# Biventricular Apical Thrombi in a Patient Presenting with New-Onset Dilated Cardiomyopathy

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### **ABSTRACT**

The diagnosis of an apical left ventricular thrombus in the setting of a dilated cardiomyopathy is not uncommon. However, biventricular apical thrombi in this setting is unusual. We present a case of a 67-year-old man who was admitted with new onset heart failure with biventricular apical thrombus formation in the absence of a hypercoagulable state.

Keywords: Cardiomyopathy, echocardiography, thrombus

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#### INTRODUCTION

A 67-year-old African-American male with a past medical history of hypercholesterolemia and hypertension presented with a 3 week history of worsening shortness of breath and lower extremity edema. He denied alcohol use and familial history of cardiomyopathy. On physical examination patient was found to have notable bilateral lower extremity pitting edema along with an S3 on auscultation in the left precordium. Vitals signs were heart HR/min 91, BP in mmhg 105/63, with a respiratory rate of 19. Electrocardiogram demonstrated a left bundle branch block. Transthoracic echocardiogram revealed severe global hypokinesis with an ejection fraction of 20% with marked dilatation of both the left and right ventricles as well as the presence of  $3 \times 2$  cm biventricular apical thrombi. Left ventricular end-diastolic diameter was 6.9 cm, with a right ventricle mid cavity diameter of 3.9 cm. There was an abnormal septal motion due to

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an intraventricular conduction delay, without evidence of constrictive pericarditis [Figure 1 and Video 1]. Hypercoaguable workup was unremarkable and included evaluation of platelet count, prothrombin time, activated partial thromboplastin time, fibrinogen, thrombin time, antiphospholipid antibody, heparin-induced thrombocytopenia, factor five resistance, protein C and S activity, Antithrombin III levels. His complete blood count, including eosinophil count, was normal. Tropinins, ck-mb, and myoglobin levels were also unremarkable. Tranesophageal echocardiography was not performed secondary to patient refusal. A coronary computerized tomographic angiography revealed a coronary artery calcium score of 630 Agatston units, with no definite hemodynamically significant coronary stenosis. The patient was treated with 3mg of coumadin PO titrated to an INR between 2.0 and 3.0 as well as guideline directed heart failure therapy. Cardiothoracic surgery was consulted for evaluation of thrombus removal. Because of the stationary position of the thrombi, they

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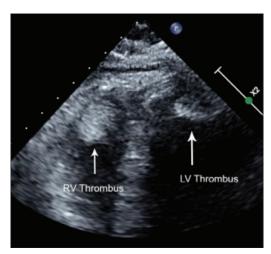


Figure 1: Apical four chamber view demonstrating bilateral apical ventricular thrombi. (LV- Left ventricle, RV-Right ventricle)

recommended solely anticoagulation. The patient desired no further intervention, workup, or scheduled follow-up.

#### DISCUSSION

An isolated left ventricular apical thrombus is well reported in patients with significantly impaired left ventricular systolic function such as dilated and/or ischemic cardiomyopathy, especially in the setting of an acute myocardial infarction. <sup>[1,2]</sup> It has also been reported in patients with hypereosinophilic cardiomyopathy, and in hypercoaguable states. <sup>[3]</sup> However, the presence of simultaneous biventricular apical thrombus in the absence of acute myocardial infarcation and hypercoaguable states is seldom reported in literature. Although this rarity has an association with peripartum cardiomyopathy, it thought to be influenced by the hypercoaguable pregnant state. <sup>[4]</sup>

The detection of ventricular thrombi using echocardiography is sometimes difficult as well as its treatment strategy.<sup>[5]</sup> Due to the possibility of mobility of thrombi, medically treating with anticoagulation or surgical removal have been performed successfully.<sup>[2,5]</sup> Surgical removal of biventricular thrombi has scarcely been reported, but several cases have been successful in removal of left ventricular thrombus via atriotomy or ventriculotomy approach with manual clot removal. [6] Cardiac CT is a noninvasive way of obtaining information about calcified plaque in the coronary arteries to suggest if coronary artery disease is present. Calcium score scaling ranges from 0 to 400, with 0 showing no evidence of coronary disease, and >400 demonstrating extensive evidence of coronary disease. The patient demonstrated a score of 630, suggesting that extensive coronary artery disease likely contributed to his acute cardiomyopathy.<sup>[7]</sup> A cardiac MRI to rule out myopericarditis was not performed due to the fact that the patient did not have clinical findings such as chest pain, elevated troponins, or segmental wall motion abnormality on echocardiogram to suggest myopericarditis. The patient presented with acute cardiomyopathy in the absence of a hypercoaguable workup. The lack of thrombus mobility was a main factor against surgical intervention. Although there are no guidelines, it can be presumed that if the thrombus has a low chance of embolization, oral anticoagulation can be warranted over surgical intervention. Because the patient did not desire interventional workup or treatment, the only form of treatment was anticoagulation therapy with coumadin.

# Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initial s will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

# Conflicts of interest

There are no conflicts of interest.

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