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 sequel 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)

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

Case Presentation

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

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

Figure 3: Echocardiogram showing asymmetric basal septal hypertrophy.

References