

## **PERIPARTUM CARDIOMYOPATHY WITH MULTIPLE ARTERIAL THROMBOSIS; A CASE STUDY OF A 26 YEAR OLD FEMALE**

**Dr. Shivendrran M. S., Dr. Jassim and \*Dr. Dolly Mathew**

Professor of Cardiology Government Medical College, Calicut, Kozhikode, Kerla.

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**Corresponding Author: Dr. Dolly Mathew**

Professor of Cardiology Government Medical College, Calicut, Kozhikode, Kerla.

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### **ABSTRACT**

A 26 years Female 5months postpartum, admitted with Left ventricular systolic dysfunction. Diagnosed with peripartum cardiomyopathy with LV thrombus. Patient had feeble carotid pulse, which on evaluation showed Left common and Left internal carotid artery occlusion. Patients was started on heart failure medications along with anticoagulation. Her symptoms gradually improved and currently under follow up.

**KEYWORDS:** Peripartum cardiomyopathy; LV apical thrombus; Carotid thrombus.

### **1) INTRODUCTION**

Peripartum cardiomyopathy (PPCM) is a rare, idiopathic, and often dilated cardiomyopathy that is marked by systolic dysfunction that presents in late pregnancy or the early postpartum period.<sup>[1]</sup> The rationale for excluding women with heart failure before the final month of pregnancy was to avoid misclassifying pre-existing cardiomyopathies, which typically become symptomatic earlier in pregnancy.<sup>[2]</sup> In 2010 the European Society of Cardiology (ESC) defined peripartum cardiomyopathy as heart failure that occurs “towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found.”<sup>[3]</sup> Clinicians must remain vigilant for a diagnosis of PPCM because its symptoms overlap with those of normal pregnancy and it may be missed on initial evaluation. PPCM is associated with higher rates of thromboembolism compared to other forms of cardiomyopathy.<sup>[4]</sup> Multiple factors contribute to the hypercoagulable state in pregnancy, including cardiac dilation, endothelial injury, increased clotting factor production, and immobility.<sup>[5]</sup> It is important to note that thromboembolism in PPCM is not always limited to the cardiac ventricles, but has also been reported in the cerebral vasculature, coronary arteries, and splenic arteries.<sup>[6][7][8]</sup> Our case of Peripartum cardiomyopathy presented with LV apical clot along with thrombosed left common carotid artery and left internal carotid artery.

### **2) CASE PRESENTATION**

26 years old female 5 months Postpartum came with complaints of dyspnea NYHA FC-II for the past 3 months. She had Paroxysmal Nocturnal dyspnea and on examination features suggestive of Congestive cardiac failure like pedal

edema and elevated JVP with her apex shifted down and out was present. Her left carotid pulse was also feeble. ECG taken showed non specific t wave inversion in V3-v6. Chest Xray showed Cardiomegaly[Figure 1]. ECHO showed global LV hypokinesia with severe LV dysfunction and an apical LV clot [Figure 2]. As her carotid pulse were feeble we proceeded with carotid doppler which showed completely thrombosed left common carotid artery. CT aortogram with arch vessels showed absent contrast opacification of left common carotid artery and left internal carotid artery [Figure 3]. ANA profile, Serum Homocysteine and other coagulation workup was done which was normal except for mild decrease in Protein C. Cardiac MRI was taken which showed irregular patches of myocardial thinning, post contrast enhancement, left ventricular dilatation and minimal pericardial effusion. Patient was hence diagnosed with Peripartum cardiomyopathy with LV apical thrombus and common carotid and internal carotid thrombus. She was started on Heart failure medications along with warfarin. Patient is currently under follow up and improved with treatment. LV apical thrombus has reduced in size and her carotid pulse improved. She is under follow up and her LV function has considerable improvement.



Figure 1: Cardiomegaly.

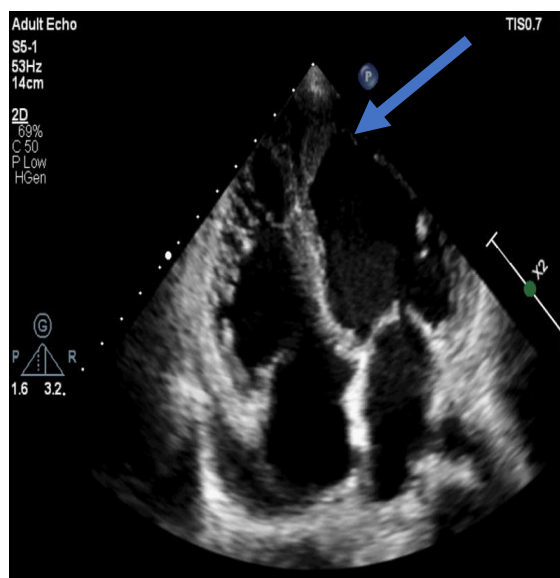


Figure 2: LV apical clot (Marker).

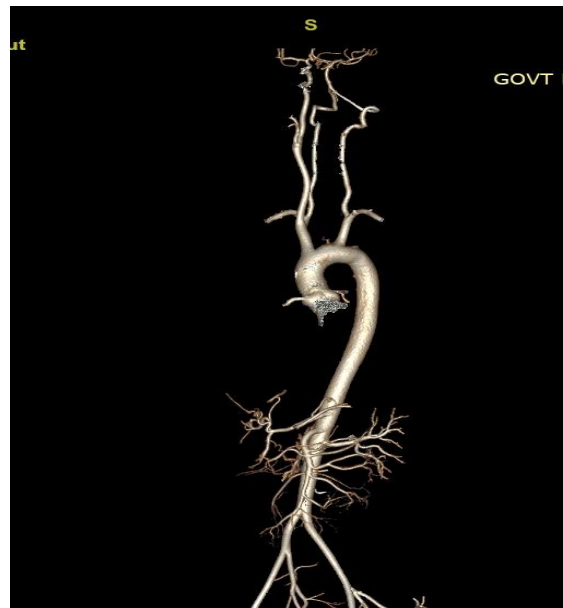


Figure 3.

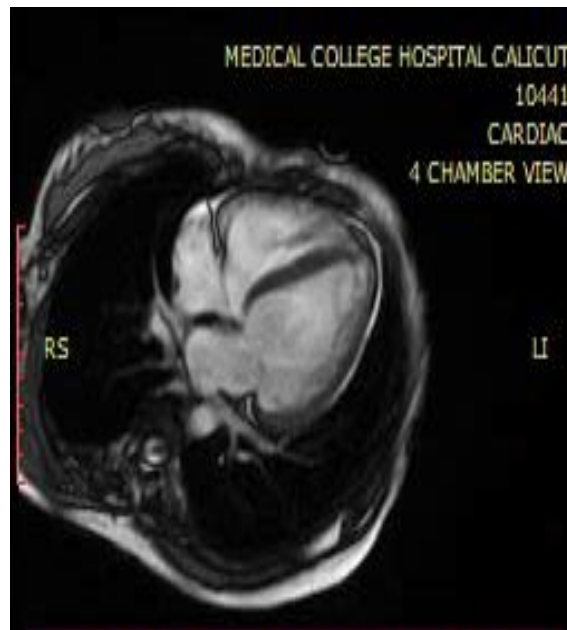


Figure 4.

### 3) DISCUSSION

Pathogenesis of Peripartum cardiomyopathy involves “two hit” model, whereby a vascular insult caused by antivasular or hormonal effects of late pregnancy and the early postpartum period induces cardiomyopathy in women with an underlying genetic predisposition.

Women with PPCM typically present with symptoms of congestion, including dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, and edema of the lower extremities. Less commonly, women present with cardiogenic shock that requires inotropic or mechanical circulatory support. Uncommon presentations include symptomatic or even unstable arrhythmias and arterial thromboembolism.<sup>[9][10]</sup>

Thromboembolism seems to be the most common severe complication of PPCM, affecting 6.6% of women with PPCM in western world; Thrombosis may occur in both left sided and right sided cardiac chambers. Mechanisms underlying intracardiac thrombosis in PPCM include cardiac dilatation and hypocontractability leading to blood stasis, as well as endothelial injury. In addition, pregnancy is a hypercoagulable state secondary to increased levels of factors VII, VIII, X, fibrinogen, and von Willebrand factor; decreased protein C and S activity; and decreased fibrinolysis—changes that normalize by six to eight weeks postpartum.<sup>[11][12][13]</sup>

Management recommendations are generally extrapolated from other forms of heart failure with reduced ejection fraction or derived from expert opinion (or a combination of both). Guidelines advise that standard treatments for heart failure with reduced ejection fraction are indicated in PPCM, with special attention to avoiding adverse fetal effects in women who are still pregnant. Levosimendan is a calcium sensitizing agent rather than a catecholamine and may thus lead to superior outcomes when used for inotropic support in PPCM. Drugs like Bromocriptine also have yielded good results but care to be taken to add anticoagulation as they have high risk of thrombotic events.<sup>[14]</sup>

Thromboembolism is a relatively common complication of PPCM. No published data are available to suggest which women with PPCM warrant anticoagulation.<sup>[15]</sup> However, ESC guidelines advise anticoagulation in patients with PPCM and LVEF  $\leq 35\%$  and in those who have received bromocriptine.<sup>[16]</sup>

#### 4) CONCLUSION

Peripartum cardiomyopathy is usually characterized by LV dysfunction and LV thrombus. Meticulous clinical examination and Investigation may provide presence of thrombus elsewhere.

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