

CASE REPORT**Thrombus Formation Within a Left Ventricular Apical Aneurysm in Hypertrophic Cardiomyopathy with Midcavity Obstruction: A Case Report**Ashwin Jagadish,¹ Shobha Hiremagalur, MD,² Ahmed Khan, MD²¹East Tennessee State University James H. Quillen College of Medicine, Johnson City, TN, USA²Ballad Health CVA Heart Institute, Johnson City, TN USA

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ABSTRACT

Midcavity left ventricular (LV) hypertrophy is a less common form of hypertrophic cardiomyopathy (HCM). It is associated with apical aneurysms, arrhythmias, and sudden cardiac death. Apical aneurysms can lead to the development of thrombi, and anticoagulation may be needed for individuals with this condition. Our case involves a 76-year-old female with HCM who had midcavity LV hypertrophy with obstruction and LV apical aneurysm formation. She developed a thrombus within the aneurysm, which was successfully treated with oral apixaban.

INTRODUCTION

Although hypertrophic cardiomyopathy can manifest with thickening anywhere in the left ventricular (LV) wall, the most common presentations involve hypertrophy of the basal anterior septum or anterior free wall [1]. A rare type of hypertrophic cardiomyopathy (HCM) can present as midcavity LV obstruction [2]. Midcavity LV obstruction

can be associated with LV apical aneurysm formation and subsequent thrombus development within the aneurysm [3]. Transthoracic echocardiograms accompanied by usage of ultrasound-enhancing agents can potentially assist in identification of thrombus formation within the aneurysm; this can help guide management with anticoagulation [1, 4]. We present the case of a 76-year-old female who developed a thrombus within the LV apical aneurysm associated with obstructive midcavity LV hypertrophy.

CASE PRESENTATION

A 76-year-old female with a medical history of known midcavity LV hypertrophy with obstruction and LV apical aneurysm, coronary artery disease, and paroxysmal atrial fibrillation presented to the outpatient cardiology clinic for a routine evaluation. Her procedural history was notable for percutaneous interventions involving the left anterior descending (LAD) artery and implantation of a defibrillator. The patient had declined surgical intervention for her

midcavity LV hypertrophy and apical aneurysm. Her most recent cardiac catheterization demonstrated patent stents within the LAD artery. Her most recent transthoracic echocardiogram with and without contrast, completed approximately 3.5 months prior to the clinic visit, did not reveal thrombus formation within the LV aneurysm.

Previously, her chronic anticoagulation regimen included warfarin for prophylaxis against thromboembolism. However, she discontinued the medication eight weeks prior to the clinic appointment due to gastrointestinal (GI) bleeding presenting as melena, anemia, and supratherapeutic prothrombin time-international normalized ratio (PT-INR). At the time of the GI bleed, her hemoglobin level was 9.7 g/dL (normal range: 13.9 – 16.8 g/dL), hematocrit level was 28.7% (normal

range: 41.0 – 51.0%), and PT-INR was 9.5 (therapeutic range: 2.0 – 3.0). Her baseline hemoglobin level was 13.1 g/dL prior to the GI bleed.

Following the outpatient visit, a transthoracic echocardiogram was obtained and demonstrated thrombus formation within the LV apical aneurysm (Figure 1). The presence of a thrombus was confirmed with an echocardiogram using the contrast agent Lumason, sulfur hexafluoride lipid Type A microspheres (Figure 2). The aneurysm size measured 3.8 x 4.05 cm. At the time of this echocardiogram, her hemoglobin had improved to 14.7 g/dL and her hematocrit had improved to 44.5%. The patient was started on 5 mg of oral apixaban to be taken every 12 hours. Three months later, a repeat echocardiogram with contrast indicated complete resolution of the thrombus.

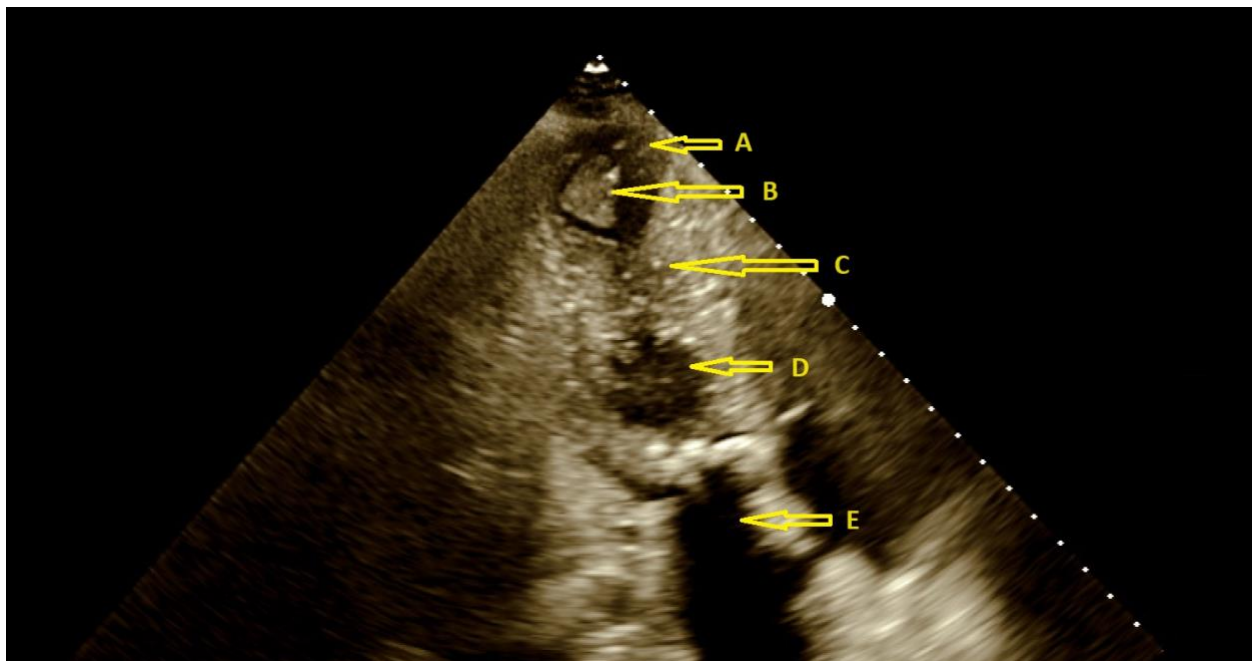


Figure 1. Transthoracic Echocardiogram Without Contrast (A) Left Ventricular Apical Aneurysm. (B) Thrombus. (C) Midcavity Left Ventricular Obstruction. (D) Left Ventricle. (E) Left Atrium.

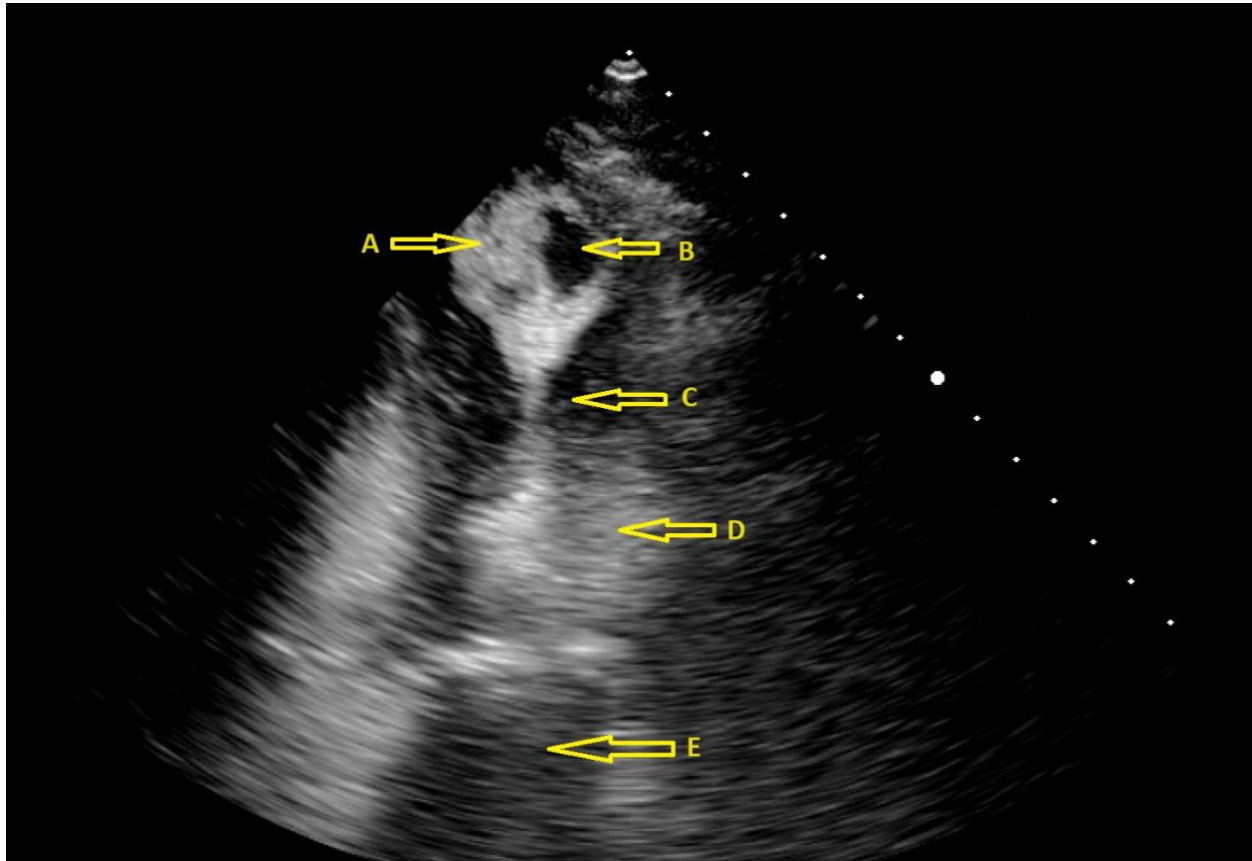


Figure 2. Transthoracic Echocardiogram With Contrast (A) Left Ventricular Apical Aneurysm. (B) Thrombus. (C) Midcavity Left Ventricular Obstruction. (D) Left Ventricle. (E) Left Atrium.

DISCUSSION

Hypertrophic cardiomyopathy has a prevalence of 1:200-1:500 [5], and echocardiography is the preferred imaging modality for monitoring patients with this condition [1]. Midcavity LV hypertrophy with obstruction is a less common form of HCM and has been reported in approximately 10% of cases [6].

LV apical aneurysms are discrete, thin-walled parts of the distal LV near the apex and can be either dyskinetic or akinetic [1]. They have been reported in approximately 5% of individuals with HCM [3]. The presence of LV apical aneurysm can increase the risk of heart failure, sudden

cardiac death, thromboembolic events, and arrhythmia [4, 7, 8]. Studies have revealed different likelihoods for thrombus formation within the aneurysm; one study indicated a risk of 6.5% [3], while another study indicated a risk of 14% [9]. The annualized rate of having a thromboembolic stroke is approximately 2.03% per year [9]. The size of LV apical aneurysms is determined using echocardiography; small aneurysms are less than 2 cm, medium aneurysms are between 2 and 4 cm, and large aneurysms are greater than 4 cm [4, 9]. Thrombus formation has been noted in aneurysms of different sizes [4]. Apical aneurysms and thrombus formation may not always be detected by echocardiography; in these cases, contrast

echocardiography and cardiac magnetic resonance imaging may be alternative imaging modalities [1, 4, 10].

Approximately 20% of thromboembolic events and apical thrombus formation were noted in small aneurysms [4]. Individuals with aneurysms greater than 2 cm can have a four-fold increase in the risk of LV thrombus formation [9]. Prophylactic anticoagulation has been proposed for individuals with aneurysms ≥ 2 cm [9]. It has also been suggested that anticoagulation should be continued even if the thrombus within an aneurysm ≥ 2 cm resolves or if the size of the aneurysm increases [11]. Either direct anticoagulation or vitamin K antagonists can be used [11]. Additionally, surgical methods for relieving obstruction, such as aneurysmectomy with LV reconstruction, can be considered [1, 12].

CONCLUSION

Midcavity left ventricular hypertrophic cardiomyopathy is a less common form of HCM and can be associated with LV apical aneurysms and thrombus formation. Echocardiography is an important imaging modality used for monitoring individuals with this condition, although other modalities can also be pursued. Aneurysms of all sizes, even ones that are small, may carry a risk of thrombus formation and subsequent embolization. While guidelines for prophylactic anticoagulation in individuals with LV apical aneurysms and midcavity LV obstruction need to be formally established, it appears to be especially important to consider prophylactic anticoagulation in those with aneurysms that are 2 cm or larger. Additionally, the ideal frequency for monitoring thrombus formation through imaging in patients not receiving anticoagulation needs further research. Medication options include either direct anticoagulation or antagonists of vitamin K.

Notes

Conflicts of Interest: None declared

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