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CASE REPORT

Left Hemiplegia: An Unusual Presentation of Peripartum Cardiomyopathy (PPCM)

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Introduction

PPCM is a rare form of heart failure of unknown cause with a reported incidence of 1 per 4,000 live births. Onset is usually between the last month of pregnancy and up to fifth month postpartum in previously healthy women, with a reported fatality rate of 20–25 %. Risk factors for PPCM include multiparity, older maternal age, black color, pre-eclampsia and twin pregnancies. Its aetiology remains unknown though viral, autoimmune and idiopathic factors may be contributory. 75 % cases present first month post-partum which suggests an autoimmune cause rather than the pregnancy exacerbating a pre-existing cardiomyopathy. PPCM usually presents initially with signs and symptoms of heart failure and rarely with thromboembolic complications. Left ventricle mural thrombus common after acute myocardial infarction but rare in PPCM. The management of PPCM is similar to that of any other forms of dilated cardiomyopathy. True incidence of thromboembolism in PPCM is not known which needs further research.

We report an unusual case of PPCM in a previously healthy woman who presented with features of heart failure and left hemiplegia due to thromboembolism.

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Case Report

34 years old lady, G_5 P_3 A_1 presented at 33 weeks of gestation with watery discharge per vagina for 2 h on 31-10-09. Her LMP was 08-03-09 and EDD was 15-12-09. At the time of admission her blood pressure was 150/110 mmHg, bilateral pitting edema was present. She delivered a live 1.3 kg female baby within 2 h of hospitalization with APGAR score of more than seven. Pre-term baby was taken to nursery for further management. Routine investigations of the mother were as follows—Hb-13.7 gm%, WBC-9,600/cu mm, N-60 %, L-34 %, M-2 %, E 4 % platelet count-120,000/cu mm, LFT-normal, total serum protein 4.05 gm %, serum albumin 1.9 gm %, urine–albumin 2+. She was treated with nifedipine, diuretics, antibiotics and FFP. She improved and was discharged on 16-11-09. She was readmitted on 23-11-09 with features of congestive heart failure. At the time of admission her JVP was raised, pulse rate 90/min, BP-130/mmHg, S3 gallop was present, bibasal crepts and bilateral pedal oedema were present. She was treated with digoxin, diuretics, angiotensin converting enzyme inhibitors (ACEIs) and oxygen.

Next morning she developed left hemiplegia with grade 2 power. ECG showed normal sinus rhythm and non specific T-wave changes X-ray chest revealed cardiomegaly and pulmonary edema. Trans thoracic echocardiography showed global left ventricular hypokinesia, LV-ejection fraction was 28 %, left ventricular mural thrombus attached to the lower portion of inter ventricular septum and apex. CT brain revealed acute infarct in right basal

ganglia and corona radiata. She was treated with low molecular weight heparin, ACEIs, diuretics and digoxin. Once features of heart failure subsided, a beta blocker, carvedilol was added. She responded to medical therapy very well and regained normal power within 15 days. Follow up echocardiography done after 4 weeks showed complete disappearance of left ventricular thrombus and ejection fraction improved from 28 to 31 %. Both mother and baby were doing well at the time of last check up.

Discussion

Peripartum cardiomyopathy is a disorder of unknown cause in which left ventricular dysfunction and symptoms of heart failure starts between the last month of pregnancy and first 5 months postpartum in the absence of preexisting heart disease. PPCM is a rare form of dilated cardiomyopathy that is associated with high maternal morbidity and mortality [1]. Risk factors for the development of PPCM include older maternal age, black, multigravida, preeclampsia and twin pregnancy [2]. Our patient is a multigravida and had features of pre-eclampsia in the form of hypertension, oedema and proteinuria. Although the underlying cause of the condition is unknown, several endomyocardial biopsy studies have revealed myocarditis [3].

PPCM initially presents with signs and symptoms of heart failure and rarely with thromboembolic complications [4]. Our case presented with features of both heart failure and thromboembolism.

The occurrence of thromboembolism in PPCM may be due to the hypercoagulable state of pregnancy and the left ventricular dysfunction or fibrillating left atrium which causes relative blood stasis. Left ventricular thrombus is common in PPCM patients with a left ventricle ejection fraction of less than 35 %. Atrial thrombus occurs almost always in patients with atrial fibrillation. Although a mural thrombus adheres to the endocardium, superficial portion of it can become detached and produce systemic arterial embolisation to any part of the body, including arterial occlusion of lower extremities, cerebral embolism and mesenteric artery occlusion [5]. In the present case, she was in sinus rhythm, ejection fraction was 28 % and had cerebral embolism due to left ventricular thrombus.

The medical management of patients with PPCM is similar to that for other forms of heart failure and has been reviewed in detail [6]. Treatment aims to reduce afterload and preload, and to increase contractility. ACEIs are usually used to reduce afterload by vasodilatation if PPCM occurs after delivery as happened in our case. Because of potential toxic effects on the fetus, hydralazine replaces ACEIs during pregnancy. Beta blockers are used since high heart rate, arrhythmias, sudden death often occurs in patients with PPCM. Digitalis, an inotropic agent, is also safe during pregnancy and may help to maximize contractility and rate control. Diuretics are also safe and are used to reduce preload and relieve symptoms. Because of the high incidence of thromboembolism in these patients, the current concept is to initiate anticoagulation therapy in the presence of left ventricular dysfunction with ejection fraction less than 35 % [7]. We treated our case with digoxin, diuretics, ACEIs and anticoagulation with good clinical outcome. Beta blockers were added after signs and symptoms of congestive heart failure have improved. Since no one knows for sure exactly when to discontinue treatment even when recovery occurs quickly, it is still recommended that both ACEIs and beta blockers be continued for at least 1 year after diagnosis [5].

Early diagnosis and treatment of PPCM are essential for a favorable outcome. Poor prognostic factors include high parity, twin gestation, age greater than 30 years, preeclampsia and a late onset of symptoms after delivery. Our present case is multiparous, age more than 30 years, preeclamptic, and her clinical presentation of heart failure and thromboembolism occurred 3 weeks postpartum. Clinicians should think of PPCM in any peripartum patient with unexplained breathlessness. Any woman in peripartum period complaining of breathlessness and with features of preeclampsia should have an echocardiographic evaluation to detect PPCM. A high index of suspicion, cardiomegaly, a severe left ventricular dysfunction and a left ventricular mural thrombus prompted accurate diagnosis and a successful therapy.

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