

REVIEW ARTICLE



Advancing Our Understanding of Peripartum Cardiomyopathy: Current Evidence and Future Directions.

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Abstract

Peripartum cardiomyopathy (PPCM) is a rare, life-threatening condition characterised by symptoms of heart failure that occur in the last month of pregnancy or within five months after delivery, with no other identifiable cause. It is identified by a left ventricular ejection fraction (LVEF) below 45%. PPCM affects approximately one in 2000 births worldwide, with significantly higher rates in Nigeria (1 in 100 births) and among Black Americans, who often experience more severe disease and poorer recovery. Common risk factors include advanced maternal age, hypertension, preeclampsia, multiple pregnancies, and genetic predisposition. The underlying causes are not fully understood but likely involve hormonal imbalances (e.g., prolactin cleavage, elevated sFlt-1), genetic mutations (e.g., in the TTN gene), and inflammatory processes. Symptoms such as exertional shortness of breath, fatigue, and leg swelling can resemble normal pregnancy changes, making diagnosis challenging. Echocardiography is key for detection, and other conditions like pre-existing cardiac disease must be excluded. While new treatments like bromocriptine show promise but require further research, management involves standard heart failure medications (beta-blockers, angiotensin-converting enzyme inhibitors, sodium-glucose co-transporter 2 inhibitors, and diuretics), with considerations for pregnancy and lactation. Early diagnosis, supported by tools like echocardiography and BNP levels, along with a multidisciplinary approach, is essential to improve outcomes. This review highlights the importance of increased awareness and ongoing research to better care for women with PPCM.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a life-threatening idiopathic cardiomyopathy defined by acute heart failure (left ventricular ejection fraction [LVEF] <45%) occurring in late pregnancy or within five months of delivery, abortion or miscarriage, without identifiable cause.^{1,2} In some cases, the disease can occur earlier in pregnancy or later than five months after delivery.¹ While diagnostic criteria emphasise LVEF <45%, emerging evidence supports including patients with LVEF of between 45% and 50% after excluding alternative aetiologies (e.g., pre-existing cardiac disease,

pulmonary embolism, acute takotsubo syndrome).² It should be noted that acute takotsubo syndrome has been reported during the last trimester or after emergency delivery, and this requires careful assessment to differentiate it from PPCM.² PPCM's diagnosis remains challenging due to overlapping symptoms with normal pregnancy (fatigue, oedema) and the need to differentiate it from obstetric conditions like preeclampsia.

Globally, PPCM affects approximately 1 in 2,000 births, but its incidence varies widely across regions. Sub-Saharan Africa bears the greatest burden, with Nigeria reporting rates

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as high as 1 in 100 deliveries.^{1,3} Northern Nigeria has the highest incidence and largest cohort in the early 70s and the longest follow-up (30-35years) was done in Zaria, northern Nigeria.⁴ In Haiti, it complicates 1:300 births.^{1,2,3} The incidence among the Caucasian population is also variable, ranging from 1:1500 in Germany and 1:10000 in Denmark.² In the United States, African Americans present at a younger age and late in postpartum with EF < 30%, which may be a result of a higher rate of hypertension and pre-eclampsia and late presentation.^{1,2} Other risk factors include advanced maternal age, anaemia, multiple gestation, multiparity, family history, smoking, diabetes, malnutrition, and prolonged use of tocolytics.^{1,2}

Patients typically present with dyspnea, orthopnea, and peripheral oedema. Diagnosis relies on electrocardiogram, echocardiography, and biomarkers (e.g., brain natriuretic peptide [BNP]); however, in resource-limited settings, limited access to these diagnostic tools often leads to delayed recognition and initiation of appropriate management.^{1,2} Nigeria has a high mortality rate for PPCM, with 18.7% of patients dying during a median follow-up of 17 months, according to the PEACE Registry.⁵ In comparison, mortality rates in other African countries vary widely, with Burkina Faso reporting 48.3% over 4 years, Zimbabwe 11.6% over 6 months, and South Africa 13.0% over 6 months.^{6,7} Globally, mortality rates are generally lower, ranging from 6% to 10% in the United States, though differences in study design and methodology make direct comparisons challenging.⁷⁻⁹

The full recovery rate for PPCM in Nigeria is about 22.6%,⁵ as reported by the PEACE Registry. However, in Africa, the recovery rates for PPCM vary between 20% and 60%,¹⁰⁻¹² reflecting the continent's diverse healthcare systems and socioeconomic conditions.¹³ Globally, approximately 58% of PPCM patients recover, according to the EORP PPCM registry, highlighting the benefits of early detection and advanced medical interventions.⁸

PPCM's toll extends beyond statistics, devastating families—a mother's late diagnosis in Nigeria, for example, may orphan her newborn, highlighting the urgent need for early intervention and research.¹

PATHOGENESIS

The pathogenesis of PPCM is still unknown despite the advances in research. However, a two-hit model that emphasises the relationship between patient susceptibility and systemic angiogenic imbalance (pregnancy stressor) as key contributors to disease development and progression has been proposed (Figure 1).^{1,2} The pregnancy stressor may be related to failed hemodynamic stress tests, which are often prominent in the second trimester.¹³ Other models postulated include selenium deficiency, autoimmune theory, viral infection theory, stress-activated cytokine, inflammation, and pathological response to hemodynamic changes in pregnancy.^{1,2}

The Vasculo-Hormonal Model

The hormonal theory emphasises the damage caused by pituitary hormones and placental hormones.¹ These hormones, when initially secreted, support maternal physiology and fetal growth and development. Prolactin, secreted by the pituitary gland in late pregnancy and early postpartum, undergoes cleavage into a 16-kDa fragment from 23-kDa by cathepsin D, a process initiated by increased oxidative stress.¹⁴⁻¹⁶ The 16-kDa isoform has potent

antiangiogenic and proapoptotic effects, disrupting cardiac microvascular endothelial cells, impairing myocardial perfusion and triggering cardiomyocyte apoptosis via exosomal microRNA-146a.^{1,14-16} The above supports the suggestions such as withholding breastfeeding or use of prolactin inhibitors (bromocriptine) in the management of PPCM.^{1,16}

Oxytocin, which is equally secreted during this period, has been implicated in patients with peripartum aortic dissection in Marfan's syndrome, though its direct link in PPCM is not known.¹

The placenta (syncytio-trophoblast) secretes several glycoproteins, growth factors and neuropeptides. Among these, soluble Smf-like tyrosine kinase 1(sFlt-1) a decoy receptor that binds vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), neutralising their angiogenic effects.^{1,17} This protein is secreted in larger quantities in pre-eclampsia and multiple gestation and correlates with subclinical myocardial injury, detectable via speckle-tracking echocardiography.¹ In PPCM, sFlt-1 may exacerbate cardiac microvascular rarefaction, impairing oxygen delivery and promoting fibrosis.¹⁸ Notably, sFlt-1 levels decline postpartum, paralleling cardiac recovery in some patients.^{1,18,19}

Progesterone, another placental hormone, exerts negative inotropic effects on the heart and potentially predisposes it to oxidative injury.^{20,21} Additionally, activin A, a glycoprotein elevated in preeclampsia, is known to induce cardiomyocyte hypertrophy and apoptosis via SMAD2/3 signalling.

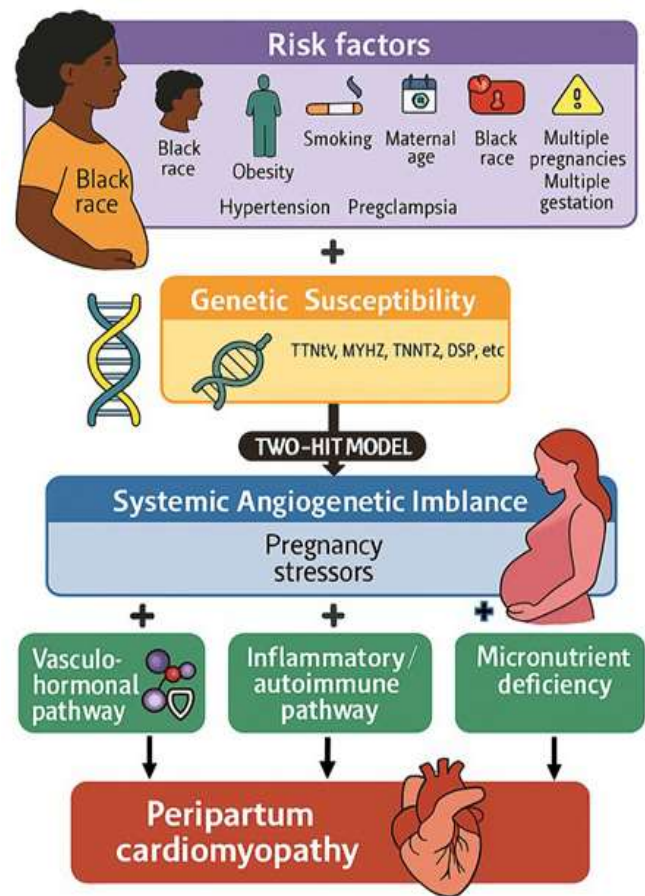


Figure 1: Proposed pathogenesis of peripartum cardiomyopathy

Meanwhile, reduced relaxin-2, commonly seen in patients with PPCM, further compromises cardiac adaptation due to the loss of its vasodilatory and anti-fibrotic effects.²² Together, these imbalances create a milieu of vascular insufficiency and metabolic stress, particularly in genetically susceptible women.

Genetic Susceptibility

Approximately 15% of PPCM patients harbour pathogenic variants in cardiomyopathy-associated genes, most commonly TTN, which encodes titin, a sarcomeric protein critical for myocardial elasticity.^{23,24} Truncating TTN variants (TTNtv) is implicated in most of the PPCM cases with genetic abnormalities, mirroring findings in dilated cardiomyopathy (DCM).^{24,25} However, TTNtv penetrance is low (<5%), suggesting that pregnancy-related stressors (e.g., hormonal shifts, volume overload) are necessary to unmask clinical disease.²⁴ Other implicated genes include MYH7 (β -myosin heavy chain), TNNT2 (troponin T), and DSP (desmoplakin), which disrupt contractility, calcium handling, or cell-cell adhesion.^{24,25}

Intriguingly, PPCM shares genetic overlap with chemotherapy-induced and alcoholic cardiomyopathies, both of which involve toxin-mediated myocardial injury in genetically predisposed individuals.^{24,26,27} This further suggests that, beyond genetic abnormality, other environmental factors may contribute to the pathogenesis of cardiomyopathy.¹

Inflammation and Autoimmune Mechanisms

Inflammation has been proposed as a key factor in PPCM, though whether it is a primary cause or a secondary consequence of disease progression remains unclear.²⁸ Elevated levels of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF- α) and interleukins, have been observed in PPCM patients.²⁹ Additionally, the role of microchimerism, in which fetal cells persist in maternal circulation postpartum, has been suggested as a potential trigger for autoimmune responses against cardiac tissue, although the exact mechanism remains poorly understood.^{30,31}

Micronutrient Deficiencies Traditional Practices.

Selenium, a trace element essential for antioxidant defence, is deficient in some patients with PPCM in Nigeria.³²⁻³⁴ Selenium deficiency impairs glutathione peroxidase activity, exacerbating oxidative stress and endothelial dysfunction. However, this association is absent in North American and European and some Nigerian cohorts, highlighting regional etiological diversity.^{1,35}

The earliest studies in northern Nigeria had associated the development of PPCM with a traditional practice after delivery called *wakan jeko*. It is made up of a triad characterised by a hot bath, intake of high salt-containing drinks (*kumun kanwa*) and lying on a hot bed for about three months.⁴ Health education on the need to avoid such practice led to the reduction in the incidence of PPCM.⁴ The content of the PEACE registry had downplayed the contribution of this traditional practice in the development of PPCM.³⁶ It suffices to say that the practice of *wakan jeko* has drastically reduced, if not completely out of practice, and it is therefore not surprising that the PEACE registry did not find an association with PPCM and *wakan jeko*.

The pathogenesis of PPCM is complex and likely multifactorial, involving hormonal imbalance, genetic predisposition, inflammatory responses, and environmental influences. While significant progress has been made in understanding these mechanisms, further research is needed to delineate the interplay between these factors and identify targeted preventive and therapeutic strategies.

RISK FACTORS AND CLINICAL PRESENTATION

The exact cause of PPCM is still unknown, but several risk factors have been identified which including advanced maternal age, with a higher incidence in women over 30 years old, and a significant increase in risk for women older than 40 years.^{36,37} PPCM is also known to disproportionately affect Black women, who tend to present with more advanced disease and more severe left ventricular systolic dysfunction, with a corresponding lower rate of left ventricular ejection fraction (LVEF) recovery and a higher risk of death.^{36,38} Chronic hypertension and pre-eclampsia are common risk factors, with the prevalence of pre-eclampsia among patients reaching up to 46% in Asians.^{38,39} Other risk factors include multiple gestations, multiparity, obesity, smoking, and genetic predisposition.

PPCM patients typically present with signs and symptoms of heart failure (HF).³⁸ Two-thirds of patients develop HF symptoms postpartum, with the majority in the first month postpartum.³ Women with PPCM typically present with fatigue, dyspnea, orthopnea, nocturnal paroxysmal dyspnea, chest tightness, tachycardia, tachypnoea, elevated jugular venous pressure, and pulmonary oedema. Some women may also experience syncope, atrial/ventricular arrhythmias or thromboembolic events.³⁶⁻³⁸ The severity of symptoms can vary, ranging from mild to severe, potentially leading to cardiogenic shock.

Diagnosing PPCM during late pregnancy and early postpartum can be challenging, as some symptoms overlap with common pregnancy complaints.³⁷ Differential diagnoses, such as pre-existing heart disease, pulmonary embolism, preeclampsia-induced pulmonary oedema, spontaneous coronary artery dissection, myocarditis, Takotsubo syndrome, myocardial infarction, and aortic dissection, should be ruled out.

DIAGNOSTIC CRITERIA AND TOOLS

PPCM diagnosis is primarily clinical but should be confirmed with cardiac imaging. The diagnostic criteria include the following: development of heart failure within the last month of pregnancy or within five months postpartum; no pre-existing heart disease before the last month of pregnancy; echocardiographic evidence of left ventricular systolic dysfunction, typically with a LVEF of less than 45%; and absence of an identifiable cause of heart failure.^{38,39}

Investigations for PPCM are similar to those for other causes of heart failure. Transthoracic echocardiography (TTE) is essential for suspected cases, as it typically shows an LVEF of less than 45%. TTE may also reveal left and right ventricular dilatation and/or dysfunction, functional mitral and tricuspid regurgitation, pulmonary hypertension, and atrial enlargement. Initial LVEF <30%, marked LV dilatation (LV end-diastolic diameter ≥ 6.0 cm), and RV involvement are associated with adverse outcomes.³⁷ Intracardiac thrombus may occur, and the LV apex should be

visualised, especially when LVEF is severely reduced. If TTE results are unclear for suspected LV thrombosis, perform a transoesophageal echocardiogram.

The electrocardiogram (ECG) may show abnormalities such as features of chamber enlargement, hypertrophy, ST-T wave changes or arrhythmias. These findings are not specific to PPCM. However, a normal ECG does not rule out PPCM. Some ECG changes may also be detected in uncomplicated pregnancies, such as left axis deviation, transient ST/T abnormalities, inverted T waves in the anterior leads and in the inferior leads.³⁷

Additional investigations may be required, depending on the clinical presentation. Chest x-rays may show cardiomegaly, features of pulmonary oedema and pleural effusion. Cardiac magnetic resonance imaging provides accurate measurements of LVEF, chamber sizes and is more reliable in detecting LV thrombus. It is invaluable in situations where the TTE study is inadequate, but gadolinium should be avoided during pregnancy. Levels of brain natriuretic peptide (BNP) and N-terminal pro-BNP (NTpro-BNP), which do not change significantly during normal pregnancy and may be mildly elevated in the setting of pre-eclampsia, are usually markedly elevated in PPCM.⁴⁰ Cardiac troponin levels may be increased in PPCM. High troponin levels (especially high-sensitivity troponin) have been associated with poorer outcomes in PPCM.⁴⁰ Endomyocardial biopsy (EMB) is only indicated if an alternative diagnosis, such as myocarditis, is being considered, which would necessitate a different management plan.³⁶

Newer diagnostic tools are being developed. Recently, deep learning models based on ECG data have been developed to assist in the diagnosis of PPCM in women during pregnancy and the puerperium.^{41–43} The SPEC-AI study among pregnant Nigerians showed that such a model may be invaluable in early diagnosis of PPCM, especially in resource-poor settings with little knowledge or non-availability of diagnostic tools such as ECG or TTE.⁴² Comparable tools were developed using data from patients at the Mayo Clinic and Asian populations. The European Society of Cardiology (ESC) EUR Observational Research Programme (EORP) PPCM Registry also developed a model for predicting LV recovery in PPCM patients. How these tools will be integrated into daily clinical practice is yet to be determined.⁴⁴

MANAGEMENT STRATEGIES: CURRENT EVIDENCE AND EMERGING THERAPIES

Effective management of PPCM requires a coordinated, multidisciplinary team involving cardiologists, intensivists, obstetricians, neonatologists, anaesthetists, and cardiac surgeons. By integrating their expertise, this team works to optimise both maternal and fetal outcomes through comprehensive care. The European Society of Cardiology (ESC) guidelines on cardiovascular disease in pregnancy provide the framework for PPCM management, emphasising treatment strategies that are safe for both mother and baby.^{2,37,45,46}

Standard Heart Failure Treatment

PPCM treatment during pregnancy focuses on managing heart failure while ensuring fetal safety. Dietary sodium restriction and loop diuretics are first-line for the

management of fluid retention, and medications like beta-blockers, hydralazine/nitrates, and digoxin are generally considered safe. However, angiotensin converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARBs), angiotensin receptor-neprilysin inhibitors (ARNi), sodium-glucose co-transporter inhibitors (SGLT2i), and mineralocorticoid receptor antagonists (MRAs) are typically avoided due to potential risks to the fetus. Vaginal delivery is recommended for haemodynamically stable and epidural analgesia may be given during labour. Emergency caesarean section should be considered for patients who remain hemodynamically unstable despite optimal heart failure treatment and to prevent abrupt pressure or volume changes, epidural anaesthesia might be the method of choice.²

Post-delivery, medications such as ACEi/ARBs/ARNi, SGLT2i, beta-blockers and MRAs are recommended for patients with heart failure with reduced ejection fraction (HFrEF) for those patients who are not breastfeeding.² However, during breastfeeding, beta-blockers e.g metoprolol, ACEi like captopril or enalapril, and spironolactone are often considered safe, but careful assessment is needed, especially for preterm infants.⁴⁵

Discussion on contraception is very important before discharge, and oestrogen-containing contraceptives should be avoided, if possible, in the early postpartum period due to the risk of thromboembolism. Progesterone-releasing subcutaneous implants or intrauterine devices are safe and effective; tubal ligation and vasectomy are good options for those not considering future pregnancy.^{37,47}

The general opinion among experts is that patients with PPCM should continue standard heart failure medications for a minimum of 12 months, and lifelong therapy is recommended for those with persistently reduced LVEF. Those with full recovery of LV structure and function should continue medications for at least 6 months after full recovery before considering tapering down.² The best guide for those considering subsequent pregnancy is LVEF, as those with LVEF of > 50% are likely to better outcome.⁴⁸ Below are some of the novel medications specifically proven to be effective in the management of PPCM.

Pentoxifylline

Pentoxifylline, a xanthine derivative with phosphodiesterase inhibiting and anti-inflammatory properties, showed improved outcomes in PPCM patients exhibiting elevated inflammatory markers (TNF- α , CRP) when combined with standard heart failure therapy in early clinical investigations.⁴⁹ However, recent evidence challenges this benefit, indicating inconsistent therapeutic effects across contemporary studies.⁵⁰

Bromocriptine

Bromocriptine, a prolactin inhibitor, is an established targeted therapy for PPCM that counters prolactin-mediated cardiac injury. Clinical evidence confirms its efficacy: multiple studies report improved LVEF and outcomes with bromocriptine plus standard therapy, including one registry documenting a 96% recovery rate (LVEF \geq 50%) using low-dose regimens (2.5–5 mg/day). Safety data from over 100 patients show no major adverse events.^{51,52} Guidelines recommend initiating bromocriptine in severe cases (LVEF <25% or cardiogenic shock) with anticoagulation, following a tapered regimen (2.5 mg twice daily for 2 weeks, then 2.5

mg daily for 6 weeks). Key cautions include lactation suppression (necessitating patient counselling) and thrombotic risk (mandating anticoagulation). Further large-scale trials are needed to validate its role, optimise dosing, and confirm long-term safety.^{51,53}

Antisense Treatment to Combat MicroRNA-146a

A key theoretical advantage of antisense therapy targeting microRNA-146a is its compatibility with lactation, allowing nursing patients to continue breastfeeding, unlike some other treatments. While antisense oligonucleotides targeting miR-146a reduced systolic dysfunction in mouse models, this strategy remains untested in humans and failed to fully reverse PPCM pathology.⁵⁴

Vascular Endothelial Growth Factor (VEGF) Agonism and Removal of Anti-Angiogenic Proteins

Therapeutic apheresis targeting circulating soluble sFlt-1 has been explored in severe cases. In a documented clinical scenario, apheresis was utilised to remove sFlt-1 in a patient with refractory PPCM requiring continuous biventricular mechanical circulatory support.⁵⁵ Likewise, VEGF, an angiogenic factor counteracting sFlt-1's effects, has demonstrated therapeutic potential in murine PPCM models, restoring cardiac function by promoting angiogenesis.⁵⁶ However, VEGF-based interventions have not yet been translated to human trials, leaving their efficacy and safety in PPCM patients unestablished.

Prognosis and Maternal/Fetal Outcomes

PPCM has a very variable prognosis because most women regain left ventricular systolic function.⁵⁷ Others develop end-stage dilated cardiomyopathy and require either heart transplantation or advanced heart failure treatment, such as left ventricular assist devices (LVAD). Additionally, among PPCM patients who were thought to have recovered had abnormal echocardiogram features documented, either because of persistent arterial stiffness or residual global longitudinal strain.^{58,59}

A 2023 retrospective cohort study by Pachariyanon et al. reviewed 45 women with prior PPCM who had subsequent pregnancies.⁶⁰ Among those who had recovered LVEF ($\geq 50\%$) before the subsequent pregnancy, 23.3% experienced a relapse of PPCM.⁶⁰ In contrast, the relapse rate was 53.3% among women with persistent LV dysfunction. Moreover, the study reported a 20% all-cause mortality rate over a median follow-up of 8 years, with deaths occurring in both groups.⁶⁰ These findings underscore that LVEF recovery does not eliminate the risk of adverse outcomes in future pregnancies.^{60,61} In general, PPCM plays a major role in maternal morbidity and mortality.

A study from the ESC EORP registry examining 6-month outcomes in women with PPCM reported an overall mortality rate of 6%, with regional variations—4% in Europe and 10% in the Middle East. Most deaths were due to heart failure (42%) or sudden cardiac death (30%). Re-admissions occurred in 10% of patients, over half of which were due to heart failure. Thromboembolic events were reported in 7%. Myocardial recovery (LVEF $> 50\%$) was observed in 46% of cases, highest in the Asia-Pacific region (62%) and lowest in the Middle East (25%). Neonatal death occurred in 5% of cases, ranging from 2% in Europe to 9% in the Middle East.³⁸ It is, therefore, pertinent to acknowledge the fact that significant morbidity and mortality are associated with PPCM in both mothers and newborns.

PRIORITY AND INNOVATIONS IN THE MANAGEMENT OF PPCM

PPCM, with its complex and largely unclear pathogenesis, calls for focused research to uncover its genetic underpinnings, identify new therapeutic targets, and enhance patient outcomes.

Understanding the genetic basis of the disease.

This involves investigating the evolving role of genetics in PPCM, particularly in African populations and individuals of African descent where the condition is more prevalent or may be at greater risk.⁸ Though several studies have identified TTN truncations in familial clustering of PPCM^{62,63}, other commonly implicated mutations are in the sarcomeric genes MYBPC3, MYH6, MYH7, TNNC1 and TNNT2, especially in Dutch and American PPCM cohorts.⁶²⁻⁶⁵ As a stress-associated condition, heat shock protein (Hsps) mutations⁶² have also been identified in a PPCM model, suggesting that variation in these stress-response genes may contribute to PPCM pathogenesis. Areas of future investigation may include the screening of newly-identified cardiomyopathy genes for their roles in PPCM and correlating genetic information with phenotypic presentation and disease outcome. Genetic testing is of greater importance in African women with PPCM, where the prognosis is poorer⁵, and research on cardiomyopathy genetics is scarce⁶⁶.

Identifying novel therapeutic targets

Biomarker Discovery: PPCM, being a diagnosis of exclusion, can only be diagnosed after ruling out other causes of heart failure. This, in a way, delays the diagnosis, making the need for biomarkers to quickly identify PPCM from its mimics, such as hypertensive heart failure of Pregnancy, myocarditis, pre-existing dilated cardiomyopathy and pulmonary embolus.⁶⁷ Biomarkers ideally offer a non-invasive and easily measurable method for diagnosing a disease. Several biomarkers such as troponins, BNP, NT-proBNP, and mid-regional pro-atrial natriuretic peptides (MR-proANP) have presently been identified in heart failure.⁶⁸ Identifying specific biomarkers for early diagnosis and prognosis of PPCM will go a long way in mitigating the late detection and treatment, presently worsening the outcome. It is worth mentioning that biomarkers such as soluble suppression of tumorigenicity 2 (sST2), Galectin 3 (Gal-3), relaxin, VEGF, growth-differentiation factor-15 (GDF-15), adrenomedullin (ADM), long noncoding RNA, and heat shock proteins are currently under investigation for their clinical significance in routine medical practice.⁶⁹

Improving patient outcomes

There is still a significant unmet need for active research into potential therapeutic targets in PPCM management.⁷⁰ Without disease-specific, evidence-based data, the current therapeutic strategy for PPCM is similar to other forms of HF with left ventricular systolic dysfunction as directed by international guidelines.⁶⁶ To improve PPCM patients' outcome, a multi-disciplinary approach that involves cardiologists, obstetricians, intensivists, cardiac surgeons, anaesthesiologists, neonatologists, and nurses is crucial.⁴⁶ However, research towards developing tailored treatment plans based on individual patient characteristics, such as genetic profiles and biomarker expression using Stem Cell Therapy, is a priority.⁷⁰

Other innovative ways of improving PPCM patients' outcomes include the use of telemedicine and remote

monitoring, risk stratification using artificial intelligence-driven predictive models collaborative registries for data sharing and research.⁷¹

CONCLUSION

PPCM is a serious maternal health condition marked by an unpredictable course, high mortality, and a disproportionate burden on Black women and underserved populations. Despite advances in understanding hormonal, genetic, and vascular factors, leading to treatments like bromocriptine and standard heart failure therapies, its pathogenesis remains unclear. Outcomes vary: while about half of patients recover cardiac function, others suffer persistent cardiomyopathy, thromboembolic events, or death, especially in vulnerable groups. These disparities highlight systemic gaps in healthcare access and research representation. Future efforts must focus on uncovering genetic risk factors, validating early diagnostic biomarkers, and refining targeted therapies. Multidisciplinary care, patient education, and global registries are key to improving outcomes. Innovations such as AI-driven diagnostics and telemedicine offer hope for earlier detection in low-resource settings. Addressing racial and socioeconomic inequities, along with providing safe, long-term care options, is essential to ensure recovery extends beyond survival to lasting well-being.

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