Case Report

Peripartum cardiomyopathy in an elderly woman: A case report

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Abstract

Cardiomyopathy accounts for 11.5% cause of pregnancy-related deaths. The causes of peripartum cardiomyopathy (PPCM) are unknown, including predisposing factors such as abnormal response to the greater hemodynamic burden of pregnancy, viral myocarditis, malnutrition, inflammation, and apoptosis. Elderly women, twin gestation, preeclampsia, smoking, and anemia are potential high-risk factors for PPCM. Here, rare case of a patient is described that presented with complaints of breathlessness, cough, and symptoms of heart failure and was diagnosed early as PPCM based on 2D echocardiography and was managed with medical therapy and timely delivery of fetus. Symptomatic relief of symptoms was relieved by diuretics, inotropic drugs, and beta-blockers. The left ventricular ejection fraction increased eventually over 3 weeks and now the patient is a follow-up case since two years from the time of episode. In conclusion, PPCM is rare, which requires prompt management and heightened mindfulness; therefore, early detection and timely treatment can reduce maternal mortality.

Keywords: Echocardiography, left ventricular ejection fraction, lower segment caesarean section, peripartum cardiomyopathy, pregnancy

Introduction

A rare but potentially fatal condition, peripartum cardiomyopathy (PPCM) affects women in the final month of pregnancy or the first five months following delivery. It can occur in 1 in 1300 to 1 in 15,000 pregnancies, depending on the individual case.[1] Pre-eclampsia, numerous pregnancies, multiparity, advanced maternal age, chronic hypertension, smoking, alcoholism, malnutrition, and long-term tocolysis are risk factors of PPCM.[1-3] PPCM is determined by four criteria: the onset of heart failure in the final month of pregnancy or five months after delivery, the absence of a known cause for the cardiac failure, and the absence of heart disease before the final month of pregnancy. Additionally, M-mode fractional shortening ≤30%, left ventricular (LV), end-diastolic dimension ≥2.7 cm/m², and LV ejection fraction (EF) ≤0.45 are suggestive of PPCM.

PPCM has a number of potential causes, including myocarditis, an unusual immunological reaction to pregnancy, an unsuitable reaction to pregnancy hemodynamic strains, extended tocolysis, and cytokinins activated due to stress. The fact that there have been a few examples of familial PPCM raises the possibility that some cases of PPCM are indeed familial dilated cardiomyopathy concealed by pregnancy.[4-6] Fatigue, edema, and dyspnea are among the signs of PPCM, which are also present typically during pregnancy period and complications in pregnancy such as eclampsia and pulmonary emboli.[7]

Echocardiogram (ECG), chest radiography, and imaging studies in general add up to diagnostic evaluation. ECG results can

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include voltage anomalies, sinus tachycardia, and generalized ST- and T-wave abnormalities even though they are frequently normal.[8] Chest radiographs may infrequently reveal evidence of pleural effusions, heart enlargement, or even pulmonary congestion.[7] LV dilatation without hypertrophy and reduced contractility are frequently seen on ECG.[9]

The treatment for PPCM is the same as for other types of congestive heart failure (CHF), with the exception of angiotensin-converting enzyme inhibitors and angiotensin–receptor blockers, which are contraindicated during pregnancy. To avoid dehydration as well as placental insufficiency during pregnancy, diuretics (DU) can be taken with caution. Anticoagulants (AC) should be taken into consideration, particularly in high-risk patients with significant LV dysfunction, as there is an increased risk of thrombus formation in PPCM patients. The EF returns to normal in roughly half of the cases. It is normally not recommended for these people to get pregnant again irrespective of recovery due to the high danger that both mother and baby face when more than 30% of subsequent pregnancies develop PPCM.[9,10] Hence, the main aim of the primary care physicians is to reduce symptoms that will consequently result into control of PPCM.

Case Presentation

A 30-year-old woman, housewife, married for 9 years G4P2L1A1D1 with prior Caesarean section (LSCS) scar in the lower (uterine) segment with gestational age 26 + 2 weeks presented to the emergency with chief complaints of generalized swelling over the face, upper limb, and lower limbs since 15 days, breathlessness, and decreased appetite since 6 days, dry cough since 3 days, chest pain with palpitations since 2 days, nausea, and loose stools since two days.

On inspection, the general condition was moderate and presented afebrile orthopnea along with generalized edema. The vital parameters demonstrated blood pressure of 110/70 mm of Hg, respiratory rate of 36 breaths per minute associated with tachycardia having pulse rate of 110 beats per minute (bpm). On auscultation, S1 and S2 heart sounds were heard normally, but there was systolic murmur present along with bilateral crepitations all over the lungs. There were no premonitory signs and symptoms and deep tendon reflexes were normal. Per abdominal her uterus was 24 weeks, which was well relaxed, LSCS scar mark was present with no scar tenderness. Fetal heart sounds could be heard with 140 bpm.

On investigation, kidney function test was deranged with urea having value of 55 mg/dl and urine albumin of 3+. The parameters of complete blood count and liver function tests were within normal limits. The patient was shifted to intensive care unit. Diagnostic assessment included ECG that was suggestive of T wave inversion [Figure 1] in 3rd lead along with chest X-ray that demonstrated pulmonary edema [Figure 2].

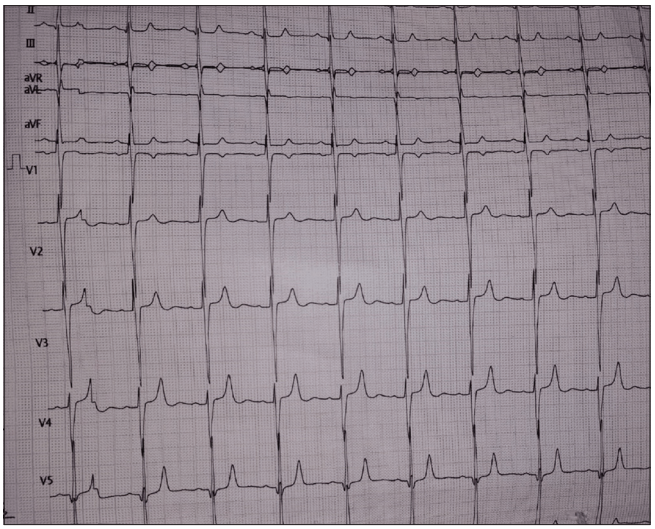

Figure 1: Echocardiography demonstrating T-wave inversion

Additionally, 2D ECG was performed, which showed diagnostic features of cardiomyopathy with global hypokinesia and dilated LV with a 20-25% ejection fraction. There was also grade 3 diastolic dysfunction and LV apical clot was evident. The general condition of the patient was deteriorating with increasing respiratory distress and progressing heart failure. The patient was hence taken for emergency LSCS, following which male baby of 540 g was delivered, the baby cried after birth but died 2 hours later.

Before commencing the treatment, written informed consent was obtained from the patient and emergent medical management was started with Tab Carvedilol 3.125 mg half twice a day and injection Heparin 5000 IU thrice a day, injection Torsemide 20 mg twice a day. The 2D ECG was repeated on day 3 of LCSC, which showed evident pericardial effusion, but left ventricular ejection fraction was increased to 56% and there was no left ventricular apical clot. On day 6, the 2D ECG showed moderate left ventricular dysfunction and dilated LV with grade 2 diastolic dysfunction. The patient adhered well to the surgical and medical intervention and the symptoms of heart failure improved eventually.

Discussion

This case report highlights a rare case of PPCM with a gestational age of 26 + 2 weeks and described the positive outcome in terms of adherence and effectiveness of the surgical and medical intervention. PPCM diagnosis is based upon ECG findings of systolic impairment of LV in the last month before delivery. Dyspnea, tiredness, and edema of the feet, which are similar to the characteristics of early CHF, are common in the final month of a typical pregnancy.[11] Therefore, PPCM may go unnoticed, resulting in an underestimation of incidence.[1]

The indications of cardiomyopathy mainly involve dyspnea, fatigue, edema, palpitations, and dry cough and palpitations,

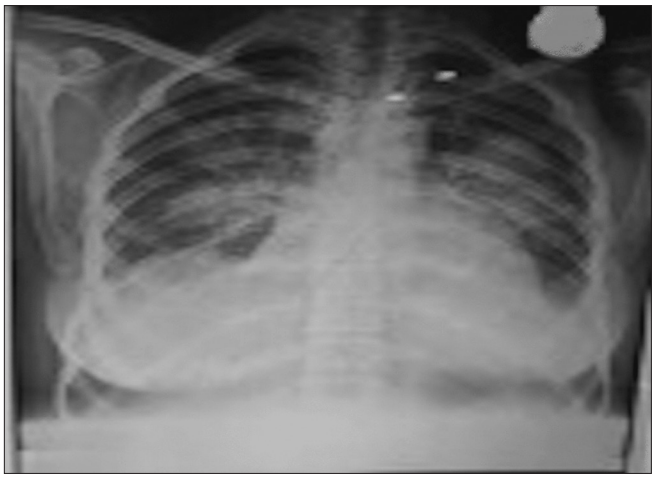

Figure 2: Chest X-ray demonstrating pulmonary oedema

which correspond with the symptoms seen in the present case that included generalized swelling over the face, upper limb, and lower limbs, breathlessness and decreased appetite, dry cough, chest pain with palpitations, nausea, and loose stools.[12]

It is necessary to rule out alternative causes of cardiomyopathy before making the diagnosis of PPCM, which is then supported by a conventional ECG assessment of LV systolic failure, including depressed fractional shortening and EF.[1] Similarly, in this case, the main diagnostic assessment involved a chest X-ray and 2D ECG.

In the absence of thorough investigations comparing therapeutic methods in PPCM, conventional heart failure therapy should be commenced.[13] Following birth, it is crucial to pay close attention to both the safety of the fetus and the excretion of drugs or drug metabolites. There must be cooperation between cardiologists, perinatologists, obstetricians, and neonatologists.[14] To treat pulmonary congestion or peripheral edema, furosemide was prescribed in our case which performs as a diuretic[1] and is safe during pregnancy and lactation. Additionally, for prolonged systolic dysfunction management, carvedilol was administered, which is a β-blocker in which β1-selective blockers are mostly preferred. It has been demonstrated that carvedilol combined with a blocker to reduce peripheral vasoconstriction is efficient in PPCM.[1] Furthermore, heparin was administered for anticoagulation. Cooperation with cardiologists and anesthesiologists should be used to determine whether early delivery is necessary along with the mode of delivery.

In consensus with the aforementioned therapy, salt and water restriction is advised. Once heart failure symptoms are under control, light exercise may improve symptoms in addition to vascular and peripheral muscle tone. Patients should receive appropriate treatment and should be encouraged to adopt a healthy diet that is good for the heart as well as a fine lifestyle, and should be firmly advised to avoid becoming pregnant again if their left ventricular size or function does not return to normal. As a result, if additional pregnancies are unavoidable, they should be coordinated with a high-risk perinatal center for management.[1]

The incidence and prognosis differ according to the geographical variations, and the disease is mainly caused due to a number of variables. This idea shows that the oxidative stress cathepsin D-16 kDa prolactin cascade plays a significant role in the development of PPCM in both experimental animals and PPCM patients.[5] According to a recent theory, bromocriptine-induced blockage of this pathway may be a unique disease-specific therapy strategy for PPCM. The emergence of international and national registries and cooperative research initiatives will substantially aid future research regarding the incidence, pathogenesis, genetics, therapy, and prognosis of patients with PPCM as these investigations are necessary.

Conclusion

PPCM is an uncommon disease that primarily affects females in their reproductive years, has a high fatality rate, and may reoccur. The combination of peripartum physiology with pathogenic, genetic, inflammatory, metabolic or hormonal variables is the focus of theories regarding pathogenesis. The diagnosis of PPCM is difficult and demands attention. The main objective of therapy after PPCM has been detected using the criteria is to reduce congestive heart failure symptoms. The prognosis is anticipated to be good in the short term if the LV size returns to normal after delivery, whereas it is yet unclear how long-term effects, particularly with further pregnancies, may emerge. Overly high rates of morbidity and mortality are linked to the heart's inability to change back to its regular size. Therefore, it is critical for doctors to be knowledgeable about PPCM and to take it into account when making a dyspneic patient's diagnosis to begin treatment as soon as possible for a potentially fatal condition.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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