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A Shifting Cardiomyopathy: Transition Between Stress and Apical Variant Hypertrophic Cardiomyopathy: A Case Report

Cooper Erickson[1] and Grant Bailey[2]✉

[1]Alice L. Walton School of Medicine, 1007 NE J St Bentonville, Arkansas, USA

[2]South Denver Cardiology Associates, 1000 South Park Dr Littelton, Colorado, USA

Corresponding author: Dr. Grant Bailey, South Denver Cardiology Associates, 1000 South Park Dr Littelton, Colorado, USA. E-mail: grantb@southdenver.com.

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Abstract

Cardiomyopathies span a broad range of phenotypic expression and contribute to systolic and diastolic heart failure. In the present case, a previously healthy woman presented with a typical stress cardiomyopathy. Her hospital stay was uncomplicated and there was predictable resolution of LV function. Several months later, she elected to have left atrial appendage occlusion. Her case was complicated by a pericardial effusion due to a wire micro perforation. Repeat echocardiography weeks later revealed an apical variant hypertrophic cardiomyopathy with associated EKG changes. These echocardiographic and electrocardiographic abnormalities normalized in the months following. This represents a transient phenotype switching between a stress and apical variant HCM with possible novel genetic propensities for both.

Keywords: Apical hypertrophy; Stress cardiomyopathy; Shifting Cardiomyopathy; Echocardiography; Hypertrophic cardiomyopathy

Introduction

Cardiomyopathies, both congenital and acquired, span a broad range of phenotypic expression and contribute to systolic and diastolic heart failure [1]. The phenotypic expression of cardiomyopathy is broad–ranging from mild and subclinical left ventricular dysfunction to severe congestive symptoms. There is still much to learn about cardiomyopathies and their varied presentation. Stress cardiomyopathy is typically an acquired and reversible cardiomyopathy whereas apical variant hypertrophic cardiomyopathy has a well-defined genetically mediated pathway. We present a case of a ‘shifting cardiomyopathy’ with transient phenotypes of stress cardiomyopathy and apical variant hypertrophic cardiomyopathy.

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Case Presentation

A 72-year-old woman with a past medical history of well-controlled hypertension presented to the emergency department an acute episode of substernal chest pain. Notable results from initial laboratory testing included pro-brain Natriuretic peptide 8,594 pg/mL (normal ≤125pg/ml), troponin 81à84à89ng/L (normal ≤28 ng/L), Glomerular Filtration Rate 50.1 mL/min/1.73m*2 (normal >60 mL/min/1.73m*2), and sodium 134mmol/L (normal 136-145mmol/L). The initial EKG taken (Figure 1) is shown below. Further examination with a CTA chest showed no evidence of pulmonary embolism with trace right pleural effusion and nonspecific mediastinal and hilar lymphadenopathy. Given her presentation of chest pain and elevated troponin, coronary angiography was performed which demonstrated mild, non-obstructive coronary disease. A transthoracic echo (TTE) was performed, demonstrating apical ballooning and LV dysfunction with a left ventricular ejection fraction of 35% (Figure 1). The patient reported an extremely stressful event 48 hours prior to her presentation to the emergency room, and thus a diagnosis of stress cardiomyopathy was made. Her hospitalization was uncomplicated. Her troponins down trended and she was discharged on guideline directed medical therapy (GDMT) of spironolactone, carvedilol, and sacubitril-valsartan. She continued this medication regimen throughout the two-year period examined in this case report.

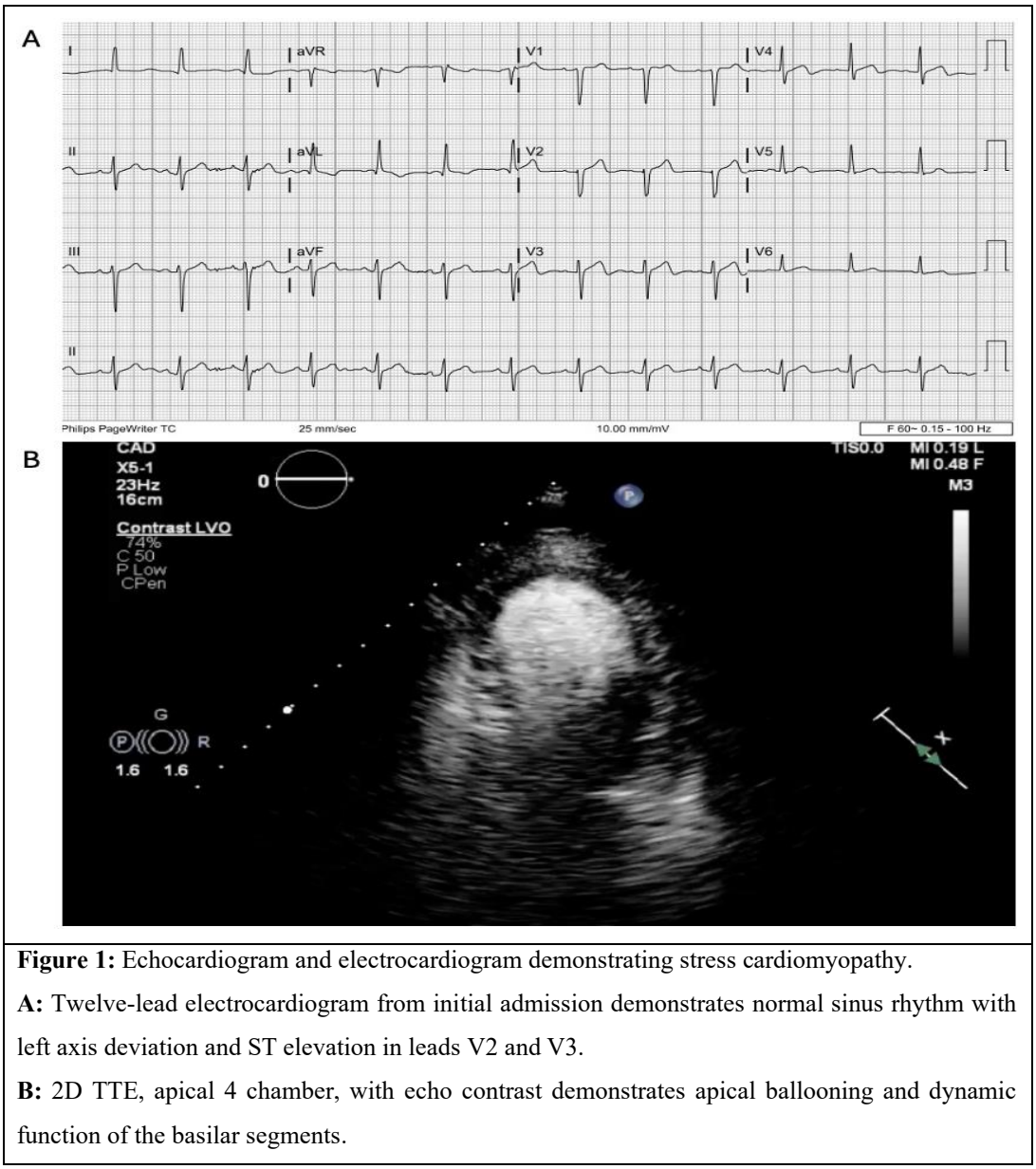

Figure 1: Echocardiogram and electrocardiogram demonstrating stress cardiomyopathy.

A: Twelve-lead electrocardiogram from initial admission demonstrates normal sinus rhythm with left axis deviation and ST elevation in leads V2 and V3.

B: 2D TTE, apical 4 chamber, with echo contrast demonstrates apical ballooning and dynamic function of the basilar segments.

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Two weeks later, she was briefly re-admitted with new onset atrial fibrillation. She was started on Apixaban and discharged. Repeat assessment of her left ventricular (LV) function was performed after 90 days of GDMT; a TTE at that time demonstrated normalization of her LV function without regional wall motion abnormalities (Figure 2). The apex was visualized and appeared normal in thickness and wall motion.

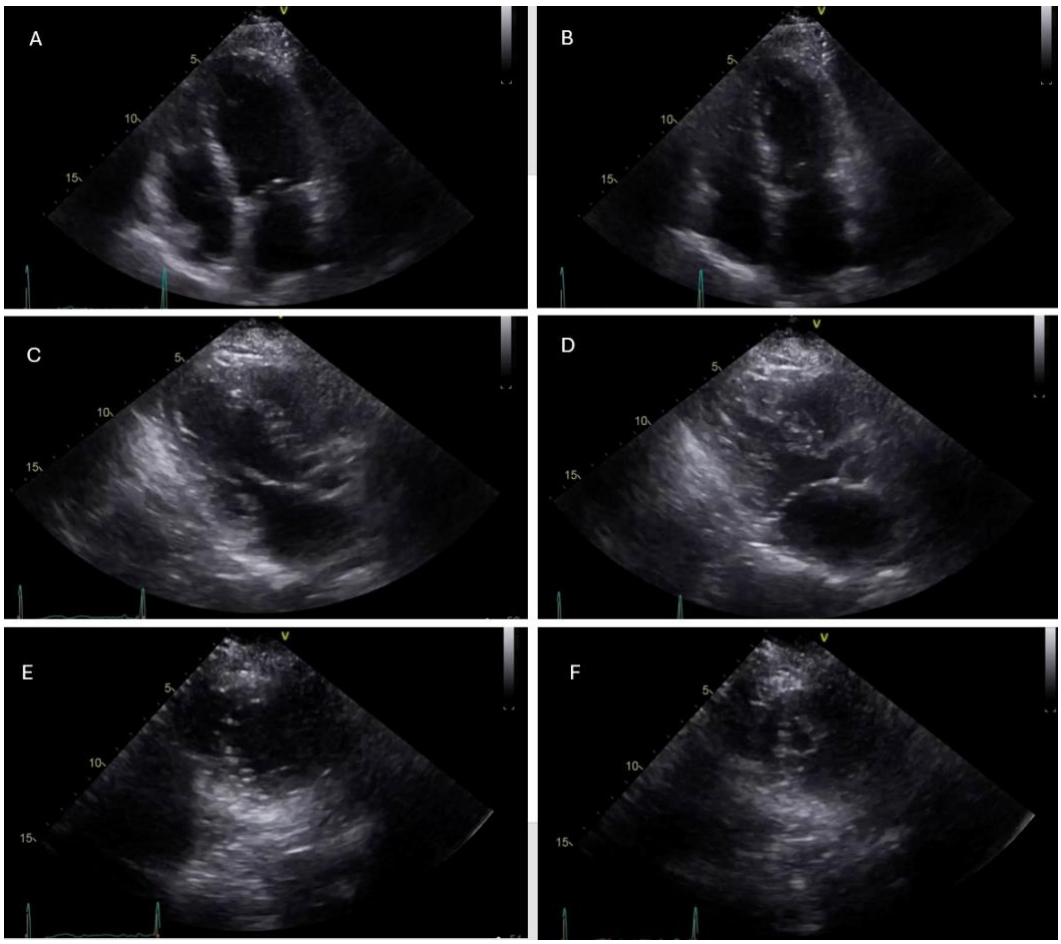

Figure 2: 2D Transthoracic echocardiogram demonstrating normal LV Function in apical 4 chamber (A at end diastole; B at end systole with normal apical wall thickness and function), parasternal long axis (C at end diastole; D at end systole), and parasternal short axis (E at end diastole; F at end systole) views.

Several months later, the patient elected to have left atrial appendage occlusion (LAAO) after having intolerance to Apixaban. The case was complicated by a pericardial effusion requiring needle drainage, resulting in acute pericardial inflammation and chest pain. This was complicated by episodes of recurrent pericarditis. Imaging was performed in the weeks following the LAAO procedure. A TTE performed 8 weeks following LAAO demonstrated the following (Figure 3). Findings were suggestive of asymmetric apical variant hypertrophy. An EKG demonstrated findings that were also consistent with new left ventricular hypertrophy with T wave inversions, typical of apical variant HCM (Figure 4) [2]. The demonstrated EKG changes are suggestive of myocardial hypertrophy rather than edema and fibrosis. As such, cardiac MRI was not performed.

Repeat imaging several months later with TTE demonstrated normalization of left ventricular wall thickness and wall motion. Her EKG pattern of LVH with strain typical of apical HCM also normalized (Figure 4).

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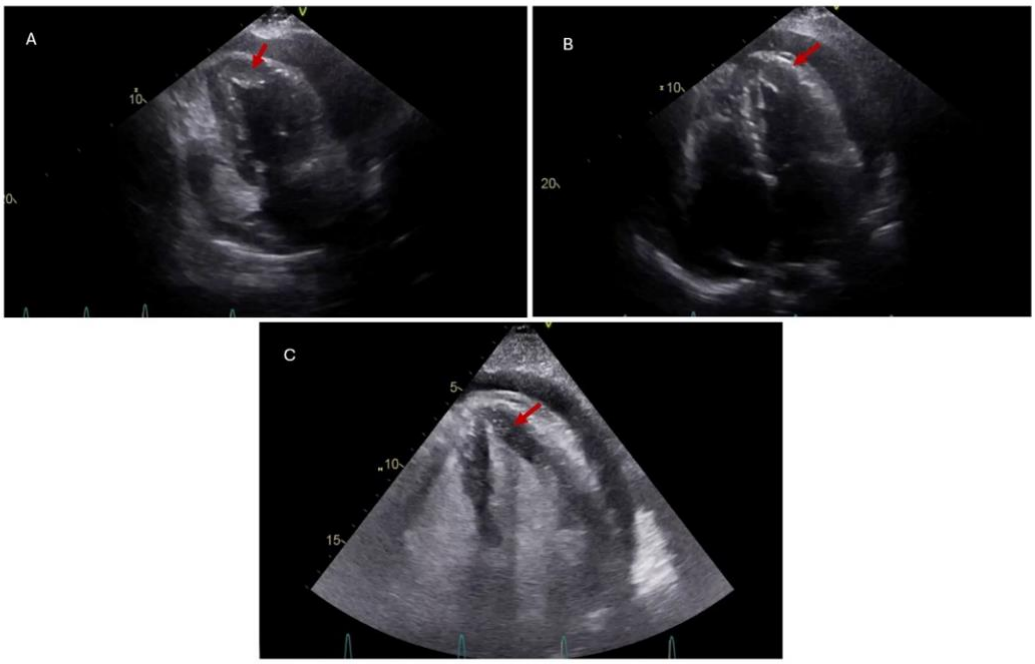

Figure 3: 2D Transthoracic echocardiogram demonstrating apical thickening consistent with apical variant hypertrophic cardiomyopathy in apical 3 chamber (A), apical 4 chamber (B), and apical 4 chamber with contrast (C, classic "spade-shaped" ventricle in systole).

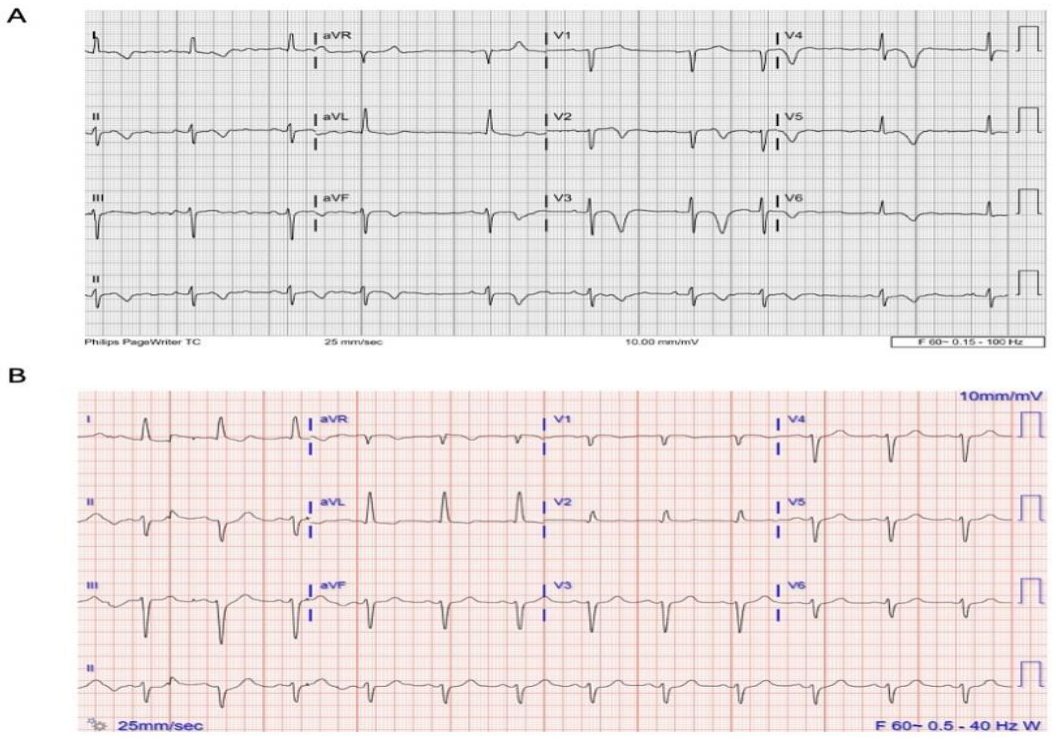

Figure 4: Twelve lead electrocardiograms demonstrating shift from apical variant hypertrophic cardiomyopathy to normal function.

A: Atrial fibrillation with giant T-wave inversion most prominent in leads V3, V4, and V5. This is characteristic of AVHCM.

B: Normal sinus rhythm. T-wave inversion is no longer present.

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Table 1: Timeline of case presentation.

Time (months since initial admission)	Notable Clinical Changes	Figures Representing Changes
0	Presentation to hospital with stress cardiomyopathy Echocardiogram demonstrated LVEF 35%	Figure 1
0.5	Presentation with atrial fibrillation	
3	LVEF normalization on echocardiogram	Figure 2
17	LAAO with wire micro perforation	
19	Apical Variant HCM on echocardiogram with associated EKG changes	Figure 3, Figure 4A
24	Normalization of LV function and EKG	Figure 4B

Discussion

Stress cardiomyopathy has previously been classified as a reversible and acquired cardiomyopathy with LV dysfunction mediated by catecholamine overproduction, coronary vasospasm, microcirculatory dysfunction, and sympathetic overdrive [3]. These acute changes can be triggered by emotional stress, physical stress, and other high catecholamine producing states such as catecholamine producing tumors [3,4]. During the acute phase, stress-induced cardiomyopathy can lead to the development of heart failure, arrythmias, thromboembolic events, and cardiogenic shock [3]. Electrocardiography of stress cardiomyopathy frequently demonstrates changes that mimic a ST-elevated myocardial infarction before normalizing [3]. Despite the typically acquired phenotype, recent cases of recurrent and familial stress cardiomyopathy (with 1.8% to 10% reported prevalence of relapse) suggest potential for a genetic predisposition which is currently poorly characterized [3,5].

In contrast to stress cardiomyopathy, apical variant hypertrophic cardiomyopathy (AVHCM) typically has a well-defined and genetically mediated pathophysiology resulting in myofibril hypertrophy and LV wall thickening [6]. Despite similar physiological changes and treatments, the prognosis of AVHCM is more favorable than that of other HCM with an overall low rate of cardiovascular mortality [6]. AVHCM is frequently misdiagnosed as an acute coronary syndrome (due to overlap in the potential symptoms) and the presence of coronary artery disease as a comorbidity in cases of AVHCM is a strong and negative predictor of survival [2,6]. Changes in AVHCM are represented in a typical fashion on EKG with giant T-wave inversion (>10mm in amplitude) most predominant in leads V4 and V5 [2,6,7]. These inversions are thought to be proportional to the extent of apical hypertrophy and occur concurrently with echocardiographic changes [2,6].

In the present case, the patient demonstrated all the necessary diagnostic criteria (including dynamic changes on EKG, apical ballooning, lack of coronary disease on invasive angiogram, and the report of a stressful event) for stress cardiomyopathy. In the weeks following, she developed atrial fibrillation and, in accordance with the accepted prognosis of stress cardiomyopathy, the patient's ventricular function and EKG normalized three months following the initial admission [3]. Normal left ventricular function was demonstrated on echocardiography for a year following the initial presentation before "shifting to an AVHCM" (with associated EKG abnormalities, deep T wave inversions and apical thickening on TTE) [2,6]. This was likely precipitated by a second stressful event, namely the iatrogenic cardiac injury with pericardial inflammation and pericarditis. There are very limited reports of transient development of LV hypertrophy and associated EKG changes which then normalize. Prior case reports have suggested myocardial edema as the cause of apical changes, although this seems unlikely given the concomitant increase in EKG voltage suggestive of myocardial hypertrophy [9-13].

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The combination of the echocardiographic and electrocardiographic findings suggest that this patient has a shifting cardiomyopathy with periods of normalization in ventricular function. The relationship between stress cardiomyopathy and AVHCM developed in this case is uncertain and merits further exploration.

Table 2: Comparison of distinguishing features of stress cardiomyopathy and AVHCM.

	Stress Cardiomyopathy,	**AVHCM**
Pathophysiology	Catecholamine overproduction Coronary vasospasm Microcirculatory dysfunction Sympathetic overdrive	Myofibril hypertrophy
EKG	ST Elevation	Deep T-Wave Inversions in precordial leads, LV strain
Echocardiogram	Apical ballooning, “octopus trap” appearance	Apical hypertrophy, “Spade-shaped” ventricle
Reversibility	Reversible	Irreversible
Triggers	Physical or emotional stress	Genetically mediated

Conclusions

Stress-induced cardiomyopathy is a usually reversible acquired cardiomyopathy with LV dysfunction, whereas AVHCM has a well-defined genetically mediated pathophysiology with a typical pattern of apical LV wall thickening. We observed a case in which a patient presented with independent episodes of stress-induced cardiomyopathy and then developed AVHCM after a second stressor; both cardiomyopathies resolved with medical treatment. We proposed that this represents a ‘shifting cardiomyopathy’ which suggests a new mechanism underlying stress cardiomyopathy, with possible genetic propensities for both types of cardiomyopathies.

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