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Never Let Failure Get Your Heart-A Case of Successfully Managed Peripartum Cardiomyopathy

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Case report

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Abstract

Peripartum Cardiomyopathy (PPCM) is a form of idiopathic heart failure that develops most commonly in the last month of gestation or during the first five months post-partum. Here we present a case of successfully managed peripartum cardiomyopathy in a patient with preeclampsia with severe features with pulmonary oedema and intrauterine death of foetus. The patient was managed conservatively until stable along the lines of pulmonary oedema and preeclampsia with severe features keeping various other differentials in mind followed by which the diagnosis of peripartum cardiomyopathy was suggested by echocardiography. Termination of pregnancy was conducted by caesarean section following which both mother and baby were discharged in stable conditions.

Keywords: Post-Partum; Cardiomyopathy; Pre-Conceptional

Abbreviations: PPCM: Peripartum Cardiomyopathy; HDU: High Dependency Unit.

Introduction

The incidence of heart diseases in pregnancies varies widely across the globe, with an estimate of about 0.46 per 1000 deliveries, out of which 0.18/1000 for apparent Peripartum cardiomyopathy (PPCM) and 0.28/1000 for other cardiomyopathies with a case fatality rate of about 5.1%. The incidence is approximately 1 in every 2657 pregnancies in Asians [1]. PPCM is rarely seen and is characterised by an acute onset of heart failure within one month before delivery or five months postpartum [2]. It can be idiopathic or caused by various aetiologies and causes increased morbidity and mortality in pregnant patients, leaving most patients with residual disease. Here we report a case of a 27-year-old female with intrauterine death of foetus having peripartum cardiomyopathy along with preeclampsia and her subsequent course.

Case Report

A 27-year-old, gravida 4, para 1, live 1, abortion 1 at 32 weeks gestation, was admitted to the High Dependency Unit (HDU) in labour room, with complaints of shortness of breath for a few hours and absent foetal movements. In her present pregnancy, she gave no history of prior elevated blood pressure records and reported no comorbidities along with all normal investigations and ultrasounds. On examination, her pulse was regular, with a rate of 112 beats/minute, and her blood pressure was 146/128 mmHg. Her respiratory rate

was 40 breaths per minute, and peripheral oxygen saturation was 80 per cent on room air. On further examination, mild pallor, pedal oedema and abdominal wall oedema were present. The patient also had coarse crepitations in the chest bilarerally with normal heart sounds and no appreciable murmurs. She had a history of preeclampsia with severe features in her previous pregnancy four years back, which was terminated at full term by caesarean section with an uneventful post-partum period. On admission, she had microalbuminuria with 300 mg/mmol of urine albumin. Her haemoglobin was 15.3 g/dl, and her platelet count was 207,000/ml. Her liver and renal function tests were normal, with elevated potassium levels. As part of initial stabilisation, she was put on oxygen by venturi mask. To relieve pulmonary oedema, blood pressure was controlled by tablet nifedipine 10 mg and intravenous furosemide.

Foetal intrauterine death was confirmed by ultrasound. Arterial blood gas analysis showed metabolic acidosis. Her electrocardiogram was suggestive of t-wave inversion in leads one and aVL. Chest X-ray showed blunting of bilateral cardio phrenic angles and haziness in middle and lower lung zones and cardiomegaly. Her CPKMB was elevated (55 IU/L), but her d-dimer was within normal pregnancy limits (2492 mcg/ml). Cardiopulmonary consultations were taken, and the patient was started on tablet carvedilol 3.125mg. Metabolic correction was given for hyperkalaemia and metabolic acidosis. 2 D ECHO suggested left ventricular systolic dysfunction with an ejection fraction of 20-25% with no valvular abnormalities, representing a possibility of cardiomyopathy.

A combined multidisciplinary team of obstetricians, cardiologists, and anaesthesiologists planned for maternal stabilisation followed by termination of pregnancy. The patient was started on dobutamine infusion to improve the contractility and injection of furosemide to decrease the cardiac preload. Considering all the risks versus benefits, it was planned to terminate the pregnancy by elective caesarean section under general anaesthesia. The caesarean was uneventful, delivering a stillborn girl weighing 1400 grams. The precarious period being postpartum, the patient was monitored strictly in the cardiac unit. We started her on tablet ramipril 1.25mg, ivabradine 2.5 mg, and a diuretic in the postoperative period. She also received thromboprophylaxis with low molecular weight heparin and tablet cabergoline.

The patient progressively improved in the post-partum period with echocardiography on postoperative day 4, suggesting an improvement in left ventricular ejection fraction to 45-50%. She was discharged in stable condition after appropriate post-partum counselling regarding contraception, chances of recurrence in subsequent pregnancy and regular follow-up in the cardiology unit.

Discussion

Peripartum Cardiomyopathy (PPCM) is a form of idiopathic heart failure that develops during gestation and is characterised by four criteria:

- Development in the last month of gestation or during the first five months post-partum.
- No other causes of heart failure.
- No history of heart disease before gestation.
- Left ventricular dysfunction on echocardiography.
- Including a left ventricular ejection fraction of <45% and fractional shortening of <30% [3].

Any patient presenting with symptoms of acute heart failure at the end of pregnancy or in the post-partum period should be evaluated thoroughly, considering the various differential diagnoses. Conditions such as myocarditis, arrhythmogenic ventricular dysplasia, pre-existing cardiomyopathies, amniotic fluid embolism, aortic spasm, pulmonary embolism, pulmonary oedema associated with excess tocolytics, etc., can present similarly and must be evaluated. Also, pulmonary oedema related to preeclampsia and eclampsia can lead to confusion in diagnosis, especially given its intense association of about 22% with PPCM [4]. Various investigations in the usual panel for evaluation can cause further dilemmas as these differentials have a similar presentation and pathophysiology. Although echocardiography is valuable and can provide us with immediate confirmation of a diagnosis of peripartum cardiomyopathy, the other differentials should still be kept in mind. Furthermore, the diagnosis should constantly be re-evaluated for differentials after initial management according to the response to treatment [5].

The treatment of PPCM is done like any other case of heart failure, viz. oxygen administration for hypoxia, intravenous diuretics and vasodilators to reduce preload and afterload, ionotropic therapy along with other supportive measures such as extracorporeal membrane oxygenation. Novel agents such as levosimendan are also being tried to manage PPCM [6]. After initial patient stabilisation and resolution of pulmonary oedema, the patient can be started on beta blockers and ivabradine for rate control and angiotensin-converting enzyme inhibitors to decrease cardiac afterload.

Mode of delivery in Peripartum cardiomyopathy should be tailor-made according to individual patients considering the risk versus benefit ratio, and Interdepartmental coordination is of utmost requirement within high facility tertiary setup. Termination of pregnancy is usually done as per obstetric indications, with vaginal delivery being the preferred mode. However, vaginal delivery should only be conducted after considering the increase in cardiac output above baseline (up to 60%) during the various stages of

labour. Thus, in patients with severe heart failure, caesarean section is considered. PPCM usually requires an early delivery to reduce the stress on maternal metabolism and for foetal well-being. But it should also be kept in mind that for patients who are not deteriorating, early delivery may not significantly reduce the overall risk of the patient and instead increase neonatal morbidity on accounts of prematurity [7].

Recently, the role of Bromocriptine, in addition to standard therapy for improving LVEF at six months follow-up, has been studied extensively given the possibility of involvement of prolactin hormone in etiology [8]. Also, prophylactic thromboprophylaxis with low molecular weight heparin is beneficial in cases of LVEF <35% as both peripartum cardiomyopathy and heart failure have the risk of thromboembolic complications [9].

Patients with PPCM also require adequate post-partum and pre-conceptional counselling as this condition is likely to have residual dysfunction and a possibility of recurrence in subsequent pregnancies. A prospective study conducted in Germany, Scotland and South Africa showed persistently reduced left ventricular function in about 47% of patients studied, and only 53% recovered entirely before subsequent pregnancy [8]. Early contraceptive counselling should be done considering the various risks associated with different contraceptives in women with heart disease. Prothrombotic hormonal contraceptives should be avoided as these women are already at a higher risk of thromboembolic events. Roos-Hesselink and colleagues have recently published a comprehensive review on the use of contraception in this women [10].

Patients should be counselled about the need for strict follow-up at a tertiary care institute, including an annual follow-up at a cardiology setup with echocardiography screening at regular intervals. Overall, such patients require a multidisciplinary approach to optimise outcomes for the mother and child.

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