Multimodality Imaging of an Idiopathic Left Ventricular Aneurysm Presenting With Frequent Premature Ventricular Beats

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INTRODUCTION

A 57-year-old man was evaluated for chest pain and premature ventricular contractions (PVCs) seen on electrocardiogram (ECG). The patient had a history of PVCs with an unremarkable transthoracic echocardiogram (TTE) done 16 years ago. Transthoracic echocardiogram now shows a left ventricular (LV) aneurysm with otherwise preserved LV ejection fraction. Cardiac computed tomography (CCT) did not show any coronary artery disease (CAD). After excluding other causes of LV aneurysm by history, blood tests, and further imaging, a diagnosis of idiopathic LV aneurysm was made. We report this case to highlight the use of multimodality imaging, including TTE, single-photon emission computerized tomography (SPECT) myocardial perfusion imaging (MPI), and cardiovascular magnetic resonance imaging (CMR) in the evaluation of an idiopathic LV aneurysm.

CASE PRESENTATION

A 57-year-old man presented to the emergency department for mild chest and epigastric pain. The patient was an ex-smoker with no chronic disease. There was a history of palpitations evaluated in a separate hospital 16 years ago, for which an ambulatory event monitor was placed that showed frequent PVCs. The TTE reported then showed preserved LV ejection fraction and mitral valve prolapse with mild mitral regurgitation.

At the emergency department the patient's blood pressure was 111/70 mm Hg, pulse 62 beats per minute, and oxygen saturation of 100% on room air. There were normal heart sounds with no murmurs or pericardial rub. The lungs were clear, and no jugular venous distension was noted. The ECG (Figure 1) showed sinus rhythm with frequent PVCs that have a left bundle brunch block morphology and inferior axis. Troponin I was 2 ng/L (reference range, 0-18). The patient was referred to the cardiology outpatient clinic for further evaluation.

In view of the patient's symptoms of chest pain as well as the frequent PVCs on the ECG, a TTE and a vasodilator stress technetium Tc-99m tetrofosmin SPECT MPI were ordered. Despite having a higher diagnostic sensitivity for CAD in this young patient with minimal risk factors, a CCT was not chosen due to concerns of technical challenges from frequent PVCs. The patient received a 2-day imaging protocol due to body weight, which was composed of an initial rest MPI followed by separate day stress MPI. The patient completed the rest MPI uneventfully and had the TTE performed on the same day. The TTE images (Figure 2, Videos 1-3) revealed the presence of an apical outpouching with a relatively wide neck and color flow between the LV cavity and the outpouching. The rest MPI (Figure 3) demonstrated a severe apical perfusion defect with an apical outpouching myocardium seen particularly in the horizonal long-axis views. Notably, radiotracer uptake can be seen within the walls of the outpouching.

When we knew the TTE results, the stress component of the MPI was canceled. The decision was made to attempt CCT to have greater diagnostic certainty for CAD as well as to visualize the outpouching better. The CCT revealed a coronary artery calcium score of 0 and nonobstructive CAD. The neck of the aneurysm was noted to be relatively wide, and a smooth transition from the surrounding normal myocardium to the thinned outpouching wall was observed. Volume-rendered reconstructions showed a complex aneurysm comprising 2 separate and interconnected outpouchings at the apex and anterolateral wall (Figures 4 and 5). The patient was started on low-dose bisoprolol 1.25 mg daily.

Cardiovascular magnetic resonance imaging (Figure 6, Video 4) was performed to further characterize the aneurysm. No LV thrombus was seen on early gadolinium enhancement images. The complex aneurysm was again visualized with a thin layer of subendocardial late gadolinium enhancement (LGE) seen lining the aneurysm. There was no CMR evidence of arrhythmogenic right ventricular cardiomyopathy.

Given the absence of obstructive CAD on CCT, as well as the lack of transmural infarct pattern on CMR LGE images, other causes of LV aneurysm were considered including Chagas disease, human immunodeficiency virus, tuberculosis, sarcoidosis, and systemic lupus erythematosus.[1] The patient denied having lived in or traveled to Latin America, thus making Chagas disease less likely. There were no systemic features of autoimmune disease, and human immunodeficiency virus screen was also negative. Although multiple congenital LV aneurysms have been reported, we were unable to draw a definite conclusion as the TTE 16 years ago was unavailable for us to review. A diagnosis of idiopathic LV aneurysm was eventually made. After a multidisciplinary heart team discussion, a consensus for nonsurgical management was reached. In view of the size of the aneurysm and possible thromboembolic risk, the patient was eventually started on warfarin. A 24-hour Holter monitor was also used while the patient was taking bisoprolol, which demonstrated a PVC burden of only 1% with no corresponding symptoms. Interestingly, the morphology of the asymptomatic PVCs did not correlate to the location of the aneurysms and was hence treated conservatively with only beta blockade. The patient remained well with no complications at 1 year. This patient will be followed annually with clinical visits and

TTE. Advanced imaging with CMR or CCT will be recommended for changes found on TTE.

DISCUSSION

This case describes the use of multimodality cardiac imaging in the evaluation of an LV outpouching incidentally found during investigation of atypical chest pain and PVCs. The main causes of the LV outpouching include aneurysm and pseudoaneurysm.[1] An aneurysm is an outpouching with all 3 layers, namely, endocardium, epicardium, and thinned scarred myocardium, that line its sac.[1] In contrast, as a pseudoaneurysm represents contained cardiac rupture with overlying adherent pericardium and no myocardial tissue, there is a higher risk of rupture compared to aneurysm.[1]

Given the above, there is a need to differentiate these 2 entities noninvasively to further guide management. The morphological features separating aneurysm and pseudoaneurysm are well described in the literature, of which many can be demonstrated on TTE. This includes using the ratio of the maximal internal neck width to the maximal parallel internal diameter of the aneurysm[1] based on the observation that aneurysms tend to have a wider orifice relative to the aneurysm cavity diameter. In a small case series, Scagliola *et al.*[1] found that this ratio was 0.5 in pseudoaneurysms compared with between 0.9 and 1.0 in true aneurysms. On color Doppler, laminar blood flow through the wide aneurysm neck can be observed, in contrast to more turbulent flow through a narrower pseudoaneurysm neck.[1] In addition, a sharp discontinuity of the endocardial border at the base of the outpouching points toward a pseudoaneurysm, whereas aneurysms exhibit a smoother transition from normal myocardium to thinned aneurysmal wall.[1] Finally, the aneurysm sac is usually dyskinetic with paradoxical bulging expansion during systole, while pseudoaneurysms tend to be akinetic.[1,2] This feature is also important to exclude a diverticulum, a less common differential for LV outpouching that displays synchronous contractility and is usually congenital and associated with midline thoracoabdominal defects and other heart malformations.[1,2]

Importantly, TTE is likely to be the first investigation to reveal the cardiac outpouching as it is readily available and considered the first-line diagnostic and risk stratification tool for CAD, valvulopathies, cardiomyopathies, and arrhythmias, as shown in our patient, who initially received a TTE for the evaluation of frequent ventricular ectopy and reported mitral valve prolapse. However, limitations of TTE include operator dependency and limited image quality with endocardial dropout.[2] The use of ultrasound-enhancing agents in
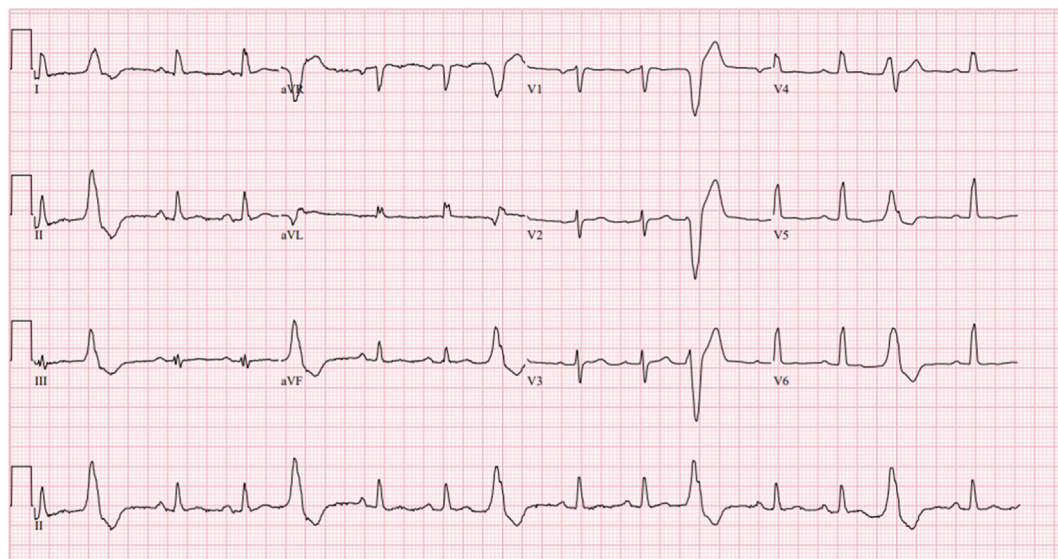

Figure 1 A 12-lead ECG demonstrates normal sinus rhythm with frequent PVCs that have an inferior axis and a left bundle branch block morphology.
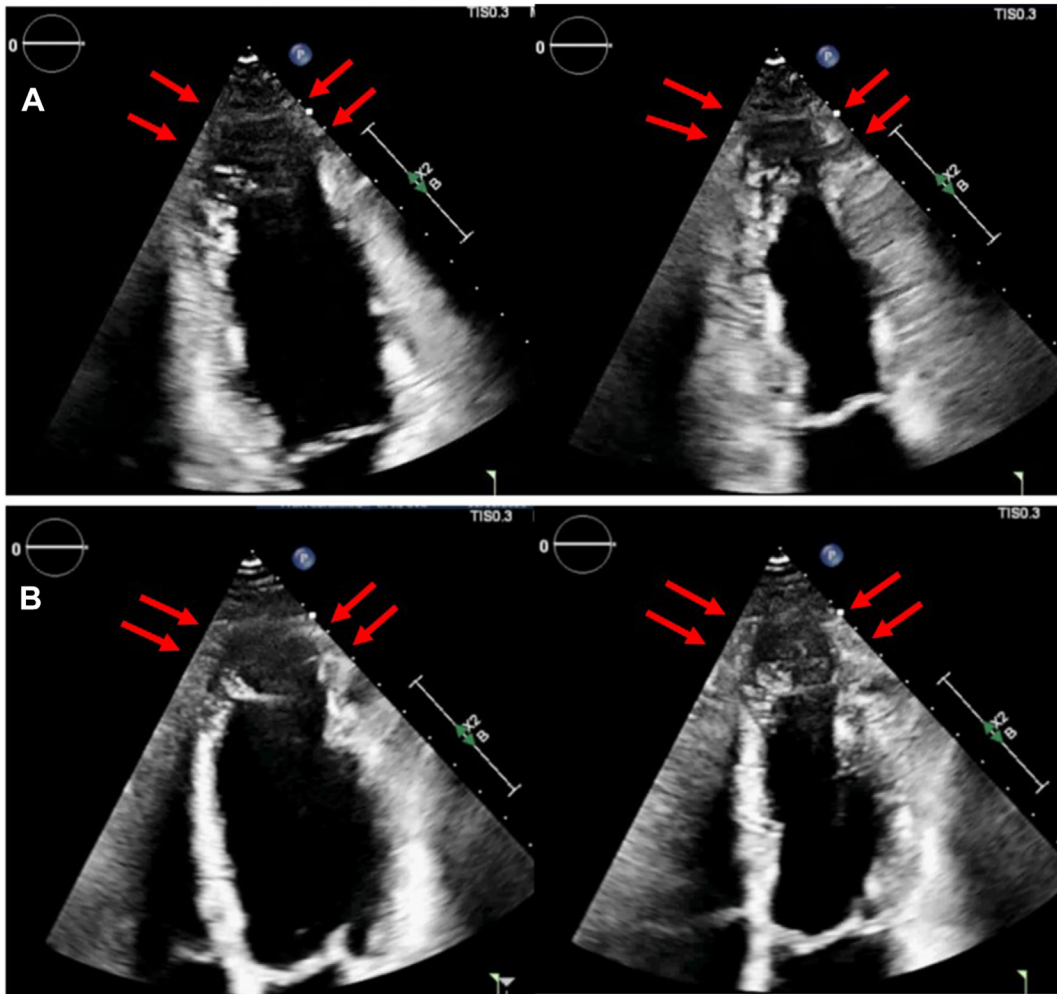
Figure 2 Two-dimensional TTE, apical 2-chamber **(A)** and apical 2-chamber **(B)** views in diastole (*left*) and systole (*right*), demonstrates the normal global LV systolic function with a focal LV apical dyskinetic outpouching segment with a relatively wide neck (*red arrows*).

TTE can aid in better image quality to visualize the neck of the aneurysm and communication with the left ventricle (LV).[3] Cardiac computed tomography has superior spatial resolution[2] and demonstrates the morphology of the outpouching neck and sac described earlier. As illustrated in our case, use of additional multiplanar and volume-rendered CCT reconstructions allows for excellent three-dimensional visualization of the LV aneurysm and appreciation of its complexity, which would have been useful if surgical intervention was contemplated. As both aneurysms and pseudoaneurysms are possible complications of CAD and myocardial infarction, CCT is useful to exclude significant CAD with high sensitivity. In addition, identification of coronary arteries overlying the wall of the outpouching excludes a pseudoaneurysm and makes aneurysm more likely.[4]

Nuclear cardiology techniques are less commonly used as the primary imaging tool to differentiate aneurysm from pseudoaneurysm due to the inherent low spatial resolution and availability of alternative modalities.[1] Prior reports typically describe the use of radionuclide ventriculography that can not only demonstrate the morphological features of the LV outpouching but also identify pseudoaneurysm, which exhibits a characteristic delayed filling of the sac after the LV.[5] Reports of LV aneurysm seen on SPECT MPI are rare, and we

highlight this case report because of the interesting findings seen on SPECT MPI. We were only able to find one other reported case demonstrating an idiopathic LV aneurysm on SPECT MPI,[6] which was initially mistaken as a pseudoaneurysm due to the presence of a large area of absent tracer uptake in the anterior and anterolateral wall seen on SPECT MPI but proven otherwise during surgery. The authors conclude that it is challenging to diagnose aneurysms based on radionuclide imaging as the typical finding is absent perfusion interpreted as infarct.[6] Unlike the previously reported case, MPI in our case demonstrated radiotracer uptake in the aneurysmal area. As the uptake and retention of technetium-based radionuclides require the presence of intact mitochondrial and sarcolemma membranes, this suggests the presence of intact and alive cardiomyocytes within the aneurysm.

Cardiovascular magnetic resonance imaging is also a useful imaging modality to visualize the outpouching and associated morphologic features such as the neck, sac, and transition from normal to thinned myocardium.[2] Wall motion abnormalities such as akinesia or dyskinesia can be appreciated on cine CMR.[2] The presence of epicardial fat next to the outpouching on CMR also excludes the diagnosis of pseudoaneurysm.[7] The main strength of CMR is in tissue
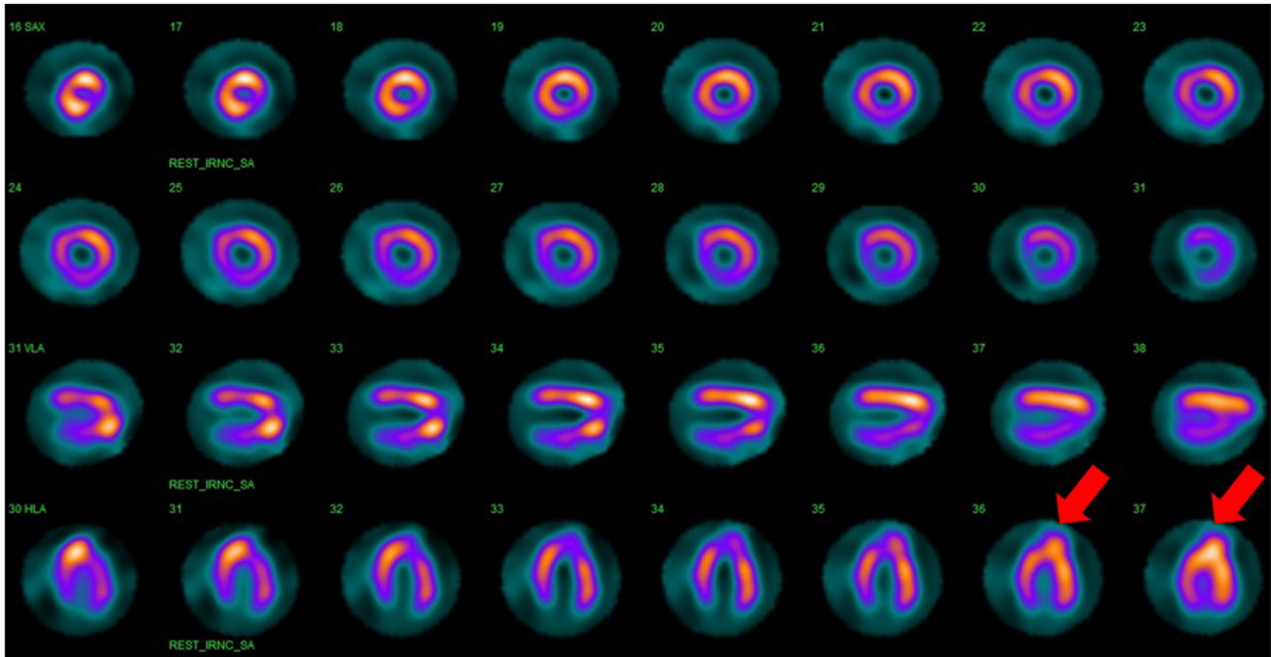
Figure 3 Rest SPECT MPI with short-axis (SAX; *top two rows*), vertical long-axis (VLS; *third row*; apical 2-chamber equivalent), and horizontal long-axis (HLA; *bottom row*; apical 4-chamber equivalent) displays demonstrates an apical perfusion abnormality with an outpouching seen particularly in the HLA views with radiotracer uptake seen within the walls of outpouching myocardium (*red arrows*).
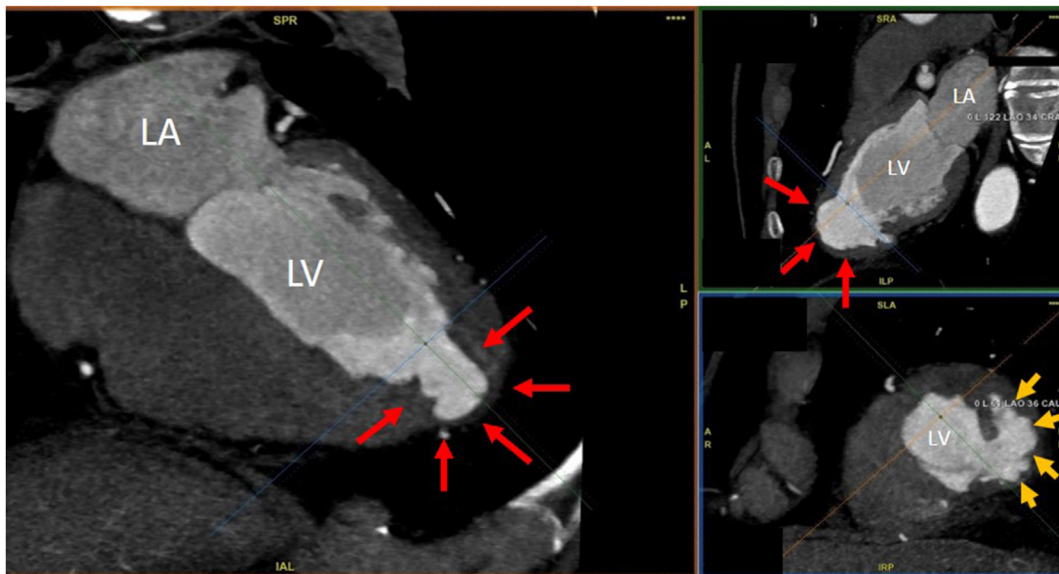

Figure 4 Cardiac computed tomography multiplanar reconstruction images in representative apical 4-chamber (*left*), apical 2-chamber (*top right*), and apical short-axis (*bottom right*) views depicting the aneurysmal outpouching at the apex (*red arrows*) and antero-lateral (*yellow arrows*) myocardial wall segments with a relatively wide neck and smooth transition from the adjacent normal myocardium to the thinned aneurysmal wall. *LA*, Left atrium; *LV*, left ventricle.

characterization, such as using early gadolinium enhancement images to exclude thrombus within the aneurysm. Late gadolinium enhancement is useful to demonstrate the underlying cause such as a transmural infarct or infiltrative disease. Importantly, aneurysms typically exhibit LGE in the wall representative of scarred and fibrotic myocardium.[2] Conversely, LGE of the overlying pericardium is more suggestive of pseudoaneurysm and is hypothesized to be due to chemical irritation, inflammation, and neovascularization of the pericardium in the acute phase of myocardial rupture.[2] Although these typical LGE patterns are often reported in the context of CAD, we note that the thin layer of subendocardial LGE lining the aneurysm in our patient appears similar to that described in another reported
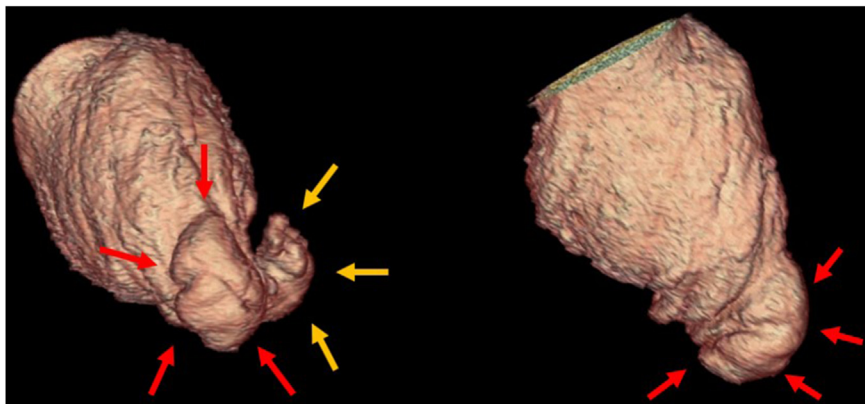
Figure 5 Three-dimensional CCT volume-rendered reconstructions demonstrate the complex LV aneurysm comprising 2 separate but interconnected outpouchings at the cardiac apex (*red arrows*) and distal anterolateral myocardial wall segments (*yellow arrows*).
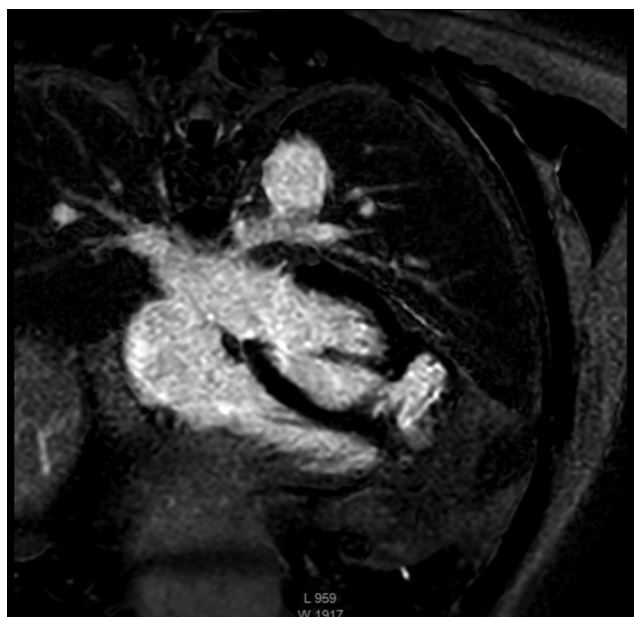

Figure 6 Cardiovascular magnetic resonance imaging, LGE sequence 4-chamber display, demonstrates the complex LV apical aneurysm comprising 2 interconnected, focal, thin-walled outpouching myocardial segments with a thin subendocardial layer of LGE lining the aneurysm walls.

case of idiopathic LV aneurysm.[8] In both cases, CMR demonstrated LV apical dyskinetic outpouching with a thin layer of LGE presumed to be fibrotic myocardium.[1] Given that we were able to demonstrate radionuclide tracer uptake in the same region, we could not completely exclude the possibility of blood pool artifact giving rise to the appearance of "LGE" lining the aneurysm.

Despite the above, differentiation of aneurysm and pseudoaneurysm based on noninvasive imaging is seldom straightforward, with many reported cases of initial imaging diagnosis eventually proven wrong during surgery.[9] Novel modalities to evaluate LV outpouchings have been reported, such as using late enhancement dual-energy computed tomography iodine mapping and virtual monoenergetic imaging reconstructions to demonstrate transmural iodine accumulation in the wall of aneurysms[10] as well as three-dimensional printing generated from CCT images to guide surgery.[11]

In terms of etiology, we excluded CAD based on the normal coronaries on CCT. Myocardial infarction with nonobstructive coronary arteries is also unlikely as there was neither a symptomatic clinical event suggestive of acute myocardial ischemia nor new ischemic ECG changes,[12] and CMR did not show typical infarct pattern LGE. Similarly, the various imaging tests, particularly CMR, were useful to exclude other possible etiologies of aneurysm such as hypertrophic cardiomyopathy, myocarditis, and sarcoidosis. Cardiovascular magnetic resonance imaging findings were not suggestive of arrhythmogenic right ventricular cardiomyopathy.[1] A congenital LV aneurysm is less likely given that it was not reported on a TTE performed in a separate hospital 16 years ago, although we could not exclude it completely as the earlier images were not accessible to us. Multiple LV aneurysms are a rare phenomenon with scarce literature. These few case reports have been mainly described in the African population and are idiopathic in nature, although some other patients had heart failure and cardiomegaly.[13]

In the context of CAD, surgery is often recommended for pseudoaneurysms due to the higher risk of rupture. For aneurysms, however, the risk of rupture is lower and surgery is reserved for cases of heart failure, arrhythmias, or thromboembolism.[1] However, the management of other causes of aneurysm, particularly idiopathic LV aneurysm, is less well described. A case series of LV aneurysm complicating myocarditis showed very good prognosis with conservative management alone, with no cases requiring surgery or implantable device at 53 months' follow-up.[14] We adopted a heart team approach for our patient and decided on initial conservative management in view of the response to medical treatment and lack of symptoms, as well as the patient's preference. Extrapolating from ST-segment elevation myocardial infarction guidelines, anticoagulation, most commonly vitamin K antagonist, is often recommended for LV aneurysm to mitigate the risk of thromboembolism despite the paucity of evidence for its use.[15] The use of direct oral anticoagulants for this purpose has been controversial, with some studies showing promise[15] and others, including a large multicenter cohort study, showing harm and possible increased risk of systemic embolism with direct oral anticoagulants over vitamin K antagonist use.[15]

CONCLUSION

In summary, we report a rare case of a complex idiopathic LV aneurysm that was evaluated using several noninvasive imaging modalities that were complementary to one another. The treatment of this uncommon condition is not well established and should be tailored to each individual patient's presentation.[12]

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with the following guidelines: Our institution does not require ethical approval for reporting individual cases or case series.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

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DISCLOSURE STATEMENT

The authors report no conflict of interest.

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2023.05.007.

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