That’s No Moon, It’s a Giant Left Ventricular Aneurysm: A Case Report and Literature Review

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ABSTRACT

Background: Although the most common causes of left ventricular aneurysm (LVA) is ischemic disease, other infectious, traumatic, genetic and iatrogenic etiologies exist. With the improvement of medical therapy for ischemic disease and earlier interventions such as PCI, the incidence of large LVA (>3cm) and surgical treatment for it is increasingly rare.

Case study: We describe a case report and literature review of a giant LVA in a patient, who presented with unclear etiology. A 61-year-old male was referred to our tertiary center. He underwent aneurysmectomy and mitral valve replacement for a giant (10cm x 10cm) LVA with severe mitral regurgitation.

Conclusion: Surgery for LVA is becoming less common. Early intervention can restore cardiac geometry with good short and long-term surgical outcomes, especially in patients with preserved EF. Ultimately, a giant ventricular aneurysm remains an indication for surgical intervention. Patients with markedly reduced EF may derive reduced benefits from aneurysmectomy.

INTRODUCTION

Left ventricular aneurysm (LVA) is most commonly a sequela of left coronary myocardial infarction and ischemic heart disease, however, the differential includes traumatic, infectious, and genetic causes [Sattar 2021]. With the improvement of medical therapy for ischemic disease and earlier interventions such as PCI, LVA is becoming increasingly rare [Khanna 2020; Olearchyk 1984]. Especially because the majority of small and moderately sized LVAs are clinically silent, surgical treatment for LVAs is uncommon [Schaitz 2014]. We describe a case report and literature review of giant LVAs in a patient, who presented with unclear etiology.

CASE STUDY

The patient is a 61-year-old veteran, who presented to his local VA hospital with 4 days of fatigue and shortness of breath. He reported increased fatigue and malaise for the prior month which improved with rest. He denied any history of chest pain, dyspnea, orthopnea, or history of myocardial infarction. At the VA hospital, he underwent a CT scan of his chest that revealed a 10cm x 10cm left inferior-lateral ventricular aneurysm and severe mitral regurgitation for which he was referred to our tertiary center. On further investigation, his prior medical history included a gunshot wound to the left chest with retained shrapnel, hypertension, diabetes, stroke, and B-cell lymphoma treated with radiation. He denied any history of myocardial infarction, however, he did report increased fatigue for about 1 month. He denied any prior chest surgery. His outpatient medications included metformin, lisinopril, and amlodipine. He reported being an active smoker. He was previously deployed to the Mediterranean during his military service.

On admission, he was breathing comfortably in room air. His B-naturetic peptide level was slightly elevated at 485,
and troponin was negative. The patient underwent coronary angiography, which identified right dominant anatomy and a 70% lesion in the posterior lateral branch. Electrocardiogram demonstrated an inferior infarct of unknown age. Preoperative echocardiography showed an ejection fraction of 20% and redemonstrated the left ventricular aneurysm with an additional finding of severe mitral regurgitation for which aneurysmectomy and mitral valve repair was recommended. (Figure 1)

Following workup, the patient underwent the planned procedure. Dense pericardial adhesions were seen on sternotomy. The left ventricular aneurysm was isolated, and a thick clot was visualized and removed. The myocardium was debrided, and the aneurysm resected. A collagen patch was patched to the ventricular defect. (Figure 2) The mitral valve was replaced with a 29mm bioprosthetic valve.

**DISCUSSION**

The incidence of LVA due to etiologies such as trauma or infection is not well documented. However, historically, the literature described that 10-30% of individuals who experienced a myocardial infarction went on to develop a LVA or pseudoaneurysm that formed due to thinning of the myocardium following ischemia. While the development of LVA commonly occurs in the weeks to months following infarction, LVA has been observed years after an ischemic event [Della Rocca 2013]. Many individuals who develop LVA are clinically asymptomatic, and moderately symptomatic patients can be medically managed with ACE inhibitors to prevent myocardial remodeling [Louagie 1987].

Based on the most likely causes of LVA, the patient in our case may have experienced a silent myocardial infarction, which would be consistent with his history of diabetes and prior stroke. We suspect the patient in our case had a silent myocardial infarction, which ultimately resulted in a ventricular aneurysm. He may have had an inferior infarct resulting from his right dominant anatomy and a 70% lesion in his posterior lateral branch the prior month. However, we do note that the rest of his coronary angiography did not detect any hemodynamically significant stenoses and, for that reason, revascularization was not performed. Our patient also sustained a gunshot wound to his chest with retained shrapnel during his military service and was treated for B-cell lymphoma with radiation, both of which are reasonable etiologies for his LVA.

For patients who are severely symptomatic with signs of heart failure, embolic events due to mural thrombus, or giant LVAs such as in our patient, surgical evaluation is indicated [ACC/AHA Guidelines 2004]. The goal of reconstruction in left ventricular aneurysms is to restore normal cardiac geometry, and possibly restoring the overall efficiency of the remaining muscle by plication or patching the weakened ventricular wall [Dor 1989]. In order to restore the normal efficiency of the myocardium, aneurysmectomy often is performed in combination with coronary artery bypass grafting, aortic valve replacement, or mitral valve repair/replacement [Olearchyk 1984; Cooperman 1975]. Historical investigations have observed a functional improvement following aneurysmectomy [Castelvecchio 2010]. However, findings from the randomized controlled STITCH trial suggested that ventriculoplasty did not have an overall clinical benefit to patients with reduced EF < 35%, despite restoring the anatomy of the ventricle [Jones 2009]. Ultimately, while reversal of the pathology is important in the treatment of clinically significant LVA, it is important to predict the actual benefit to cardiac function in the preoperative phase.
CONCLUSION

We present an interesting case report and presentation of a giant inferiolateral left ventricular aneurysm. This case is of interest to the community, due to the rare location and size of this aneurysm. Furthermore, this pathology is uncommonly encountered by young clinicians, due to more optimal medical management and early percutaneous interventions in contemporary practice. Although aneurysmectomy is an option for symptomatic left ventricular aneurysms with the goal of restoring normal cardiac geometry, surgeons should take care to optimally select patients who are likely to have restored cardiac function following the intervention.

REFERENCES


