

## SUCCESSFUL MANAGEMENT OF BIVENTRICULAR THROMBI WITH ANTICOAGULATION IN A PATIENT WITH PERIPARTUM CARDIOMYOPATHY

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**Bu yayında biventriküler trombüs ile komplike olan kardiyomiyopati vakası sunduk. Hastaya heparin tedavisi başlandıktan bir gün sonra ekokardiyografide trombüs hacminin azaldığı, warfarin tedavisi başlandıktan 2 hafta sonra ise trombüsün kaybolduğu saptandı.**

**Anahtar kelimeler: Biventriküler trombüs, Peripartum kardiyomiyopati**

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### INTRODUCTION

Peripartum cardiomyopathy is a rare cause of congestive heart failure that develops in the last months of pregnancy or during five months postpartum in women without previously known cardiac disease<sup>1</sup>.

Although the etiology has not been determined, investigators have noted a high incidence of embolism with peripartum cardiomyopathy<sup>2</sup>. Cardiac mural thrombi have been found at autopsy in some patients and thrombi have been demonstrated in the left ventricle, and in a few instances in the right ventricle, by 2-dimensional echocardiography<sup>3</sup>.

We present a case of peripartum cardiomyopathy with biventricular thrombi that was managed successfully using anticoagulant therapy.

### CASE

A 38-year-old woman was admitted to emergency department with palpitation, paroxysmal nocturnal dyspnea, and bilateral lower extremity edema 10 weeks after a normal third delivery.

There was no family history of cardiomyopathy or pregnancy-induced hypertension.

On physical examination she had tachycardia (124/bpm), blood pressure of 112/78 mmHg, third heart sound, a grade 2 pan systolic murmur at the apex, and

fine rales in lower lung fields bilaterally and bilateral lower extremity edema.

Cardiomegaly was present on the chest radiography. The electrocardiogram revealed sinus tachycardia, negative T waves in leads V2-6 and DI -aVL, and QT prolongation (QTc 0.45 s).

Echocardiography showed left ventricular dilation associated with global hypokinesia, grade 2 mitral and grade 2 tricuspid regurgitation, and apical thrombi in both ventricles (Figure 1). Left ventricular end diastolic dimension was 55 mm and ejection fraction was calculated as 36%. Right ventricle dimension was within normal limits. The thrombi were spherical, pedunculated,

**Figure 1:** Biventricular thrombi were spherical, pedunculated, shaggy and irregular in configuration, and freely mobile



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shaggy and irregular in configuration, and freely mobile (Figure 1).

Routine biochemical laboratory findings, including cardiac enzymes, were within normal limits. Anticar-diolipin, D dimer, Protein C and S were within normal limits whereas brain natriuretic peptide was elevated.

Therapy with furosemide, perindopril and anticoagulation with heparin and warfarin was initiated. Echocardiography was repeated 24 hour after intravenous heparin (aPTT was maintained between 50-70 sec) and it was seen that there was regression in the size of thrombi. After reaching an INR of 2 heparin was discontinued. A subsequent echocardiogram performed 4 weeks later showed no evidence of apical thrombi in either ventricle. Two months after admission, the left ventricular end-diastolic dimension and ejection fraction had recovered to 51 mm and 53%, respectively. However, the negative T wave changes on electrocardiogram remained.

## DISCUSSION

The true incidence of peripartum cardiomyopathy is unknown and estimates vary from 1:15,000 to 1:1,300 deliveries. The return of the left ventricle to normal size and function usually occurs within 5 months postpartum in 50% of patients and is considered an important prognostic factor for survival. High parity, twin gestation, age >30 years, conduction defects on electrocardiography, and late onset of symptoms after delivery are unfavorable prognostic factors<sup>4</sup>.

There are several case reports of peripartum cardiomyopathy with intracardiac thrombus diagnosed by echocardiography, but to our knowledge, only three of those reports demonstrated biventricular thrombi on 2-D echocardiography<sup>5,6,7</sup>, and so our finding is quite rare. Severe ventricular dysfunction noted in patients with peripartum cardiomyopathy results in blood stasis and in turn may predispose to the formation of ventricular thrombi. Embolization to the pulmonary artery, peripheral artery, coronary arteries, splenic and renal artery has been reported<sup>8</sup>. Embolic potential of fresh, protruding and mobile thrombi is relatively high<sup>9</sup>. In the present case, the biventricular thrombi appeared to be shaggy, irregular and not highly echoic, nonlaminar, and were freely mobile, suggesting that they were fresh.

In patients with CHF caused by either dilated cardiomyopathy or ischemic heart disease, there is a cardio-embolic risk of 1.5-4.5%/year, with the highest risk related to very low EF and severe clinical heart failure<sup>10</sup>. However, there are no major randomized

studies demonstrating the beneficial effect of vitamin K antagonists in these patients<sup>10</sup>. In accordance with current ACC/AHA guidelines<sup>11</sup>, patients with LV thrombus noted on an imaging study following ST-elevation MI should receive warfarin for at least 3 months, and indefinitely if no increased risk of bleeding exist. Although, it has been recommended that anticoagulants should be continued as long as the cardiomegaly persists in peripartum cardiomyopathy patients<sup>1</sup>, no consensus exists as to whether anticoagulants should be continued after the thrombus disappears.

In the present study, we preferred anticoagulation as the therapeutic choice and due to regression of thrombi within one day of heparin treatment, we continued with oral anticoagulation.

In conclusion, although rare, peripartum cardiomyopathy may be associated with ventricular thrombi and anticoagulation is a reasonable therapeutic option in this situation.

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