

# A Shocking Discovery: Apical Thrombus Formation Post-Cardiac Resynchronization Therapy Placement



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## INTRODUCTION

Biventricular (Bi-V) implantable cardioverter-defibrillator (ICD) placement, otherwise known as cardiac resynchronization therapy with a defibrillator (CRT-D), has been shown to create immediate hemodynamic benefits (increased systolic blood pressure, increased cardiac output, and increased contractility), improve left ventricular (LV) systolic function, and promote LV reverse remodeling.<sup>1</sup> Common indications for placement include patients with LV ejection fraction (LVEF) < 35%, QRS > 150 ms with a left bundle branch block (LBBB), and New York Heart Association class II-IV heart failure.<sup>2</sup> Rare complications following intracardiac pacing devices include lead thrombosis,<sup>3</sup> ventricular aneurysmatic area changes,<sup>4</sup> and LV dyssynchrony.<sup>5</sup> All three of these aforementioned complications can lead to disruption of laminar flow, blood stasis, and ultimately thrombus formation, with systemic thromboembolism being the feared outcome. This report demonstrates an echocardiographic finding of a large LV apical thrombus after a Bi-V ICD was placed. This alarming finding may have been due to the ballooning of an aneurysmal and akinetic LV apex, worsened by the forces generated by the cardiac resynchronization therapy.

## CASE PRESENTATION

A 67-year-old African American male presented with a history of mixed cardiomyopathy, an LVEF of 25% with LBBB, and New York Heart Association II systolic heart failure. During a severe case of pneumonia months prior, the patient's LVEF was noted to have fallen from 40%-45% to 20%-25%. A cardiac catheterization was performed and indicated no acute ischemic process to explain the sudden drop in LVEF (Video 1). The patient was known to have a myocardial infarction in his 20s that led to him having an anterior wall scar. His electrocardiogram (EKG) was pertinent for an LBBB with wide QRS duration (Figure 1). He had a LifeVest placed prophylactically after the decrease in LVEF.

A cardiac positron emission tomography (PET) scan was ordered to evaluate the function of this anterior wall. This cardiac viability and perfusion study noted that in addition to his expected LV hypertrophy and lack of viable myocardium in the mid to apical intraventricular septum from the old myocardial infarction, there was no activity in his apical cap, resulting in a wide-mouthed true LV apical aneurysm (Figure 2). An echocardiogram was obtained that showed, as expected, a hypokinetic apical lateral wall but no areas of akinesis in

## VIDEO HIGHLIGHTS

**Video 1:** Coronary catheterization after the patient's initial drop in ejection fraction, showing no new coronary stenosis.

**Video 2:** Echocardiogram before CRT-D placement showing ventricular dyssynchrony.

**Video 3:** CRT-D with optimal lead placement.

**Video 4:** Follow-up echocardiogram after CRT-D placement showing the newly found LV thrombus sitting within an akinetic apex.

**Video 5:** Echocardiogram 3 months after initiation of warfarin

**Video 6:** Echocardiogram with Definity contrast 1 year after discovery of the mural thrombus, showing resolution of the mural thrombus and improved contractility with a more clearly defined apical aneurysm.

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the myocardium or presence of a mural thrombus. The basal lateral and midlateral wall segments showed dyssynchrony secondary to the patient's LBBB (Video 2). Considering his EKG findings in addition to the decreased LVEF, it was deemed necessary to have CRT-D placement for the purpose of cardiac resynchronization therapy.

Chest x-ray confirmed optimal placement of the leads (Figure 3A and 3B), including the LV lead in the basal lateral site farthest away from the RV apical lead position (Video 3). On follow-up the patient reported feeling more energetic and there were no significant findings on the echocardiogram. At 3 months, an echocardiogram was obtained, and the patient was noted to have an improved LVEF of 35%. Unexpectedly, there was now a large round thrombus in the LV apex measuring 3.60 × 2.45 cm (Figure 4). Although the patient's LVEF improved on this echocardiogram as expected after CRT placement, it also revealed new apical akinesia with ballooning on systole (Video 4). The patient was immediately started on warfarin and lovenox. After 3 months on warfarin, the thrombus had decreased in size (Figure 5, Video 5), and at 1 year the thrombus was confirmed resolved with an ejection fraction of 45% on echocardiogram with Definity contrast (Figure 6, Video 6). The patient from then on was

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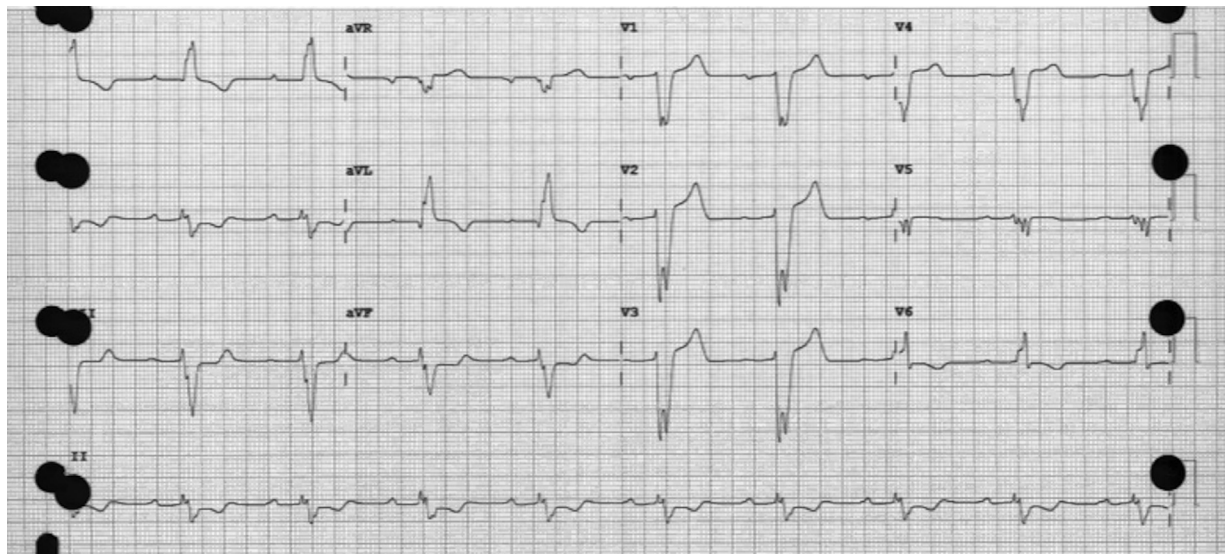
Keywords: Heart failure, Aneurysm, Apical thrombus, CRT-D, Echocardiogram

S.S. receives research grants and contracts from Medtronic and is a member of the speakers bureau for Pfizer and Bristol-Myers-Squibb.

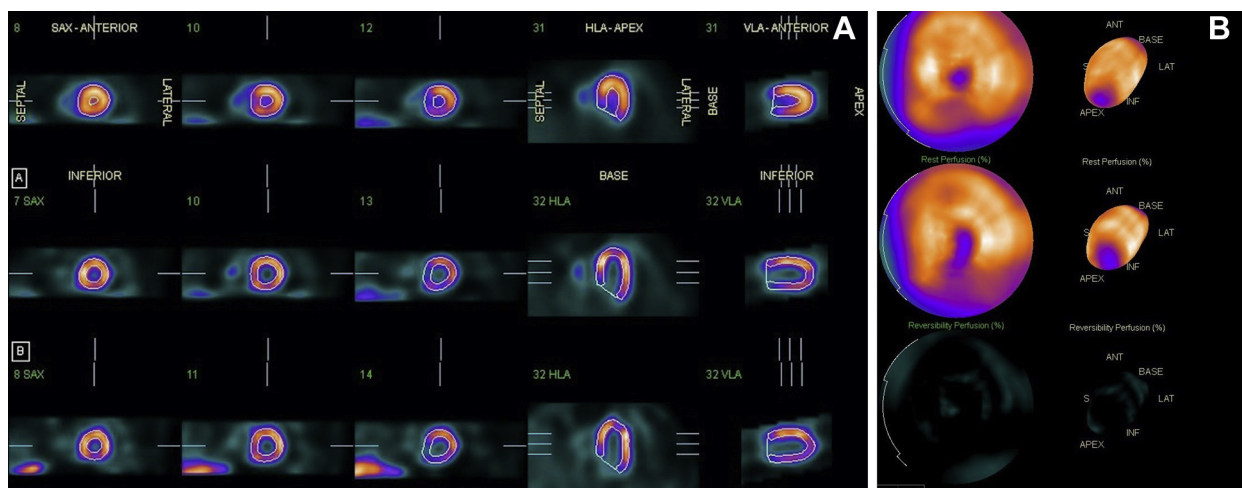
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**Figure 1** EKG showing an LBBB with widened QRS.



**Figure 2** Cardiac PET viability (A) and perfusion (B) study showing no activity in the apical cap with what was described as a “true LV apical aneurysm.”

maintained on rivaroxaban and seen by cardiology every 6 months where he reports improved symptoms and has routine echocardiograms.

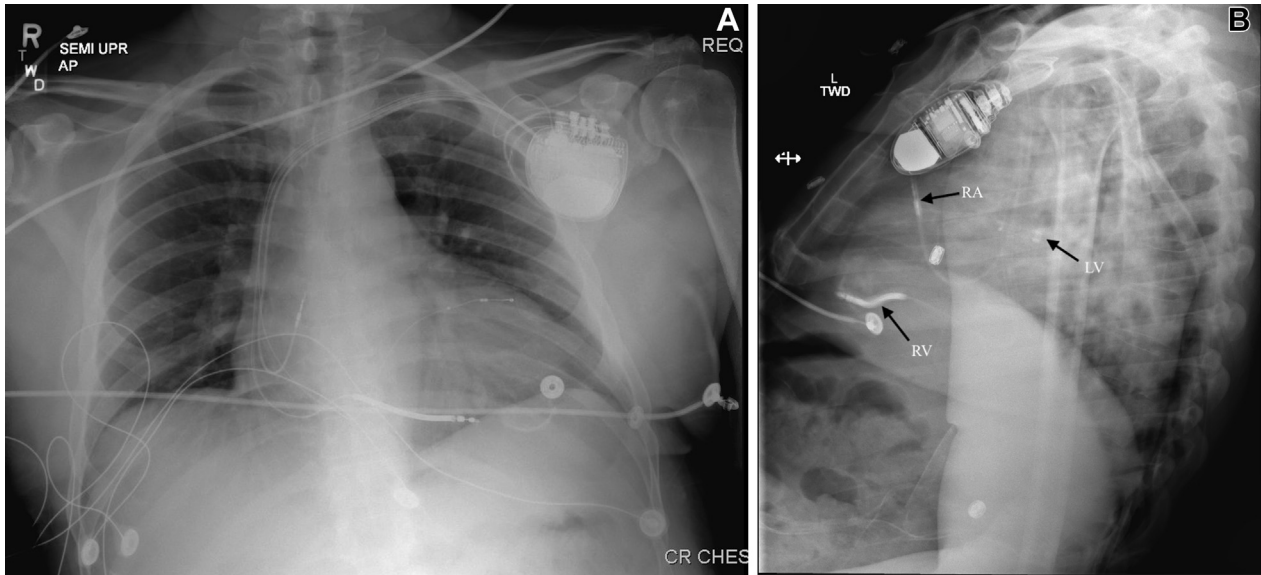
## DISCUSSION

The medical literature does not have much data on the occurrence of worsened cardiac dynamics post Bi-V ICD placement, let alone the added formation of a large apical thrombus. In our literature review, mural thrombus within a ventricular aneurysm after CRT-D placement has not been previously described.

In trying to determine why this apical thrombus formed after device placement, we must consider all three components of Virchow’s triad. Virchow’s triad tells us that for thrombus formation to occur, a combination of hypercoagulability, endothelial injury, and blood stasis

must occur. The patient had thus far never exhibited hypercoagulability. As mentioned previously, the echocardiogram obtained 1 month prior to device placement was without thrombus, indicating that the presence of a depressed ejection fraction and apical aneurysm by itself was not the primary driver for thrombus formation. Even though cardiac output and stroke volume can decrease with dyssynchrony, that alone would not be enough stasis to create this form of thrombus. With these facts in mind, we must focus our attention on the apical aneurysm seen even before device placement.

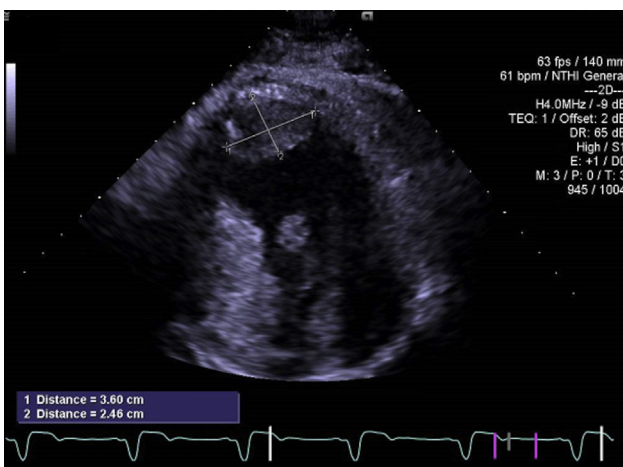
Cardiac PET confirmed before CRT-D placement that the patient was had no activity in the apical cap with a true LV apical aneurysm. A true LV aneurysm represents an area of thin myocardial wall with all three layers of heart tissue intact, composed of necrotic and fibrous tissue seen in infarcted myocardium. This is opposed to a pseudoaneurysm or “false” aneurysm, which is created when an LV wall



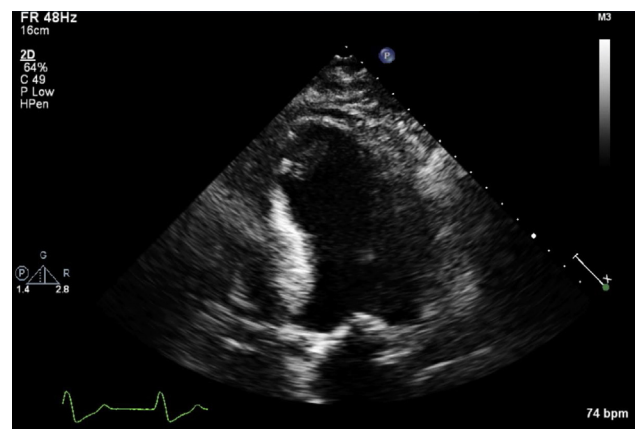
**Figure 3** (A) Chest x-ray showing LV hypertrophy. (B) The lateral view shows lead placements in each of the three chambers including the LV lead in the basal lateral site farthest away from the right ventricular apical lead position.

rupture is contained by pericardium without intact heart wall. This lack of intact wall can be seen in imaging where a pseudoaneurysm has a narrow “neck” or opening into a wide apex, which is in contrast to the wide neck of a true aneurysm as seen in our case. This distinction is significant, as a pseudoaneurysm is a potential medical emergency often requiring surgical attention. Left bundle branch block creates LV dyssynchrony, which was corrected by initiating cardiac resynchronization therapy. Although echocardiograms previous to CRT-D placement did not detect any apical akinesia, the prior cardiac PET and the echocardiograms after the device placement did. The discrepancy between PET and the echocardiograms can be explained in one way by the objective nature of cardiac PET and subjective nature of echocardiogram. Echocardiograms are much more subject to the

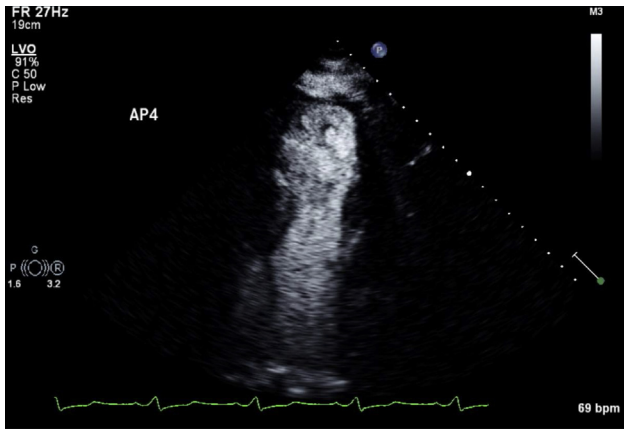
experience of the reader and inter-reader variability. Furthermore, the dyssynchrony and poor ejection fraction of the predevice echocardiograms created difficulty in evaluating comparative areas of hypokinesis and akinesis. It is easy to see the akinetic ventricular apex in postdevice echocardiograms, especially when a contrast agent was used (Figure 6), as the aneurysm was made more apparent by the fully functioning CRT-D. As intended, CRT-D generated more lateral LV forceful contraction than before, generating enough force through the ventricle to move blood across the aortic valve and toward the apical aneurysm. It is possible that the apical aneurysm might have increased in size with the more forceful contraction from the basal lateral pacing action. Thus, we meet Virchow’s requirement for the necessary blood stasis for LV thrombus formation.



**Figure 4** Frame from the echocardiogram taken after CRT-D placement showing the measurements of a newly found mural thrombus.



**Figure 5** Frame taken from the echocardiogram after 3 months on anticoagulation showing the thrombus in a ballooning apex during systole.



**Figure 6** Frame taken from the echocardiogram 1 year after the discovery of the mural thrombus. Definity contrast shows complete resolution of the clot and clear definition of the aneurysm.

In our patient it is likely the Bi-V pacing “ballooned up” the akinetic apex, creating nonlaminar flow and an increase in blood stasis, ultimately creating a large thrombus sitting within an aneurysm.

## CONCLUSION

This case is unique in showing that CRT-D has the potential for creating nonlaminar blood flow within an aneurysmal cardiac apex. Practitioners should be mindful of possible areas of aneurysm when placing these devices as the increased contraction provided to viable myocardium by the device can exacerbate the ballooning of nonviable myocardium that developed from previous infarction.

## SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2020.09.003>.

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