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Case Report

Peripartum Cardiomyopathy: Case Report

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Abstract

Peripartum Cardiomyopathy (PPCM) is a form of dilated cardiomyopathy characterized by systolic heart failure occurring in the final month of pregnancy or within five months after childbirth. Despite being identified in the 1800s, the exact cause of PPCM remains uncertain. This condition affects numerous women annually in the United States. However, its diagnosis is often challenging due to its symptoms overlapping with normal pregnancy and postpartum experiences. Delays or misdiagnosis of PPCM can lead to severe consequences as it carries a high mortality rate.

In our case report, we present a patient who developed shortness of breath six days 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, inotropes and beta-blockers. 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 ejection fraction returned to normal, maintaining a positive state of health.

This article focuses on the diagnostic and treatment strategies for peripartum cardiomyopathy, shedding light on this condition's challenging nature and the importance of timely and accurate intervention.

Keywords: Peripartum; Cardiomyopathy; Heart failure; Pirimapara

Introduction

Peripartum Cardiomyopathy (PPCM) is a condition that impacts a significant number of women in the US each year, occurring in approximately one out of every 3000 to 4000 live births [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]. The symptoms of PPCM, such as fatigue, edema, and dyspnea, closely resemble those seen in normal peripartum conditions and other pregnancy-related complications like pulmonary emboli and eclampsia [5]. As a result, the diagnosis of PPCM often encounters delays, leading to under-recognition of the disorder and severe consequences, with mortality rates ranging from 20% to 50% [5].

Case Presentation

A 21-year-old woman, primipara presented with complaints of dyspnea and chest discomfort, just six days after giving birth. 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 96/64 mm Hg. Her pulse rate was 94 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 with an S3 gallop. There was no edema observed in her extremities, and she did not exhibit any calf tenderness. 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 normal sinus rhythm. Chest radiographs displayed cardiomegaly and increased vascular congestion in both lungs. A chest CT scan, conducted to investigate possible pulmonary emboli, revealed evidence of pleural effusion and cardiomegaly, but no signs of emboli were 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 left ventricular ejection fraction (LVEF) of 40% along with minimal aortic and mitral regurgitation. With the effects of diuresis, her symptoms of fatigue and dyspnea significantly improved, leading to her discharge from the hospital after three days. Upon a six-month follow-up examination, the patient's cardiomyopathy remained stable, and a repeat echocardiogram demonstrated a notable improvement in the ejection fraction, which had risen to a range of 55% to 60%.

Discussion

Roughly 60% to 70% of women experience dyspnea during a typical pregnancy [6]. While historically, PPCM risk factors have been associated with older age and black ethnicity, recent trends indicate a growing incidence (24%-37%) among young first-time pregnant women and those of white ethnicity [7,8]. This case presented in this report align with this emerging pattern, as patient is young, first-time pregnant women of black ethnicity. The prevalence of dyspnea

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in regular pregnancy and the early postpartum period makes it challenging to detect PPCM, especially when the patient population does not match the typical epidemiological profile.

As early as the 1870s, researchers recognized a potential connection between pregnancy and dilated cardiomyopathy. By the 1930s, 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 by Felker et al. [11], found histological evidence of myocarditis in 26 out of 51 women diagnosed with PPCM based on endomyocardial biopsy.

In addition, there is a hypothesis that PPCM might be an inflammatory response triggered by pregnancy. This notion is supported by elevated levels of tumor necrosis factor-alpha and interleukin-6 in some cases [12,13]. Some researchers have also put forth the idea that PPCM could be an abnormal autoimmune response to fetal cells present in the maternal circulation and cardiac tissue. On another note, the role of nutritional deficiencies, specifically selenium deficiency, in the onset of PPCM remains debated, with conflicting evidence from different studies [14,15].

PPCM is characterized by clinical features that encompass congestive heart failure symptoms and chest pain. Detectable signs may consist of tachycardia, tachypnea, pulmonary rales, an enlarged heart, and an S3 heart sound [4]. These manifestations often share similarities with various other conditions, ranging from regular pregnancy-related symptoms to pulmonary emboli and upper respiratory infections. As a result, distinguishing PPCM from these other conditions can be challenging due to the overlapping nature of their signs and symptoms.

The diagnosis of PPCM is based on the four criteria mentioned in the beginning of this report. While there are no specific laboratory abnormalities exclusive to PPCM, elevated B-type Natriuretic Peptide (BNP) levels are often observed. However, it is essential to consider other laboratory studies for exclusionary purposes, including assessing cardiac enzymes and conducting a pre-eclampsia workup. Imaging studies play a crucial role in diagnosing PPCM. Electrocardiography (ECG) results are typically normal, but they may reveal sinus tachycardia, nonspecific ST- and T-wave abnormalities, and voltage changes [16]. Chest radiographs can display signs of pulmonary congestion, cardiac enlargement, and sometimes pleural effusions [8]. Echocardiograms are valuable in showing reduced contractility and Left Ventricular (LV) enlargement without hypertrophy [17].

Treatment and Prognosis

The treatment approach for PPCM is similar to that of other types of congestive heart failure. It includes measures like fluid and salt restriction, administration of β-blockers, diuretics, and digoxin. However, angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers are not recommended during pregnancy due to their contraindication [1]. To reduce afterload, hydralazine can be used safely during pregnancy.

When using diuretics during pregnancy, caution is exercised to prevent dehydration and placental insufficiency. Patients with PPCM are at increased risk of developing blood clots (thrombus formation) [18]. Therefore, anticoagulation therapy should be considered, especially for high-risk individuals with severe left ventricular dysfunction. Encouraging physical activity is also essential, but it should be tailored to the patient's tolerance level, taking into account their symptoms and overall condition.

The optimal duration for discontinuing these medications is uncertain, but it is generally advised to continue their use for at least one year [7]. If medical treatments prove ineffective, heart transplantation may be considered as a last resort. Fortunately, in recent times, the percentage of patients requiring transplantation has declined to approximately 4% to 7% [19].

Heart transplantation has shown promising success rates with favorable long-term survival outcomes. In about 50% of patients, the ejection fraction returns to normal. However, regardless of recovery, most patients are usually advised against having a second pregnancy due to the high recurrence rate of PPCM, which surpasses 30% in subsequent pregnancies, posing significant risks to both the mother and the baby [1].

Conclusion

Peripartum cardiomyopathy is a relatively uncommon condition affecting women during their childbearing years. Its exact etiology remains unknown, and it can recur in some cases, making it a challenging and complex condition to manage. The mortality rate associated with PPCM is notably high, warranting timely and accurate diagnosis. Diagnosing PPCM requires careful attention and vigilance from healthcare professionals due to its overlapping symptoms with other conditions. Once PPCM is identified based on established criteria, the primary therapeutic focus is on alleviating symptoms related to congestive heart failure.

A positive short-term prognosis is likely if the left ventricular size returns to normal after pregnancy. However, the long-term consequences, especially in the context of subsequent pregnancies, are not fully understood and necessitate further investigation. On the other hand, failure of the heart size to normalize is linked to increased morbidity and mortality rates, emphasizing the significance of prompt and effective management. Ongoing research is essential to gain a comprehensive understanding of PPCM and its potential implications, particularly in the context of future pregnancies.

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