LEFT VENTRICULAR APICAL ANEURYSM IN ABSENCE OF CORONARY ARTERY DISEASE: A CASE REPORT

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Introduction

- Left Ventricular apical aneurysm (LVAA) is reported as one of the dreaded complications after myocardial infarction
- The natural pathophysiology of LVAA formation is a sequela of underlying coronary artery disease (CAD), but it is also rarely seen in some patients with hypertrophic cardiomyopathy (HCM)
- Here we present a case of an underrecognized subgroup of HCM with LVAA in the absence of CAD. (figure 1a-b)

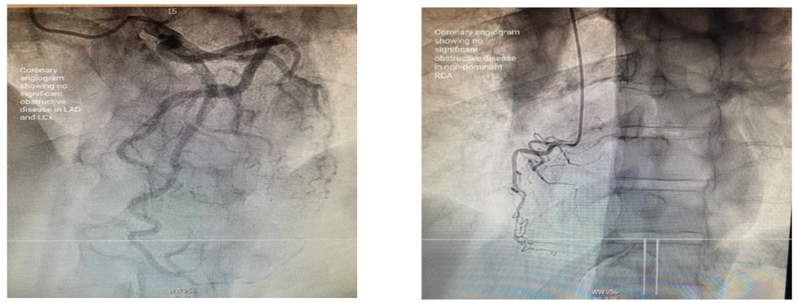

Figure 1a-b: Coronary angiogram showing no significant obstructive disease in LAD, LCX and RCA.

Case Presentation

- A 61-year-old African American male, known case of hypertension and COPD presented with the complaint of sudden onset chest pain and shortness of breath
- Afebrile, hemodynamically stable with an unremarkable cardiac exam
- Initial laboratory work was significant for Hs-troponin I of 16 ng/l and EKG exhibited no significant ST changes
- Transthoracic echocardiogram revealed HCM with isolated basal septal hypertrophy (maximal thickness of 21 cm), left ventricular ejection fraction > 70%, and apical aneurysm (figure 2 & 3)
- Cardiac catheterization was negative for coronary artery obstruction or significant left ventricular outflow tract obstruction
- Further investigation was unremarkable for infiltrative diseases
- Cardiac MRI revealed an asymmetric left ventricular hypertrophy involving the basal septal wall with small transmural scar in the apical segment suggestive of apical aneurysm
- Later, patient underwent implantable cardioverter-defibrillator (ICD) placement for primary prevention of ventricular arrhythmias
- Discharged home on beta-blockers and Coumadin with regular outpatient follow ups

Discussion

- LVAA is an outpouching of the apex of the left ventricular wall characterized by thin-walled dyskinetic or akinetic segments
- LVAA is usually caused by transmural infarct as a result of coronary artery obstruction
- On rare occasions, LVAA is associated with HCM as seen in our case

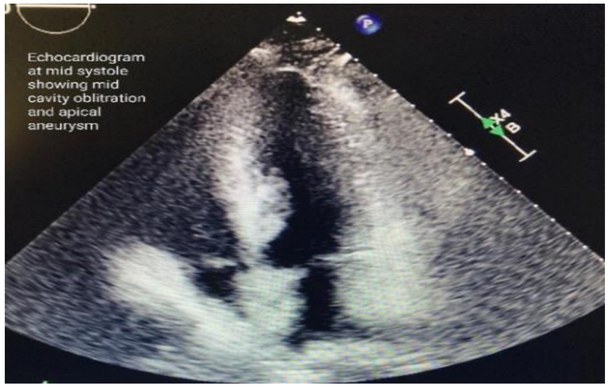

Figure 2: Echocardiogram at mid systole showing mid cavity obliteration and apical aneurysm.

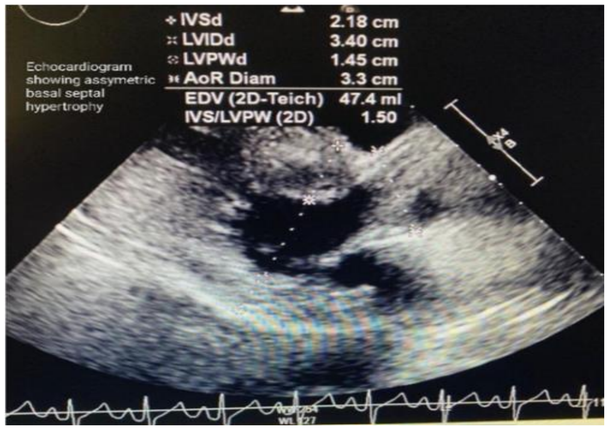

Figure 3: Echocardiogram showing asymmetric basal septal hypertrophy.

- The pathophysiology of LVAA in HCM is not well understood but several theories have been proposed
- Most appropriate for our patient is the difference in intracavitary systolic pressure, resulting in necrosis due to chronic subendocardial ischemia
- Thus, whenever LVAA is identified, HCM should be kept in the differential and further workup is warranted as treatment differs compared to patients with CAD[1]
- Patients with HCM with LVAA must undergo ICD placement for primary prevention of ventricular arrhythmias to reduce the risk of sudden cardiac death
- And also, should be started on anticoagulation to reduce thromboembolic events
- Whereas in the case of CAD resulting in LVAA, management would be guideline-directed medical therapy for obstructive CAD

References

1. Kai Yang, Yan-Yan Song, Xiu-Yu Chen, Jia-Xin Wang, Lu Li, Gang Yin, Yu-Cong Zheng, Meng-Die Wei, Min-Jie Lu, Shi-Hua Zhao, Apical hypertrophic cardiomyopathy with left ventricular apical aneurysm: prevalence, cardiac magnetic resonance characteristics, and prognosis, European Heart Journal - Cardiovascular Imaging, Volume 21, Issue 12, December 2020, Pages 1341–1350,

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