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Peripartum Cardiomyopathy Complicated by Ventricular Tachycardia during Labor: A Case Report and Literature Review

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Abstract

Background: Peripartum cardiomyopathy (PPCM) is a rare disease that typically affects young, healthy women. Because PPCM is associated with significant mortality, timely diagnosis and management are essential. Ventricular tachycardia (VT) is a major complication and contributor to sudden death. Available data on VT in patients with PPCM are limited. Aim: This case report demonstrates the clinical presentation, antenatal care, and management of labor and delivery in a patient with PPCM complicated by VT. Case report: 36-year old patient G4P3 presents at 27 weeks gestation to the emergency department complaining of chest tightness, palpitations, and profuse sweating. Peripartum cardiomyopathy was diagnosed after her last pregnancy a few years prior. Ventricular tachycardia was diagnosed at this visit and treated successfully. The remainder of the pregnancy was uneventful until she had another episode of ventricular tachycardia during labor. Treatment using antiarrhythmics (diltiazem, amiodarone, adenosine) highlights the importance of prompt intervention and the need for a range of therapeutic options. Results: This case demonstrated successful VT management during pregnancy and labor, emphasizing multidisciplinary collaboration, influencing maternal and fetal outcomes positively, providing insights into optimal care strategies. Conclusion: Peripartum cardiomyopathy complicated by ventricular tachycardia is a life-threatening combination. This case highlights the importance of timely diagnosis and management with combined care between cardiologists, maternal fetal medicine specialists and anesthesiologists to prevent morbidities and sudden maternal death.

Keywords

Peripartum Cardiomyopathy, Ventricular Tachycardia, High Risk Pregnancy,

Antenatal Care, Delivery Planning

1. Introduction

Peripartum cardiomyopathy (PPCM) is a rare cardiac condition typically occurring in the final stages of pregnancy or the months following delivery, characterized by weakened myocardial contraction leading to heart failure [1]. It is a rare disease with wide variation, ranging from 1 case per 1000 to 4000 live births in the United States of America (USA).In previously heart-healthy women, peripartum cardiomyopathy (PPCM) is an uncommon and possibly fatal condition that develops around the end of pregnancy or in the months after birth [2].

Geographical location and ethnic origin significantly impact the incidence, which is estimated to be 1 in 1000 - 1500 pregnancies in Germany [3]. There aresignificant geographical and ethnic variations in the global prevalence of PPCM. Several areas have been classified as hot zones, including Haiti and Nigeria, where the rate of births is 1 in 300 and 1 in 100, respectively [4].

Although most women who develop PPCM have no family history of cardiomyopathy, genetics and family history may potentially be factors. A number of risk factors consist of: 35 years of age or older for mothers elevated blood pressure, encompassing gestational hypertension or preeclampsia [5]. The exact incidence in Saudi Arabia is unknown. Only one retrospective study was reported on 16 patients over six years (1:15,000 live births) at Riyadh Central Hospital [6]. In extreme situations, women's health rapidly deteriorates without getting better despite medication, and they may need a heart transplant or pass away from heart failure, thromboembolic accidents, or cardiac arrhythmias [7]. PPCM can be complicated by arrhythmias, including ventricular tachycardia (VT) in 4.2 % of the patients, which can be life-threatening in 2.2%, causing cardiac arrest. Here, we report a case of PPCM from a previous pregnancy, presents pregnant with symptomatic tachycardia and dyspnea [8].

2. Literature Review

Mother morbidity and death have significantly increased globally as a result of pregnancy-related heart failure [9]. The mother's circulatory system is extremely stressed by hemodynamic changes, especially in the latter stages of pregnancy and during delivery. Due to the potential for adverse effects on heart function, women with congenital or acquired cardiomyopathies are at an increased risk [10]. Hospitalizations following childbirth are attributed to maternal cardiac problems in around 4% of cases, with an increasing tendency [11].

Healthcare practitioners struggle to diagnose and treat patients in an environment of insufficient evidence-based therapies. Young women are disproportionately affected by peripartum cardiomyopathy (PPCM), a severe, frequently

misdiagnosed, and potentially fatal pregnancy-related cardiac disease [12]. This thorough study aims to outline disease-specific therapy approaches and compile the most recent research on PPCM. The urgent need for efficient therapies in the face of diagnostic obstacles highlights the importance of improving our knowledge and treatment of PPCM [13].

3. Definition, Epidemiology, and Risk Factors

Peripartum cardiomyopathy (PPCM) is s a rare cardiac condition typically occurring in the final stages of pregnancy or the months following delivery, characterized by weakened myocardial contraction leading to heart failure. It usually appears in previously healthy women in the last month of pregnancy or the month following birth. A left ventricular ejection fraction (LVEF) that is nearly always below 45% is the hallmark diagnostic criteria, as established by the Study Group on PPCM of the Heart Failure Association (HFA) of the European Society of Cardiology (ESC) [14]. Due to the exclusionary nature of the PPCM diagnosis, thorough clinical evaluations are required to rule out other possible causes of heart failure, such as pregnancy-associated myocardial infarction or pulmonary embolism, as well as pre-existing heart conditions, such as congenital heart defects or toxic cardiomyopathy brought on by chemotherapy. Even though PPCM and dilated cardiomyopathy share certain traits, they are considered separate clinical entities [15].

There is much heterogeneity in the global incidence of PPCM, with substantial geographical and ethnic variations. With incidence rates of one in 100 and one in 300 births, respectively, several regions—Nigeria and Haiti, for example—are identified as hot zones [16]. The estimated incidence in Germany is around 1:1000 - 1500, which is consistent with data from the United States (1:1500) and South Africa (1:1000). It is essential to recognize that milder types of PPCM may remain undetected because of vague symptoms and low awareness. Global death rates range from 2 to 30%; however, new German data show that individuals with Parkinson's disease who receive prolactin blocker bromocriptine in addition to normal heart failure medication had a lower death rate of 0% [17].

Preeclampsia, gestational hypertension, and HELLP syndrome are among the pregnancy-associated hypertensive illnesses that have been linked to a higher risk of developing postpartum depression (PPCM). In vitro fertilization, multifetal pregnancies, older mothers, and multiparity are also linked to an elevated risk of postpartum hemorrhage (PPCM). African American women are more likely to experience this risk than their Caucasian and Asian counterparts [13].

3.1. Pathophysiology of PPCM

Although several explanations, including autoimmune processes, inflammatory factors, viral infections, and low selenium levels, have been hypothesized, the pathophysiology of PPCM remains largely unclear. All of these methods haven't

been thoroughly shown yet, though. One possible beginning and driving cause of PPCM surfacing in recent years is a change in the angiogenic balance toward an anti-angiogenic environment [18]. The cleaved N-terminal 16-kDa prolactin fragment and sFlt-1 are critical components of these elevated anti-angiogenic factors [19].

3.2. Prolactin Pathway

Using a mouse model with a cardiomyocyte-specific knockout of the signal transducer and activator of transcription factor-3 (STAT3), Hilfiker-Kleiner and colleagues made a significant breakthrough in the disease-specific treatment strategy for peripartum cardiomyopathy (PPCM) in 2007 [20]. They discovered the critical role of the nursing hormone prolactin. The key mechanism is elevated oxidative stress, which causes proteolytic enzymes such asmatrix metalloproteinases and cathepsin D to cleave the protective 23-kDa prolactin fragment into the smaller 16-kDa prolactin fragment. Vasoinhibin, a 16-kDa fragment, is also known to directly impair endothelium function and trigger the release of micro-RNA 146a, both of which are harmful to cardiomyocytes. Systolic heart failure is the outcome, it is essential to note that it may be reversed [21].

Restoring the damaged myocardium requires a specific course of therapy with bromocriptine, a drug that inhibits the pituitary gland's secretion of prolactin. Bromocriptine became a targeted treatment strategy for PPCM by stopping the cleavage of full-length prolactin into the hazardous 16-kDa fragment. By targeting the disease's underlying pathophysiological pathways, this milestone offers a potentially reversible strategy to controlling and comprehending post-poliomyolysis (PPCM) [22].

3.3. sFlt-1 Pathway

The soluble fms-like tyrosine kinase-1 (sFlt-1) receptor of vascular endothelial growth factor (VEGF) is a key component in the pathogenesis of preeclampsia, a hypertension condition during pregnancy. Among other places, the placenta produces sFlt-1 in late pregnancy, which is linked to a systemic angiogenic imbalance. In a mouse model devoid of cardiac peroxisome proliferator-activated receptor gamma coactivator 1 alpha (PGC-1α), Patten and colleagues showed that vascular dysfunction caused by increased sFlt-1 produces peripartum cardiomyopathy (PPCM), which results in significant impairment of heart function [23].

Interestingly, in mice with PPCM, pro-angiogenic treatment with VEGF alone had no positive effects. Nevertheless, bromocriptine and recombinant VEGF worked well together to save these animals. These results underscore the importance of prolactin while stressing the role of additional anti-angiogenic variables in the pathogenesis of PPCM. The applicability of such combination therapy, including recombinant VEGF, to patients with PPCM in humans is still unclear and needs more research despite these insights from animal models [24].

3.4. Genetics

New research supports the notion of an everyday genetic basis by showing that 15% of individuals with peripartum cardiomyopathy (PPCM) had mutations in genes associated with dilated cardiomyopathy. Heart myosin heavy chain (MYH), titin (TTN), and SCN5 are among the identified genes with alterations. It seems that the significant hemodynamic changes that occur during late pregnancy, delivery, and the early postpartum period may reveal genetic cardiomyopathies that were previously quiet [25]. Many mutation carriers are asymptomatic before becoming pregnant. This implies that in susceptible people, the hemodynamic stress experienced during pregnancy and delivery may function as a trigger, revealing underlying genetic predispositions to cardiomyopathies [26].

3.5. Signs and Symptoms

Peripartum cardiomyopathy's (PPCM) range is quite broad, ranging from milder types with modest, nonspecific symptoms to severe instances with potentially fatal cardiogenic shock. In the later stages of pregnancy or postpartum, physicians often see seemingly healthy women who describe nonspecific symptoms such as tiredness, peripheral edema, and general discomfort [27]. These minor signs frequently mimic peripartum problems that are prevalent. On the other hand, people who are more seriously impacted could have dyspnea, orthopnea, and restlessness. Cardiogenic shock, which is characterized by peripheral hypoperfusion and pulmonary edema, may appear in the most extreme instances [28].

3.6. Diagnostics

It is imperative that a detailed diagnostic work-up not impede early diagnosis. Signs of congestion, such as peripheral edema, jugular vein distention, and pulmonary rales, may be discovered during a physical examination. Due to centralization and volume overload, patients may seem pale, and those in cardiogenic shock may have chilly, wet skin. Two essential diagnostic tests are advised when pregnancy-related cardiac disease is suspected [29].

Brain natriuretic peptide (BNP) and its N-terminal prohormone (NT-proBNP) are screening biomarkers for heart failure that may be measured in the bloodstream. Elevated levels can promptly rule out acute heart failure even if they are not specific to cardiac disorders related to pregnancy [30].

Transthoracic echocardiography: This easily accessible test can quickly determine the function of the left ventricle (LV), supporting or refuting the diagnosis of PPCM. Additionally, it assists in detecting concurrent diseases, including mitral regurgitation and involvement of the right ventricle [31].

While not commonly used, cardiac magnetic resonance imaging (MRI) has a unique function in evaluating biventricular systolic function and describing myocardial tissue. The electrocardiogram (ECG) helps identify indications of myocardial ischemia/infarction, and endomyocardial biopsies and coronary angiography are investigated in some circumstances, albeit they are not regularly advised [32].

3.7. Therapy

The care of PPCM is by the European Society of Cardiology's guidelines for both acute and chronic heart failure, the Study Group on PPCM's practical recommendations and a landmark study on the subject. Assessing hemodynamic state and analyzing indicators of congestion and cardiogenic shock are essential factors to consider [33].

Treatment of hemodynamically unstable acute PPCM

Women who are experiencing cardiopulmonary distress and abrupt heart failure need to be transferred right away to an intensive care unit with experience. Optimize preload with fluid or diuretics based on volume status. Vasodilators are advised for systolic blood pressure of more than 110 mm Hg; hydralazine is the preferred medication for pregnant women [34]. Inotropes and/or vasopressors may be required when a patient is hemodynamically unstable, although catecholamines should be used cautiously. An inodilator like levosimendan is a better choice. Mechanical circulatory support (MCS) should be considered early; for isolated left ventricular failure, percutaneous devices such as Impella® are preferred; veno-arterial ECMO should be combined with MCS for biventricular failure. Biventricular failure requires immediate cesarean delivery, along with corticosteroids for fetal lung maturation up to the 34th week of pregnancy [35].

3.8. Treatment of Hemodynamically Stable Acute PPCM

Optimizing maternal hemodynamics and fetal monitoring are the main goals of therapy for hemodynamically stable PPCM patients who are still pregnant. Heart failure patients are treated with beta-blockers, vasodilators (ideally hydralazine), and diuretics; diuretics are only used in situations of fluid excess. Before week 34 of pregnancy, fetal lung maturity should be induced, and vaginal birth is recommended for hemodynamically stable individuals [36].

Following birth, conventional heart failure care is started, which includes beta-blockers at dosages guided by guidelines and ACE inhibitors/ARBs. For LVEF <40%, mineralocorticoid receptor antagonists (MRA) are advised; in younger female patients, eplerenone is preferred. For individuals with ongoing symptoms, ACE inhibitors and ARBs are substituted with valsartan and sacubitrin. Ivabradine is taken into consideration for poorly regulated heart rates. Diuretics are only used in cases of excessive fluid [37].

3.9. Bromocriptine

For patients with premature paternal congestive heart failure (PPCM), bromocriptine has three advantages: it stops breastfeeding to lower metabolic needs, it makes the safe introduction of heart failure medicine easier, and it treats the condition specifically by blocking prolactin production. Clinical and experimental data support its usage. Sixty-three women with LVEF <35% were randomly assigned to short- and long-term bromocriptine regimens in a German

trial [38]. There was a trend favoring the long-term group, particularly in patients with severe illness, even though the primary endpoint (LVEF change) was neutral. No fatalities, LVAD implantations, or heart transplants indicated the safety and possible advantages of bromocriptine. Comparing it to a U.S. cohort indicated it effectively stopped LVEF decrease and catastrophic events. It is advised to use prophylactic anticoagulation when using bromocriptine [39].

3.10. BOARD scheme

The BOARD plan describes the postpartum PPCM treatment: all patients are prescribed conventional oral heart failure medicine in approved doses, in addition to Bromocriptine. When using bromocriptine, prophylactic anticoagulation is essential to preventing thrombotic events. When systolic blood pressure is higher than 110 mm Hg, vaso-relaxing medications are recommended to lessen afterload; diuretics are used in situations of fluid overload. The plan aligns with the most recent suggestions for efficient postpartum PPCM care [40].

3.11. Prevention of sudden cardiac death

German research found that 12% of patients with LVEF ≤35% in PPCM had ventricular arrhythmias, which can cause sudden cardiac death (SCD). However, the precise reasons are unknown. Within three to six months, most patients recover or exhibit notable improvement, lowering the risk of malignant arrhythmias. Implanting an immediate permanent cardioverter/defibrillator (ICD) might not be appropriate [41]. To avoid SCD, it is advised to use a wearable cardioverter/defibrillator (WCD; LifeVest®, Zoll, Pittsburgh, PA, USA). According to current recommendations, implantation of an ICD (transvenous or subcutaneous) or cardiac resynchronization device defibrillator (CRT-D) is recommended for patients who are not recovering or who are suffering unstable ventricular tachycardia or fibrillation [42].

3.12. Prognosis

Even with different clinical paths, PPCM patients treated according to current standards have a good prognosis. About 35% - 40% partially recover (LVEF improvement >10%, at least one NYHA class), and about 50% fully recover (LVEF >55%, NYHA class I). There is still a minority in NYHA class III/IV who require heart transplantation or the installation of a left ventricular assist device. Poorer outcomes are predicted by parameters such as initial LVEF <30%, left ventricular end-diastolic diameter >60 mm (IPAC study), and right ventricular dysfunction revealed by MRI. For these high-risk individuals, bromocriptine could be especially beneficial [43].

3.13. Contraception and subsequent pregnancies

Given that the majority of heart failure medications are contraindicated during pregnancy and breastfeeding, women with heart failure are at greater risk during

(or after) pregnancy. For this reason, effective contraception is essential to avoiding side effects and teratogenicity. Because of their unreliability, barrier procedures are discouraged; also, estrogen-based methods are not advised. Alternatively, progesterone-only oral contraceptives and intrauterine devices that release copper and levonorgestrel are thought to be safe. Tubal ligation may be a safe alternative for patients with chronic severe heart failure (LVEF <30%) who have completed family planning [44].

With chronic left ventricular dysfunction, women who become pregnant again (SSP) are more likely to experience heart failure consequences, such as increased symptoms, decreased left ventricular ejection fraction, and increased maternal death. SSP has a better prognosis in women who fully recover, but generally, PPCM patients are at risk for heart failure, recurrence, and mortality [45]. According to a recent trial conducted in Germany, Scotland, and South Africa, bromocriptine added to routine heart failure medication after birth improved the prognosis for SSP patients. In the bromocriptine group, LVEF was considerably more significant, and the total complete recovery rate was significantly better. Regardless of LV function, bromocriptine is advised in PPCM patients with SSP [46].

4. Case report

36-year-old G4P3A1 presented at 27 weeks gestation to the emergency department complaining of chest tightness, palpitations, and profuse sweating. PPCM was diagnosed after a previous pregnancy five years prior. The ECG showed Ventricular Tachycardia with wide complex tachycardia (**Figure 1**). Sinus rhythm was achieved after one dose of diltiazem. She was admitted to the intensive cardiac unit due to congestive heart failure. The echocardiogram showed severe ventricular systolic dysfunction with an ejection fraction of 20%, suggesting the diagnosis of PPCM after the exclusion of other causes of heart failure by the cardiologist. Bisoprolol and furosemide were started with significant improvement. The pregnancy continued with no further complications and the patient was followed in the Maternal Fetal medicine unit in our hospital. Induction of labor was planned at 37 weeks. Continuous maternal cardiac monitoring was implemented in addition to fetal heart rate monitoring. Epidural anesthesia was given to decrease catecholamine release. During the first stage of labor, the patient developed ventricular tachycardia with a heart rate of 220/min; she remained hemodynamically stable; she was treated with 150 mg of amiodarone followed by 10 mg of diltiazem followed by 6mg of adenosine to sustain a sinus rhythm. Fetal heart rate remained reassuring. In the first portion of the second stage of labor, the descent of the presenting part was facilitated by uterine contractions under epidural coverage. The latter portion of the second stage of labor was shortened by forceps delivery. Both were instituted to decrease the mother's expulsive efforts in the second stage of labor. The outcome was a live-born baby boy weighing 3 kg with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. Catheter Ablation was done the following year.

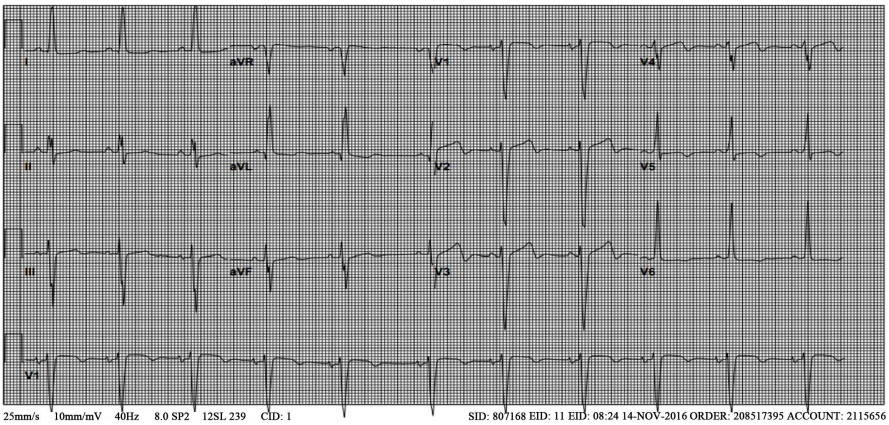

Figure 1. Sinus rhythm with sinus arrhythmiaand left ventricular hypertrophy with QRS widening and repolarization abnormality.

5. Results

The provided case report mentions several significant findings and outcomes in terms of diagnostic discovery where the patient presented with peripartum cardiomyopathy (PPCM) and a history of the same condition in a previous pregnancy. Ventricular tachycardia (VT) was diagnosed during the current pregnancy, leading to additional complexities. Successful treatment of VT during the pregnancy using antiarrhythmics, including diltiazem, amiodarone, and adenosine. Initiation of Bisoprolol and furosemide, resulting in significant improvement. Catheter ablation was performed in the following year.

For the maternal and fetal outcomes there was continuous maternal cardiac monitoring during labor. Use of epidural anesthesia to mitigate catecholamine release. Successful management of VT during the first and second stages of labor. A live-born baby boy with reassuring Apgar scores at 1 and 5 minutes, weighing 3 kg.

Interdisciplinary collaboration involves cardiologists, maternal-fetal medicine specialists, and anesthesiologists for comprehensive care. Use forceps delivery to shorten the second stage of labor and decrease maternal expulsive efforts.

These outcomes collectively underscore the successful interdisciplinary management of PPCM complicated by VT during pregnancy, emphasizing the importance of timely diagnosis and collaborative care.

6. Discussion

The case report outlines a complex presentation of peripartum cardiomyopathy (PPCM) in a 36-year-old G4P3A1 patient at 27 weeks gestation. Several notable results and aspects emerge from the case, prompting valuable discussion regarding the PPCM Diagnosis and Previous History, where the patient had a history of PPCM from a previous pregnancy, indicating a recurrence of the condition. Similar findings were noted by a case report by Mary Wang, the patient had previous history of igh risk pregnancy with PPCM indicating recurrence of the

condition [47].

Timely diagnosis during the current pregnancy was crucial, emphasizing the need for vigilant monitoring in patients with a history of PPCM. The occurrence of VT during pregnancy is a severe complication and poses a risk of sudden maternal death. While rare, ventricular tachycardia can occasionally make pregnancy more difficult. Its existence might be a sign of an unidentified congenital arrhythmic illness or an underlying anatomical anomaly of the heart. Nonetheless, some ventricular tachycardia patients who are pregnant have hearts that are anatomically normal. We describe two examples of ventricular tachycardia in pregnant individuals with structurally normal hearts and talk about how to diagnose and treat these patients [48].

Successful treatment using antiarrhythmics (diltiazem, amiodarone, adenosine) highlights the importance of prompt intervention and the need for a range of therapeutic options. The case underscores the significance of collaborative care involving cardiologists, maternal-fetal medicine specialists, and anesthesiologists. This approach ensures comprehensive management, especially during critical periods like labor, where continuous maternal cardiac monitoring and careful use of anesthesia become crucial. Antiarrhythmic medication appears to be most beneficial for individuals in whom early administration of the medication follows unsuccessful early attempts at defibrillation and CPR. There doesn't seem to be a definite survival advantage for any one treatment, thus when choosing a medication, other aspects like cost and availability should be considered. Furthermore, in cases where defibrillation efforts and antiarrhythmic medication are ineffective, other therapies (such as extra-corporeal CPR and percutaneous coronary intervention) may offer a further advantage to life [49].

The essential element in diagnosing PPCM is the development of left ventricular systolic dysfunction. Specific echocardiographic measures of left ventricular systolic dysfunction have been proposed as additional criteria; these include an ejection fraction of less than 45%, or M-mode fractional shortening of less than 30%, or both, an end-diastolic dimension of greater than 2.7 cm/m^2 body surface area [14].

PPCM is strongly associated with maternal age, with the majority of affected women in their 30s. Multiple gestations seem to be another risk factor for PPCM; in a recent meta-analysis, the association between twin pregnancies and the diagnosis of PPCM was noted. In the same study, the prevalence of hypertension and preeclampsia were markedly higher than in the general population. Other associations have been suggested, such as prolonged tocolysis, obesity, and diabetes [50].

Data on VT in patients with PPCM are limited. Diao *et al.* reported a case series of 19 women with PPCM, and VT was reported in four women (21%) [51].

In another case series of 45 patients with PPCM in Pakistan, 3 developed VT (6.6%) [52]. A nationwide inpatient sample database from the United States reported on 9841 hospitalizations of women with PPCM from 2007 to 2012. Arrhythmias were noted in 18.7% of admissions, of which VT was the most fre-

quent, seen in 4.2%. PPCM with arrhythmias had a more extended hospital stay, higher charges, and worse in-hospital outcomes. In-hospital mortality was noted in 2.1% of PPCM with arrhythmias, which was 3-fold higher than those without arrhythmia [53]. The underlying mechanisms of the association between PPCM and VT are not fully understood, but several hypotheses exist. One theory suggests hormonal impacts, autonomic alterations, cardiac fibrosis, and inflammation. Another theory is that the hemodynamic changes associated with PPCM, such as increased left ventricular end-diastolic pressure and decreased left ventricular ejection fraction, may predispose patients to VT [54].

This case highlights the importance of timely diagnosis and management with combined care between cardiologists, maternal-fetal medicine specialists, and anesthesiologists to prevent morbidities and sudden maternal death. Continuous monitoring during labor, including fetal heart rate, helped manage VT episodes while ensuring fetal well-being. Successful forceps delivery and a live-born baby with good Apgar scores suggest effective management and positive outcomes. The decision to perform catheter ablation in the following year indicates a long-term management strategy to address the recurrent nature of PPCM in this patient.

Limited data on VT in PPCM patients are highlighted, emphasizing the need for more research into this aspect of the condition. The association between PPCM and VT may involve hormonal, autonomic, fibrotic, and inflammatory factors, warranting further investigation. The case emphasizes the critical need for heightened monitoring, early diagnosis, and a collaborative approach in managing PPCM during pregnancy.

Recommendations include enhanced monitoring, collaborative care among specialists, and prompt intervention to improve outcomes for PPCM patients at risk of VT during pregnancy.

7. Conclusion

In summary, this case report contributes valuable insights into the intricate management of PPCM and its complications during pregnancy. The successful outcomes highlight the importance of interdisciplinary collaboration, timely intervention, and ongoing care for patients with PPCM, especially those at risk of ventricular tachycardia. PPCM and VT are severe conditions that require prompt diagnosis and treatment to prevent complications and improve outcomes. While the relationship between the two is not yet fully understood, several studies suggest that there may be an association between PPCM and VT. Further research is needed to understand this association's underlying mechanisms and develop optimal treatment strategies.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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