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(CASE REPORT)



Peripartum cardiomyopathy: Case report

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Abstract

Systolic heart failure which develops in the last month of pregnancy or within five months following childbirth is an important hallmark of peripartum cardiomyopathy (PPCM), a type of dilated cardiomyopathy. Despite being identified in the 1800s, the exact cause of PPCM remains uncertain. The incidence of peripartum cardiomyopathy in India is estimated to be 1 in 453 to 1 in 1340 live births. However, because its symptoms mimic those of a typical pregnancy and postpartum period, diagnosing it can be difficult due to its high mortality rate and PPCM can have serious consequences if it is misdiagnosed or delayed.

In our case report, we present a patient who developed shortness of breath since past 1 week associated with orthopnea after a spontaneous delivery. After careful evaluation, we identified the symptoms as heart failure resulting from peripartum cardiomyopathy. The treatment approach included symptomatic measures employing diuretics, ACE inhibitors and HMG Co A reductase inhibitors, Anti platelets. Fortunately, mechanical cardiac support was not necessary, and the signs of heart failure improved within three weeks. Furthermore, during the follow-up period of one year, the patient's left ventricular clot returned to normal, maintaining a positive state of health.

Keywords: Peripartum; Cardiomyopathy; Heart Failure; Treatment

1. Introduction

Approximately one out of every 453 to 1340 live births in the India results in peripartum cardiomyopathy (PPCM), a infrequent but critical disorder that affects women with in the last months of pregnancy or postpartum. [1] The diagnostic criteria for PPCM consist of four key elements: 1) the onset of cardiac failure during the last month of pregnancy or within five months after childbirth, 2) the absence of any identifiable cause for the cardiac failure, 3) no evidence of pre-existing heart disease before the last month of pregnancy, and 4) Left Ventricular (LV) dysfunction characterized by an ejection fraction below 45% or reduced shortening fraction [2, 3].

PPCM is associated with several risk factors, including multiparity, black race, older maternal age, pre-eclampsia, and gestational hypertension [1, 4]. Fatigue, edema, and dyspnea are some of the symptoms of PPCM that are quite similar to those of other pregnancy-related complications such pulmonary emboli and eclampsia.[5]. Consequently, there are frequently delays in PPCM diagnosis, which results in a lack of awareness of the illness and severe complications, with mortality rates between 20% and 50% [5].

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2. Case presentation

A 26-year-old woman, primipara presented with complaints of difficulty in breathing and abdominal distension, edema noted (anasarca) first started in the feet slowly progressed over 1 week, six months back delivered child. Her medical history did not reveal any significant health issues, and her pregnancy had been uneventful. She mentioned that being a first-time mother, she initially considered her symptoms to be a normal part of the post-delivery experience.

During the examination, the patient was found to have a normal body temperature and a blood pressure of 110/70 mm Hg. Her pulse rate was 90 beats per minute, respiratory rate at 24 breaths per minute, and oxygen saturation was 94% with the assistance of a 2-L nasal cannula. Auscultation of her lungs revealed clear sounds, and her heart rate was regular. There was edema observed in her extremities, and facial puffiness (Anasarca + pitting). Urinalysis results were negative for protein presence. The levels of D-dimer and B-type Natriuretic Peptide (BNP) in her plasma were measured at 2105 pg. /mL and 884 pg./mL, respectively. An electrocardiogram indicated a poor R wave progression low voltage. Chest radiographs displayed cardiomegaly and increased vascular congestion in both lungs. A chest CT scan, conducted to investigate possible emboli, revealed evidence of pericardial effusion and cardiomegaly, left ventricular clot (5.1cm²) detected.

Following the diagnosis of new-onset PPCM, the patient was admitted to the hospital, and intravenous furosemide was administered for diuresis. An initial transthoracic echocardiogram conducted upon admission revealed a heart failure with reduced ejection fraction (HfrEF) of 18% along with left ventricular clot $(5.1~{\rm cm}^2)$. With the effects of diuresis, her symptoms of fatigue and dyspnea significantly improved, ascetic tap procedure was conducted to remove ascetic fluid . Leading to her discharge from the hospital after two weeks.

3. Discussion

During an atypical pregnancy, dyspnea affects about 60% to 70% of women.[6] Older age and Black ethnicity have historically been linked to PPCM risk factors, but current trends show that incidence is rising among young, first-time pregnant women and those of white ethnicity (24%–37%). [7, 8]. Given that the patient is young and pregnant for the first time, the case described in this report fits this new trend. It is difficult to identify PPCM because dyspnea is common in both normal pregnancy and the early postpartum period, particularly when the patient population does not fit the usual epidemiological profile. As early as the 1870s, researchers recognized a potential connection between pregnancy and dilated cardiomyopathy. By the1930s, this association was formally classified as a distinct clinical condition [9, 10]. However, the exact cause of PPCM remains elusive. Several theories have been proposed to explain its origin. One prevailing belief is that PPCM may be linked to the cardiovascular strain experienced during pregnancy, such as increased fluid volume. Alternatively, some experts have suggested that myocarditis could play a role in the development of PPCM. Research conducted byFelker et al. [11], found histological evidence of myocarditis in 26 out of 51 women diagnosed with PPCM based on endomyocardial biopsy.

There is also a theory that suggests PPCM could be an inflammatory reaction brought on by pregnancy. In certain instances, increased levels of interleukin-6 and tumor necrosis factor-alpha support this idea. [12, 13]. PPCM may also be an aberrant autoimmune reaction to fetal cells found in the mother's heart and bloodstream, according to some researchers. In addition, there is ongoing discussion regarding the potential contribution of nutritional deficiencies—more especially, selenium deficiency—to the development of PPCM, with conflicting findings from various reports. [14, 15].

Chest pain and symptoms of congestive heart failure are among the clinical characteristics that define PPCM. Signs that can be identified include pulmonary rales, tachycardia, tachypnea, an enlarged heart, and an S3 heart sound. [4]. These symptoms frequently resemble those of other illnesses, from upper respiratory infections and pulmonary emboli to typical pregnancy-related symptoms. Because of this overlap in their signs and symptoms, it can be difficult to differentiate PPCM from these other conditions

The four criteria listed at the beginning of this report are used to diagnose PPCM. Elevated B-type Natriuretic Peptide (BNP) levels are frequently seen, but there are no particular laboratory abnormalities that are unique to PPCM. Nonetheless, it is crucial to take into account additional laboratory tests for exclusionary purposes, such as measuring cardiac enzymes and performing a preeclampsia workup. Imaging studies are essential for PPCM diagnosis. The results of electrocardiography (ECG) are usually normal, but they can show abnormalities of the ST and T waves, voltage changes, and inustachycardia.[16] Signs of pulmonary congestion can be seen on chest radiographs. Enlarged heart and

occasionally pleural effusions [8] Echocardiograms are useful for demonstrating decreased contractility and LV enlargement without hypertrophy.[17]

4. Treatment

Like other forms of congestive heart failure, PPCM is treated in a similar manner. It involves actions such as restricting salt and fluid intake, administering digoxin, β -blockers, and diuretics. However, because they are contraindicated during pregnancy, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are not advised. [1] During pregnancy, hydralazine can be administered safely to lower afterload. Diuretics should be used carefully during pregnancy to avoid placental insufficiency and dehydration. Patients with PPCM have a higher chance of thrombus development, or blood clots. [18]

Anticoagulation medication should thus be taken into consideration, particularly for high-risk patients who have significant left ventricular dysfunction. Promoting physical activity is also crucial, but it should be customized for each patient based on their tolerance level, symptoms, and general health. Though it's unclear how long is the best time to stop taking these drugs, it's usually recommended to keep taking them for at least a year. [7]. As an alternative, heart transplantation may be considered if medical treatments are unsuccessful. Thankfully, the proportion of people in need of transplants has decreased recently to between 4% and 7% [19]. Heart transplants have demonstrated encouraging success rates and good long-term survival results. The ejection fraction returns to normal in almost half of the individuals. The high recurrence rate of PPCM, which exceeds 30% in subsequent pregnancies and poses serious dangers to both the mother and the unborn child, often discourages most patients from being pregnant again, even if they recover.[20]

5. Conclusion

Per partum cardiomyopathy is a very uncommon condition that affects women towards the end of pregnancy or in the months after delivery. It is a difficult and complicated disorder to treat because its precise origin is yet unknown and it might occasionally return. Because of the significantly high death rate linked to PPCM, prompt and precise diagnosis is necessary. Because PPCM's symptoms might overlap with those of other illnesses, diagnosing it requires considerable attention and monitoring from healthcare providers. The main goal of treatment is to reduce congestive heart failure symptoms when PPCM has been recognized using predetermined criteria.

If the left ventricular size recovers to normal following pregnancy, the prognosis is likely to be favorable in the near term. However, further research is necessary to completely understand the long-term effects, particularly in relation to subsequent pregnancies. However, failure of the heart size to return to normal is associated with higher rates of morbidity and death, highlighting the importance of timely and efficient treatment. To fully comprehend PPCM and its possible implications, especially in light of future pregnancies, more study is necessary.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of informed consent

Informed consent was obtained from individual participants included in the study.

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