Case Report

Idiopathic ‘true’ left ventricular aneurysm

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A B S T R A C T

We report the case of a 67-year-old patient who presented with anginal symptoms to the hospital. Computed tomographic angiography, to rule out a pulmonary embolism, showed a left ventricular apical outpouching. The patient underwent further imaging modalities, including contrast echocardiography (TTE) and cardiovascular magnetic resonance imaging (CMR), which were suggestive of a true left ventricular aneurysm (LVA). The absence of obstructive coronary artery disease on coronary angiography, absence of late enhancement on the CMR, and ultimately the intraoperative findings during surgical resection of the aneurysm, were strong indicators of a non-ischemic etiology of the patient’s LVA. Additionally, the patient denied any previous history of cardiac instrumentation to rule out iatrogenic causes of LVA and congenital causes were excluded by a previous echocardiogram. Finally, history and presenting electrocardiogram did not reveal any other underlying obvious causes for the LVA. Excluding all common causes for the LVA an idiopathic cause seemed most likely.

<Learning objective: Patients with an idiopathic left ventricular aneurysm are at risk for life-threatening ventricular arrhythmias and sudden death, which may sometimes occur as the first clinical presentation. Echocardiography, cardiovascular magnetic resonance imaging, and cardiac contrast angiography can reliably detect the location, extent, and morphology of the aneurysm. Management strategies should be individualized and are mainly directed toward prevention of sudden death and recurrent arrhythmias.>

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Introduction

A left ventricular aneurysm (LVA) is an infrequent (5–10%) and devastating complication of myocardial infarction that can result in severe congestive heart failure and cardiogenic shock [1]. Other reported potential etiologies of LVAs are trauma, hypertrophic cardiomyopathies, congenital LVAs, myocarditis, arrhythmogenic right ventricular dysplasia/cardiomyopathy with involvement of the left ventricle, glycogen storage disease, and more exotic causes such as Chagas’ disease [1]. In the majority of cases, the electrocardiogram (ECG) and transthoracic echocardiogram (TTE) help to identify the underlying etiology. However, in rare instances, an exclusion diagnosis of true idiopathic LVA can be made.

Case report

We report a case of a 67-year-old woman with paroxysmal atrial fibrillation and coronary artery disease risk factors of age and dyslipidemia, who initially presented to a community hospital with left-sided chest discomfort and shortness of breath that she attributed to indigestion. She had undergone an unremarkable TTE years before, and she denied any previous cardiac catheterizations or instrumentation. On initial presentation, troponin T level was <0.012 ng/ml and ECG showed non-specific T wave changes. As a part of her initial evaluation to rule out pulmonary embolism, computed tomographic angiography incidentally showed a focal 1.5 cm outpouching of the left ventricular apex. TTE confirmed the presence of a left ventricular apical outpouching without additional wall motion abnormalities and preserved left ventricular ejection fraction without evidence of asymmetric septal hypertrophy (Fig. 1A and supplemental video 1). Subsequent cardiac catheterization revealed non-obstructive coronary artery disease and normal left ventricular end-diastolic pressure, but
confirmed the presence of a left ventricular apical outpouching (Fig. 1B and supplemental video 2).

Supplementary Videos 1 and 2 related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jccase.2015.08.013.

The patient was transferred to Johns Hopkins Hospital and she underwent cardiovascular magnetic resonance imaging (CMR), which was notable for a focal outpouching of the left ventricular apex measuring 1.2 cm in depth with a neck measuring 0.3 cm in systole and 1.1 cm in diastole without visualized scar tissue on delayed post-gadolinium contrast imaging (Fig. 1C and supplemental video 3).

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The patient was diagnosed with an LVA and ultimately underwent surgical repair. The LVA was approached through a standard median sternotomy and using cardiopulmonary bypass. Intra-operative examination of the heart confirmed the imaging findings of a true LVA with 1 cm narrowing at the neck without significant surrounding scar tissue or features of prior myocardial infarction (Fig. 1D and supplemental video 4). The LVA was then repaired using a linear repair technique. The aneurysm was incised and opened. The base of the aneurysm was then closed with felt strips on either side of the defect and multiple interrupted 2-0 polypropylene mattress sutures. The closure was then reinforced with a continuous 4-0 polypropylene suture.

Supplementary Video 4 related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jccase.2015.08.013.

Discussion

Due to the small size of the aneurysm, no histopathology was submitted. However, based on the multiple modes of imaging that were obtained via echocardiography and CMR, and given the intraoperative findings, we are confident that the visualized outpouching was a true aneurysm. In addition, our team carefully reviewed the previous TTE and confirmed the absence of an LVA prior to this hospital admission.

The absence of obstructive coronary artery disease, absence of late enhancement on the CMR, and the intraoperative findings are strong indicators of a non-ischemic etiology of our patient's LVA. Additionally, the patient denied any previous history of cardiac instrumentation to rule out iatrogenic causes of LVA and congenital causes were excluded by a previous echocardiogram. However, we are aware that small aneurysms are sometimes missed on such studies and since there was no other mode of imaging in the past we cannot entirely exclude the possibility of a small congenital aneurysm. Finally, history and presenting electrocardiogram did not reveal any other underlying obvious causes for the LVA. Excluding all common causes for the LVA an idiopathic cause appears most likely.

While previous studies have documented the importance of post-infarct LVAs, and current recommendations largely follow these data, there are few data on the natural history of rare idiopathic LVAs of unclear etiology. A previously published case series by Paul et al. has reported that life-threatening arrhythmias are often the first manifestation of an idiopathic LVA and further emphasizes the importance of modern imaging techniques [2]. Thus, given the
relatively thin nature of the aneurysm and overall uncertain
gnosis, we relied upon anecdotal reports of morbid outcomes
including development of ventricular arrhythmias, rupture, and
hemopericardium with tamponade to guide therapeutic decision-
making [2,3]. Notably, previously studied cases of non-infarct
related LVAs suggest an even higher incidence of complications than
in patients with infarct-related true aneurysms [4–6].

Unfortunately, long-term survival from the presence of true
LVAs is still unclear, with previous reports ranging from 12% [7] to
71% survival [8]. However, the reported long-term survival in
otherwise low-risk patients who undergo surgery has been
reported to be as high as 77% [9]. Thus, in our patient with no
significant coronary artery disease, no known history of lethal
arrhythmias, and no other significant comorbidities that would
pose a perioperative risk, she was deemed to be a low-risk
candidate for surgery. After discussion of both medical and surgical
options, the patient ultimately wished to undergo surgical
prophylaxis in light of the possible complications. Alternative
resection options for LVAs, including percutaneous closure
approaches, have been described in the literature [10] and were
offered to our patient.

Conflicts of interest

None declared.

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