palpitations with exercise, her stress electrocardiogram was notable for six beats of ventricular bigeminy at peak exercise. There was no electrocardiographic or echocardiographic evidence of inducible ischemia.

Subsequently, cardiac catheterization with coronary angiography and left ventricular angiography was performed. The coronary arteries were angiographically normal, and the left ventricular angiogram was notable for a discrete aneurysmal outpouching in the apical anterior segment of the left ventricle (Figure 3 and Video 3).

At this point, the differential for this outpouching included a nonischemic aneurysm, a pseudoaneurysm, or a diverticulum. Cardiac magnetic resonance imaging (cMRI) was pursued to help delineate the etiology of this outpouching. The cMRI revealed a focal outpouching of the anterior wall approximately 3 cm in length at the base in systole. The outpouching was characterized as a thin-walled dyskinetic structure spanning the mid and apical segments of the anterior wall (Figure 4 and Video 4). Since the outpouching contained all the layers of the myocardium (excluding pseudoaneurysm) and had >50% late gadolinium enhancement (consistent with scarring), the outpouching was consistent with a true aneurysm of nonischemic etiology (Figure 5). The cMRI was otherwise unremarkable.

A two-week ambulatory cardiac monitor (Ziopatch) performed for palpitations revealed three short runs of nonsustained ventricular tachycardia (the longest run being five beats). She also had a 12.5% burden of isolated ventricular beats. An electrophysiologic study performed to evaluate for any inducible ventricular arrhythmias (and risk stratification) was negative. Risk stratification for ventricular arrhythmias was important since the presence of intractable ventricular arrhythmias unresponsive to conventional therapy is an indication for left ventricular aneursymectomy per the American College of Cardiology/American Heart Association Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction.[1] Since our patient had a negative electrophysiologic study, she was initiated on a low-dose metoprolol for the ventricular ectopy with improvement in palpitations. Given the patient's age and lack of high-risk features on the cardiac imaging, conservative management with close follow-up was pursued. Three years later, a repeat cMRI did not demonstrate any progression in the size of the aneurysm. The patient continues to be managed conservatively and is doing well.

DISCUSSION

The differential diagnosis of outpouchings of the left ventricle includes true aneurysm, pseudoaneurysm, and diverticulum. Differentiating among these entities can often be challenging but is extremely important for prognostication and clinical management.

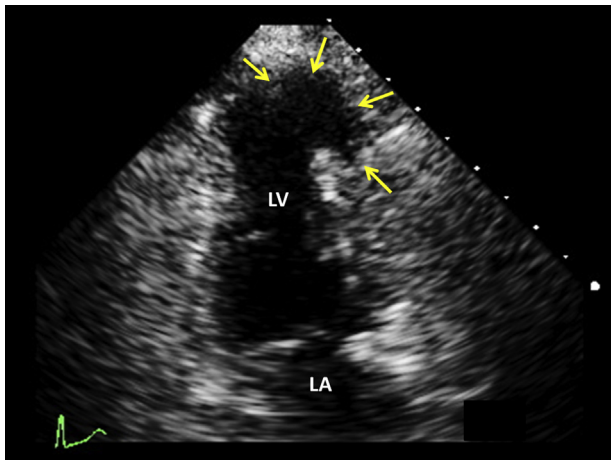

Figure 1 Apical two-chamber view of the left ventricle on TTE demonstrating the left ventricular outpouching (*yellow arrows*). *LA*, left atrium; *LV*, left ventricle.

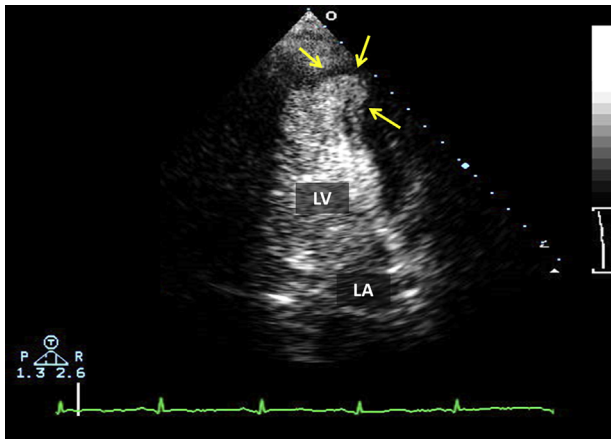

Figure 2 Apical two-chamber view of the left ventricle on TTE with microbubble echo contrast demonstrating the left ventricular outpouching (*yellow arrows*). *LA*, left atrium; *LV*, left ventricle.

A left ventricular aneurysm is defined as a region of ventricular wall, thinner than the adjacent myocardium, which exhibits either akinesis or dyskinesis. It occurs most commonly in the anterior and apical segments.[2] It involves the endocardium, myocardium, and the pericardium, which distinguishes it from a pseudoaneurysm (which contains only the pericardium). The most common cause of a left ventricular aneurysm is transmural myocardial infarction. However, other less common causes of psuedoaneurysm include trauma, iatrogenic (postsurgical), hypertrophic cardiomyopathy, congenital, infective endocarditis, and inflammatory conditions like Chagas disease or sarcoidosis.[2] Left ventricular aneurysms have also been rarely described in idiopathic dilated cardiomyopathy.[3] In our case, all of the above etiologies were ruled out and the cause of left ventricular aneurysm was idiopathic: a rare diagnosis.

Pseudoaneurysm represents focal rupture of the myocardium contained by the pericardium and typically carries a worse prognosis than true aneurysm.[4] Most pseudoaneurysms occur due to transmural myocardial infarctions, but other rare causes include iatrogenic (postsurgical) or trauma.[5] Unlike aneurysms, which have a wide neck,

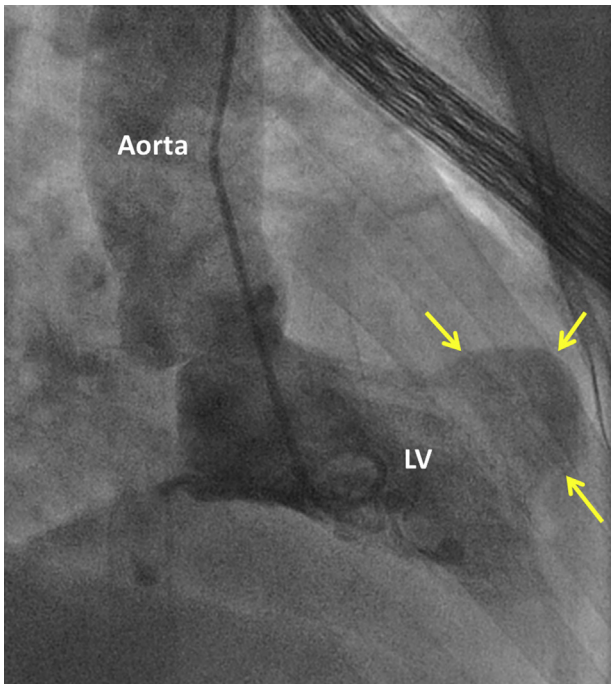

Figure 3 Right anterior oblique view of the left ventricle on cardiac catheterization demonstrating outpouching of the left ventricle on ventriculography (*yellow arrows*). *LV*, left ventricle.

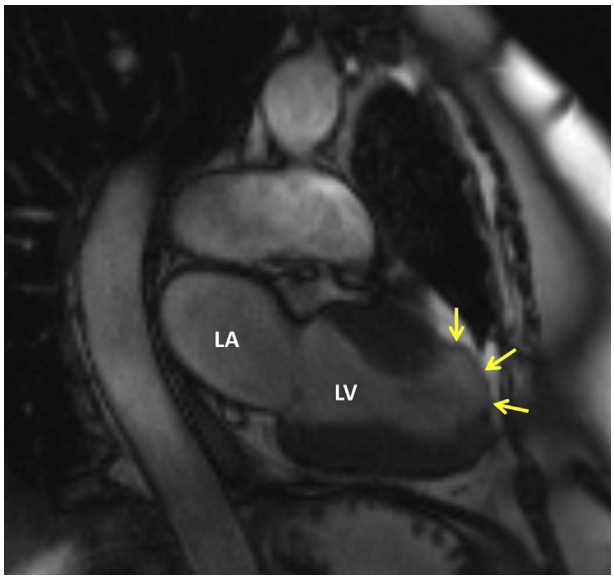

Figure 4 CMRI demonstrating left ventricular outpouching in a two-chamber view in the steady state free precession (*yellow arrows*). *LA*, left atrium; *LV*, left ventricle.

pseudoaneurysms have a narrow neck with a wide sac.[6] They typically require surgical intervention to prevent progression.

Left ventricular diverticulum also involves the endocardium, myocardium, and pericardium.[7] The normal contractility of the walls usually helps to differentiate it from an aneurysm. Left ventricular diverticulum is typically congenital in etiology, and the prevalence based on autopsy studies is estimated to be around 0.4%.[8] Left

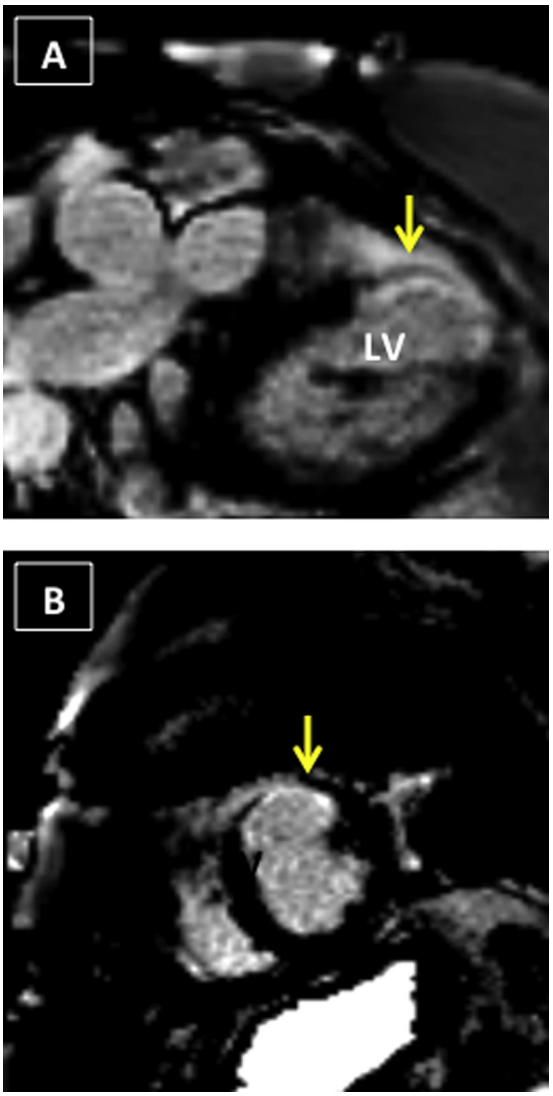

Figure 5 CMRI with delayed gadolinium enhancement demonstrating a left ventricular outpouching (*yellow arrow*) containing a thin fibrotic left ventricular myocardium in a paraxial view **(A)** and short-axis view **(B)**.

ventricular diverticula are often associated with midline thoracoabdominal congenital abnormalities, diaphragmatic/sternal defects, and partial absence of the inferoapical pericardium, a condition known as Cantrell syndrome.[9] They are usually discovered incidentally on cardiac imaging as many patients remain asymptomatic. The definitive prognosis and true complications are not well described due to the rarity of the condition.

Clinical implications of a left ventricular aneurysm include heart failure, angina,[10] and/or ventricular arrhythmias.[11] Other complications include systemic embolization[12] (secondary to stasis of blood pool in the dsykinetic ventricular cavity) and catastrophic ventricular rupture of the thin scarred left ventricular wall.[4] The prognosis of non-ischemic left ventricular aneurysms is not well described in literature. However, based on the existing literature for ischemic left ventricular aneurysms, one may extrapolate that smaller size aneurysms carry a better prognosis.[13]

TTE is the initial modality used to diagnose left ventricular aneurysms; however, transesophageal echocardiography, left ventriculography, cardiac computed tomography, and magnetic resonance imaging have a higher sensitivity and are more helpful in distinguishing aneurysms from pseudoaneursyms.[14–16]

Small-size, asymptomatic aneurysms can perhaps be managed medically.[13] Systemic anticoagulation is indicated if a thrombus is present. The 2013 American College of Cardiology/American Heart Association heart failure guidelines recommend surgical aneurysmectomy be considered (class IIb; level of evidence B) for patients with intractable heart failure and or ventricular arrhythmias.[1]

CONCLUSION

We add to the literature a geriatric presentation of an idiopathic left ventricular aneurysm, a rare diagnosis. Our patient remained clinically stable and asymptomatic, and on serial imaging, no progression in the size of the aneurysm was noted. Therefore, she was treated conservatively, given the lack of any absolute indications for surgery.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.01.008.

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