Biventricular Mural Thrombi in Patients With Dilated Cardiomyopathies: Case Reports and Review

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Introduction: The combination of the aging of the population and improved survival after acute myocardial infarction has created a rapid growth in the number of patients currently living with chronic heart failure, with a concomitant increase in morbidity and mortality.

Case Presentation: We present two case reports of post-myocardial infarction sequel leading to ischemic cardiomyopathy and peripartum cardiomyopathy leading to biventricular mural thrombi formation and provide a brief review of literature regarding their etiopathogenesis and management.

Discussion: There are other causes of dilated cardiomyopathies which could be transient like peripartum cardiomyopathy. The development of biventricular mural thrombi is rare, and it mainly increases the risk of embolization in the systemic and pulmonary circulations.

Keywords: Dilated Cardiomyopathy; Heart Failure; Echocardiography

1. Introduction

The detection of biventricular mural thrombi is rare during routine echocardiography, although this imaging modality is a valuable tool for their diagnosis. We describe two patients who presented with symptoms of heart failure with different etiologies whose routine echocardiography revealed biventricular thrombi. We also discuss the possible mechanism of biventricular thrombus formation and its management in these cases and present a brief review of literature.

2. Case Presentation

2.1. Case One

A 38-year-old man presented with symptoms of breathlessness on exertion (New York Heart Association [NYHA] class III) with paroxysmal nocturnal dyspnea of one week's duration. He had suffered extensive anterior wall myocardial infarction three months before, for which he was administered thrombolytic therapy in the form of injection of 1.5 million units of streptokinase. However, as he developed massive gastrointestinal bleeding, he was placed on conservative medical management. At the time of admission, he was on oral Clopidogrel (75 mg per day) and Atorvastatin (20 mg per day). Upon admission, he was started on anti-fail-ure treatment, including intravenous diuretics, angiotensin-converting enzyme inhibitors, statins, and beta blockers, which relieved his symptoms (NYHA class II).

To our surprise, two-dimensional echocardiography showed a large mural thrombus in the left ventricle (LV) extending from the mid and distal septal segments into the LV apex and thence to the apical anterior and apical inferior segments (Figure 1 A). The LV was dilated with akinetic and scarred segments supplied by the left anterior descending artery (LV ejection fraction = 35% by modified Simpsons' method). The other segments were contracting normally. There was no mitral regurgitation or pulmonary hypertension. The right ventricle also showed a small thrombus attached to the mid part of the interventricular septum (Figure 1 B). The right atrium and ventricle were normal in size.

The patient was started on low-molecular-weight Heparin (1 mg/kg/dose) twice a day. His coagulation profile showed elevated lipoprotein and homocysteine levels. Hence, he was started on 145 mg per day of Fenofibrate and 5 mg per day of oral folic acid. The patient was discharged on oral anticoagulation (Warfarin 5 mg) with the advice of bed rest and close follow-ups, with a plan of surgical intervention if the thrombi did not dissolve. At six-months follow-up, two-dimensional echocardiography revealed the disappearance of the right ventricular thrombus and a significant reduction in the left ventricular thrombus.

Figure 1. Two-Dimensional Echocardiography of Ischemic Cardiomyopathy
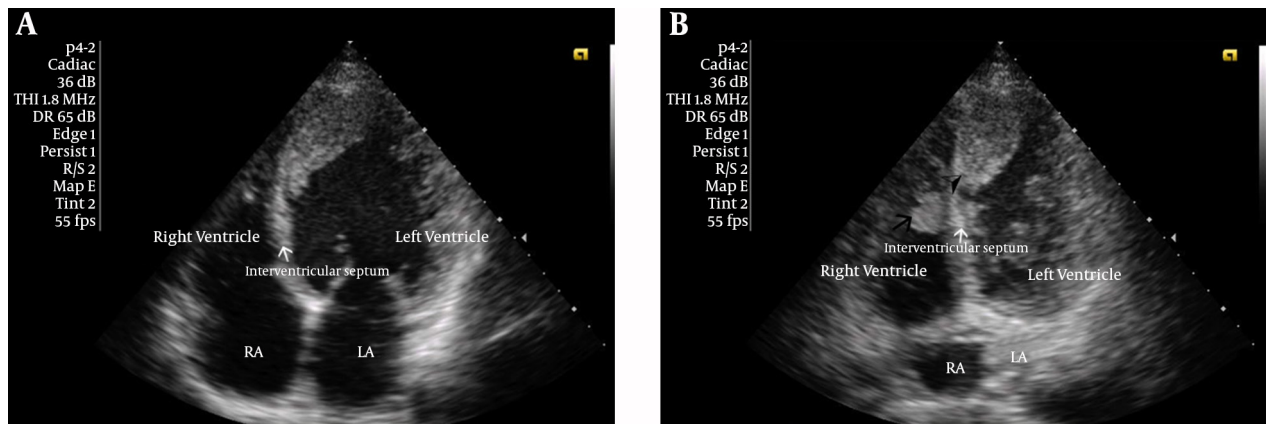
A) Two-dimensional echocardiograph in the four-chamber view in diastole, showing a dilated left ventricle with a left ventricular mural thrombus attached to the distal septum and apical segments. The left atrium (LA), right atrium (RA), and right ventricle are normal in size and function. B) Modified four-chamber view, showing the left ventricular apico-setpal attachment of the mural thrombus (arrow head). In addition, there is another mural thrombus attached to the mid septal segment of the right ventricle (arrow).

Figure 2. Two-Dimensional Echocardiography of Peripartum Cardiomyopathy
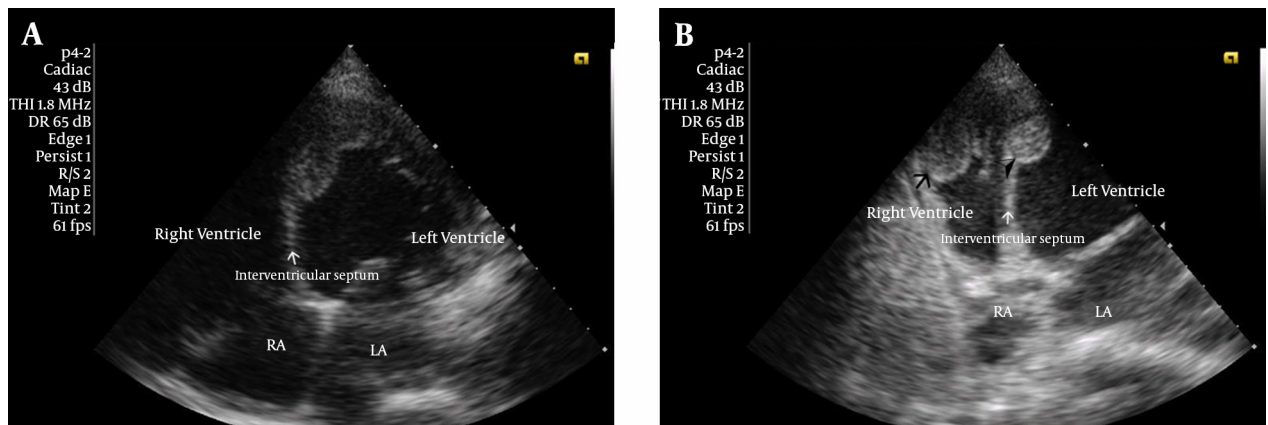
A) Four-chamber view in diastole, showing the dilatation of the left ventricle, right ventricle, left atrium (LA), and right atrium (RA) with a large mural thrombus attached to the distal septum and apical segments. B) Modified four-chamber view with an apical tilt to delineate the thrombi, showing biventricular thrombi. In addition to the left ventricular thrombus (arrow head), a right ventricular mural thrombus (arrow) is attached to the apical right ventricular free wall.

2.2. Case Two

A 28-year-old female patient presented one month after her first pregnancy in the outpatient department with complaints of breathless at rest with orthopnea (NYHA class IV) and bilateral pedal edema. There was no history of previous cardiac ailment in the past. Two-dimensional echocardiography was advised by the general physician as the chest X-ray showed cardiomegaly. Echocardiography revealed biventricular dilation with a reduced LV ejection fraction (LV ejection fraction = 32%). There was global hypokinesia of the left and right ventricles. There was a large LV apical mural thrombus starting from the distal septal segment (Figure 2 A).

Also, there was a right ventricular apical mural thrombus starting from the distal free wall of the right ventricle (Figure 2 B).

The clinical diagnosis of peripartum cardiomyopathy was made, and the patient was started on oral diuretics, angiotensin-converting enzyme inhibitors, oral Carnitor, and oral anticoagulation. She showed significant improvement of symptoms. At regular follow-ups, there was a complete resolution of the biventricular thrombi at the end of 4 months. Repeat two-dimensional echocardiography demonstrated mild LV dysfunction (LV ejection fraction = 46%). Hence, the patient was advised

to continue oral anticoagulants until further improvement in the biventricular function. Her coagulation profile was normal.

3. Discussion

The LV thrombi usually occur in the setting of acute myocardial infarction, LV aneurysms, or dilated cardiomyopathy (1). In the absence of ventricular wall motion abnormalities, these thrombi are rare (2). The LV thrombi occur in at least 5% of patients after acute myocardial infarction, which increases the morbidity and mortality of patients (3). Peripartum cardiomyopathy is a rare cause of congestive heart failure which develops in the last month of pregnancy or during the first 5 postpartum months in women without a previously known cardiac disease. It is a rare cause of intraventricular thrombus formation (4). As such, any etiology which causes transient or permanent insult/damage to the myocardium leading to dilated cardiomyopathy (ischemic/nonischemic) can cause the formation of thrombi because of the akinesia/dyskinesia of the apical segments of the LV wall.

The characterization of thrombi can help predict the risk of embolism, which is higher for sub-acute clots than for organized thrombi. Mobile, larger thrombi extending up to the LV outflow tract are at higher risk of embolization (5, 6). The risk of systemic embolization is between 5% and 50%, particularly cardio-embolic stroke and acute limb ischemia (1). The embolization of a right ventricular thrombus can lead to pulmonary embolism and infarction (7).

Echocardiography is valuable for the diagnosis and characterization of the LV thrombi. (8) It is reproducible and can be done bedside in sick patients. Cardiac magnetic resonance imaging (MRI) has an approximate sensitivity of 90% in detecting ventricular thrombi using gadolinium contrast. Delayed enhancement cardiac MRI is the gold-standard test for detecting the complications of ventricular dysfunction and also studying the viability of the myocardium (9, 10). Though contrast echocardiography also can be used for the diagnosis of ventricular thrombi, it has a sensitivity of 60%. In our cases, the diagnosis of biventricular thrombi was evident on two-dimensional echocardiography, precluding the need for other imaging modalities.

To our knowledge, there are no randomized clinical trials on this topic in the existing literature, and our experience is based on individual case-based studies. Medical management involves mainly oral anticoagulation to dissolve thrombi and prevent further recurrences (7). The European Heart Journal guidelines of 2012 state that patients with mural thrombi require oral anticoagulation with vitamin K antagonist therapy for up to 6 months. According to the 2013 American College of Cardiology/American Heart Association (ACC/ AHA) guidelines, vitamin K antagonist therapy can be limited to three months in patients with LV thrombi or are at risk of LV thrombi such as patients with antero-apical akinesis or dyskinesis (11).

The combination of oral anticoagulation with dual antiplatelet therapy increases the risk of bleeding. Accordingly, we suggested oral anticoagulation with one antiplatelet agent at least for our patients. Given the existing controversy surrounding the duration of oral anticoagulation therapy and case reports of the recurrence of ventricular thrombi (11), we advised lifelong therapy or therapy until improvement in the LV function.

A small number of case reports have hypothesized that the presence of the hypercoagulable state may be an important causative mechanism for the formation of the LV thrombi. Laboratory tests like fibrinogen, protein S, protein C, antithrombin III, Factor V Leiden, lupus antibody, and homocysteine should be performed in all cases to rule out the hypercoagulable state (12). The correction of the underlying factors such as hyper eosinophilia, hypercoagulable states, and chronic inflammatory disorders (e.g. ulcerative colitis) may help resolve ventricular thrombi (13-15).

The hyper eosinophilia syndrome with Loffler's endocarditis can present with biventricular mural thrombi, bilateral lung infiltrates, deep vein thrombosis, or pulmonary embolism. Usually diagnosis is confirmed by blood picture, which reveals peripheral blood eosinophilia more than 1500 cells/mm^3 for at least a 6-month period after ruling out other secondary causes of hyper eosinophilia. Usually these patients respond to steroid therapy with the resolution of the symptoms and biventricular thrombi (14, 16).

Larger thrombi which are at the risk of embolization need aggressive management. Fibrinolysis should not be advocated as it carries risk of embolization at the time of the lysis of the thrombus (17). Additionally, it carries higher risk of hemorrhagic complications.

The management of these patients mostly remains conservative, but surgical thrombectomy has been described along with myocardial revascularization. Patients with large LV apical aneurysms with thrombi and those who have refractory heart failure symptoms despite optimal medical therapy benefit from surgical LV restoration procedures such as Dor' procedure aimed at reducing the LV volume and restoring the heart geometry (18).

Echocardiography is the gold-standard technique for the diagnosis of ventricular thrombi. Anticoagulation is the accepted therapy to resolve thrombi and to prevent embolization, but it is probably insufficient in situations of high embolic risk, in which surgical thrombectomy with or without surgical LV restoration may be indicated. There are, however, no specific scientifically validated guidelines as to the best therapeutic approach. More randomized clinical trials or observational retrospective data will delineate the future course of patients presenting with biventricular thrombi formation secondary to dilated cardiomyopathy.

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