

Review

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Peripartum cardiomyopathy: a clinical review

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Abstract

Peripartum cardiomyopathy (PPCM) is a potentially life-threatening form of heart failure that occurs in late pregnancy or the early postpartum period in previously healthy women. Characterized by left ventricular (LV) dysfunction and reduced LV ejection fraction (LVEF), PPCM shares pathophysiological features with other forms of dilated cardiomyopathy but presents unique challenges due to its association with pregnancy-related hormonal and vascular changes. While the exact etiology remains incompletely understood, oxidative stress, prolactin cleavage, inflammation, and genetic predisposition have been implicated in its pathogenesis. Diagnosis relies on echocardiographic evidence of systolic dysfunction in the absence of other identifiable causes of heart failure. Management strategies focus on optimizing heart failure therapy while considering the safety of both mother and fetus when treatment is initiated during pregnancy. Bromocriptine, an inhibitor of prolactin secretion, has emerged as a promising therapy, but its widespread use remains under investigation. Most patients experience significant recovery of cardiac function within six months postpartum; however, a subset progresses to chronic heart failure, transplantation, or death. This systematic review provides an in-depth analysis of current knowledge on the epidemiology, diagnostic challenges, and evolving therapeutic strategies for PPCM.

Keywords: Bromocriptine, heart failure, cardiovascular medicine, peripartum cardiomyopathy, women



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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is the leading cause of new-onset heart failure in pregnancy. However, pre-existing cardiomyopathies can also manifest or worsen due to the physiological changes associated with pregnancy. These changes can unmask previously undiagnosed cardiac diseases or precipitate the decompensation of subclinical cardiomyopathies^[1-3].

Diagnostic criteria and differential diagnosis

Accurately distinguishing PPCM from other cardiomyopathies guides appropriate management. PPCM is classically defined as an idiopathic cardiomyopathy presenting with heart failure due to systolic dysfunction^[3,4]. By contrast, dilated cardiomyopathy (DCM) is characterized by ventricular dilation and depressed systolic function; hypertrophic cardiomyopathy (HCM) by left ventricular (LV) hypertrophy (LVH), often without systolic impairment; arrhythmogenic right ventricular cardiomyopathy (ARVC) by fibro-fatty replacement of the right ventricular myocardium; LV non-compaction (LVNC) by prominent LV trabeculations; and restrictive cardiomyopathy (RCM) by increased myocardial stiffness leading to abnormal ventricular filling^[5]. Diagnosis generally requires careful clinical assessment, echocardiography, and sometimes cardiac magnetic resonance imaging to further elucidate structural abnormalities^[4].

Although PPCM is often highlighted as the most common form of cardiomyopathy during pregnancy^[6], the prevalence of other forms remains significant. DCM is relatively uncommon as a *de novo* condition in pregnancy, as most cases are diagnosed prior to conception. Recent studies indicate potential genetic similarities between DCM and PPCM, raising questions about whether they represent truly separate entities^[1,3,7]. HCM, while rare, can have notable maternal and fetal repercussions. Women with HCM may experience heart failure or arrhythmias, particularly in the third trimester or during the postpartum period^[3,8]. RCM is exceedingly rare but can lead to heart failure or arrhythmic complications due to the hemodynamic stress of pregnancy^[5]. ARVC and LVNC, though less common, must be considered in the differential diagnosis of new-onset heart failure during pregnancy^[9].

PERIPARTUM CARDIOMYOPATHY

Definition

PPCM presents as heart failure with a reduced LV ejection fraction (LVEF) (< 45%). It typically manifests at the end of pregnancy or within a few months postpartum, with no identifiable direct cause. Originally, PPCM was defined as developing between the last month of pregnancy and the fifth month postpartum. However, recent cases outside this timeframe suggest a more inclusive, time-independent definition^[10,11].

Epidemiology

PPCM incidence varies globally^[11-14]. The highest reported incidence is in Nigeria (1:100), followed by Haiti (1:299) and South Africa (1:1000)^[15]. In Western populations, the incidence is generally lower but has been steadily increasing. For example, in the USA, the rate increased from 1:2500 in 2004 to 1:1316 in 2011, while the latest data from Denmark reports an incidence of 1:10,000^[16-19]. Incidence rate differences may be due to variations in case reporting, genetic predisposition, environmental factors, and healthcare practices^[18]. The recent increase in U.S. incidence may be linked to rising maternal age and the growing prevalence of cardiovascular risk factors, such as hypertension and diabetes, among pregnant women^[15,20]. Moreover, a multicentric study conducted across 43 countries underscores that PPCM is a global disease, suggesting that its incidence may be underestimated in many regions^[21].

Pathophysiology

PPCM is a complex, multifactorial condition whose underlying mechanisms remain incompletely understood. Several potential etiologies have been proposed, including genetic predisposition, myocarditis

(both antigen-induced and viral: Echovirus, Coxsackie, and Parvovirus B19), aberrant immune responses to fetal antigens, maladaptive reactions to the physiological cardiovascular changes of pregnancy, angiogenic imbalance, hormonal disturbances, and cytokine activation resulting from stress and nutritional deficiencies such as selenium^[22-27].

Genetic Factors

Genetic factors play a significant role in PPCM pathogenesis. Studies have demonstrated notable similarities between PPCM and dilated cardiomyopathy^[27], particularly with respect to truncating variants in genes such as TTN, FLNC, BAG3, and DSP, with TTN variants being the most prevalent (observed in approximately 10% of patients). These TTN truncations result in the production of an abnormally shortened titin protein, which compromises the structural integrity and function of striated muscles, particularly in the heart and skeletal muscle^[28,29]. Despite this genetic overlap, in PPCM, the hormonal milieu of pregnancy and the specific role of prolactin (PRL) cleavage are unique to the condition. It is therefore recommended that genetic testing strategies for PPCM align with those used for dilated cardiomyopathy, as about 20% of patients undergoing genetic evaluation for cardiomyopathy harbor a pathogenic mutation^[7,18,29,30].

Other cardiomyopathies

Several cardiomyopathies have a well-established genetic predisposition. HCM is primarily associated with mutations in sarcomeric proteins, particularly MYH7 (myosin heavy chain 7) and MYBPC3 (myosin-binding protein C3). These mutations are responsible for a significant proportion of HCM cases, with MYH7 and MYBPC3 mutations accounting for approximately 40%-50% of cases, and lead to the characteristic features of HCM, including LVH, myocyte disarray, and myocardial fibrosis. Approximately 15% of PPCM patients harbor sarcomere gene mutations; however, it is important to note that while these mutations are present, they are not the primary feature of PPCM, which is more commonly associated with non-sarcomeric genes^[18,31]. On the other hand, arrhythmogenic cardiomyopathy is primarily associated with mutations in desmosomal proteins. In contrast, PPCM also exhibits desmosomal protein mutations-specifically in the *DSP* gene-but only in approximately 1.5% of cases, suggesting that while such mutations can occur in PPCM, they remain relatively uncommon^[29,32].

Angiogenic imbalance and the role of PRL

A key factor in the development of PPCM is the imbalance in angiogenesis mediated by PRL. Under conditions of oxidative stress, the standard 23-kDa PRL is cleaved into a 16-kDa isoform, which has been implicated as a primary pathogenic factor in PPCM^[33]. The 16-kDa PRL damages cardiomyocytes indirectly by inducing the release of microRNA-146a from endothelial cells, thereby activating the NF- κ B pathway. This angiogenic imbalance, coupled with microvascular dysfunction-stemming from endothelial cell apoptosis and subsequent capillary occlusion by apoptotic bodies-appears to be a critical nexus influenced by multiple PPCM risk factors^[15].

Response to stress and hemodynamic changes

Pregnancy induces significant hemodynamic alterations, including a reduction in afterload and increases in both cardiac output and blood volume. These changes can prompt structural and homeostatic remodeling of cardiovascular tissue, exacerbating cardiac stress. Furthermore, hormonal fluctuations associated with parturition may precipitate endothelial dysfunction, thereby contributing to the development of PPCM in susceptible individuals^[18].

Immune response

There is growing interest in the role of an autoimmune response in the development of PPCM. This view comes from findings of elevated antibody levels targeting specific cardiac tissues, suggesting that autoimmune myocarditis may underlie the condition. The proposed mechanism is linked to the immunosuppressed state of pregnancy, which allows maternal exposure to fetal antigens. Once the immune system rebounds postpartum, it may recognize these antigens as foreign and initiate an immune response, potentially contributing to PPCM. A study by Haghikia *et al.* demonstrated the presence of autoantibodies against cardiac troponin I and sarcomeric myosin in a significant proportion of PPCM patients. These autoantibodies were associated with more severe LV dysfunction and a lower rate of full cardiac recovery, indicating a potential role in disease progression^[34]. Similarly, Liu *et al.* found that autoantibodies against 1-adrenergic receptors and M2-muscarinic receptors were significantly elevated in PPCM patients compared to healthy controls, correlating with worse cardiac function and an increased risk of PPCM onset^[35].

Risk factors

Risk factors for PPCM include demographic (e.g., maternal age, ethnicity), genetic (e.g., TTN gene mutations), clinical (e.g., hypertension, obesity), and socioeconomic factors. Advanced maternal age (above 30 years) and very young maternal age (below 18 years) are associated with an increased risk. In terms of ethnicity, African women exhibit a higher incidence of PPCM^[29,33].

Women with a history of cancer treatment, particularly those who have received anthracycline chemotherapy or thoracic radiotherapy, are at heightened risk of developing cardiac insufficiency during pregnancy. This risk is even more pronounced in patients exposed to a cumulative doxorubicin dose of ≥ 250 mg/m² or thoracic radiation doses of ≥ 35 Gy^[36]. Anthracycline cardiotoxicity is largely driven by mitochondrial reactive oxygen species (ROS) generation through redox cycling and iron complexation, leading to cardiomyocyte apoptosis and dysfunction-features also seen in PPCM. Genetic predispositions affecting iron metabolism, such as Homeostatic Iron Regulator (High Fe, HFE) mutations, further increase susceptibility, highlighting a shared role of iron dysregulation in both conditions. More recently, topoisomerase II β (Top2 β), found in non-dividing cells like cardiomyocytes, has emerged as a key mediator. Anthracycline-top2 β complexes cause DNA damage and suppress mitochondrial regulators like PGC-1 α and MnSOD, impairing energy metabolism and antioxidant defenses-mechanisms also relevant to PPCM. Anthracyclines also inhibit STAT3, a crucial stress-response pathway. Similarly, STAT3 disruption is implicated in PPCM, particularly in PRL-mediated endothelial damage, suggesting overlapping molecular vulnerabilities. Anthracyclines interfere with cardiac growth and angiogenesis via suppression of the vascular endothelial growth factor (VEGF) pathway, which mirrors the angiogenic imbalance seen in PPCM. These effects can be amplified by concurrent ErbB2 inhibitors like trastuzumab, which are independently linked to peripartum heart failure^[37].

Socioeconomic determinants play significant roles in the development and progression of PPCM. A recent study found that higher levels of education are paradoxically associated with an increased risk of cardiomyopathy, possibly due to delayed childbearing and the resulting higher maternal age^[38]. Mental health problems, including stress and depression, may influence overall health and adherence to prenatal care. Additionally, lower socioeconomic status is associated with poorer maternal and neonatal outcomes in PPCM, including higher mortality rates and reduced use of guideline-directed heart failure therapies, particularly in countries with lower health expenditures and human development indices^[39]. Additional factors include multiple pregnancies and obesity, both of which contribute to an elevated risk. Hypertensive disorders, including hypertension and preeclampsia, are significant risk factors; indeed, 38% of women with PPCM have a history of pregnancy-related hypertensive conditions^[33,40]. Other risk factors include genetic

predispositions, such as truncating mutations in the TTN gene, prolonged use of beta-agonists, smoking, and a family or personal history of PPCM^[41]. Effective risk management should involve a thorough evaluation of these factors, along with careful monitoring during pregnancy and the postpartum period^[42].

Diagnosis

The diagnosis of PPCM is based on a combination of clinical criteria, imaging exams, and the exclusion of other known causes of cardiac insufficiency^[43-45].

Timing and clinical presentation

The first month peripartum is when most women are diagnosed with PPCM^[46]. Due to the under-recognition of this disease and the overlap between generalized symptoms of normal pregnancy and those of heart failure, delays in diagnosis occur, leading to an increased incidence of complications and worse outcomes^[46,47]. PPCM typically presents with symptoms of heart failure, such as fatigue, dyspnea, orthopnea, edema, and nocturnal cough^[44].

Absence of other causes

The diagnosis of PPCM is established in the absence of other identifiable causes of heart failure. It is essential to rule out pre-existing heart disease, valvular disorders, or coronary artery disease. A key diagnostic criterion is the presence of LV systolic dysfunction, defined by a LVEF of less than 45% in the last month of pregnancy or in the months following delivery^[13].

Echocardiography

Echocardiography is the primary imaging tool for diagnosing PPCM. It allows for the assessment of LVEF, LV size, wall motion abnormalities, and other structural changes. The examination can also reveal LV dilation and global hypokinesis. Additionally, echocardiography evaluates right ventricular involvement, chamber dimensions, and the presence of mitral or tricuspid regurgitation. Importantly, it helps exclude LV apical thrombus^[48,49], which should always be ruled out in cases of severe LVEF reduction. Hibbard *et al.*^[50] established stringent echocardiographic criteria for diagnosing PPCM, which include LVEF < 45%, fractional shortening under 30%, and a LV end-diastolic dimension exceeding 2.7 cm/m². However, while systolic dysfunction is essential for the commonly accepted diagnosis of PPCM, LV enlargement is not a required criterion.

Additional diagnostic tests

Electrocardiogram (ECG) findings may be nonspecific, including atrial and ventricular arrhythmias and other electrocardiographic abnormalities (LVH, sinus tachycardia, and nonspecific ST-T abnormalities), which are common in these patients^[51-53]. However, a normal ECG does not rule out PPCM^[54].

Biomarkers

Levels of natriuretic peptides (BNPs or NT-proBNPs) are often elevated in PPCM, aiding in differentiation from normal pregnancy-related changes; however, these levels can also be mildly elevated during preeclampsia, requiring cautious interpretation^[55,56]. Chest radiograph should also be performed as part of the initial diagnostic workup for suspected PPCM, to exclude other potential causes of the patient's symptoms such as pneumonia, pulmonary embolus, or other acute lung injuries^[51,53].

Cardiac Magnetic Resonance (CMR) provides additional insights into myocardial tissue characterization and right ventricular involvement, which have prognostic implications. However, gadolinium-based contrast agents are not recommended during pregnancy and should only be considered in the postpartum

period; biopsy is generally not required and should only be considered if other conditions requiring specific treatment are suspected^[43]. Genetic testing is recommended in patients with PPCM to identify potential genetic causes and facilitate risk stratification and management^[57].

MANAGEMENT

PPCM management involves a multidisciplinary team of cardiologists, obstetricians, intensivists, and pediatricians, focusing on maternal and fetal well-being^[7].

PPCM stratification: a severity-based approach

Management is individualized based on the patient's clinical presentation and diagnostic evaluation, enabling stratification into one of three clinical categories defined by the European Society of Cardiology (ESC) Study Group on PPCM^[13]. Mild PPCM is characterized by subacute heart failure with stable hemodynamics and an LVEF of 30% to 45%; moderate PPCM presents with significant heart failure symptoms while maintaining hemodynamic stability, with an LVEF of 20% to 35%; and severe PPCM involves cardiogenic shock, hemodynamic instability, and respiratory failure, with an LVEF below 20%^[13]. Treatment strategies are tailored according to disease severity, as outlined in Figure 1. Generally, mild cases of PPCM are managed in a normal ward or on an outpatient basis. Moderate cases are typically admitted to an intermediate care unit or a heart failure unit (HFU), while severe cases require admission to an intensive care unit (ICU)^[13].

	CLINICAL PRESENTATION	NATRIURETIC PEPTIDES	ECHOCARDIOGRAPHY	TREATMENT
MILD PPCM	Subacute heart failure Hemodynamic stability	Mildly elevated	LVEF 35-45%	1) Oral HF medications 2) Oral diuretics if needed 3) Consider bromocriptine 1 week
MODERATE PPCM	Acute heart failure Hemodynamic stability	Moderately elevated	LVEF 20-35%	1) Oral HF medications 2) Intravenous diuretics 3) NIV if needed 4) Consider vasorelaxants 5) Consider anticoagulation if LVEF < 25% 5) Consider Bromocriptine 8 weeks if LVEF < 25%
SEVERE PPCM	Cardiogenic shock Hemodynamic instability	Severely elevated	LVEF < 25% possible RV dysfunction	1) Intravenous diuretics 2) Inotropes/catecholamines 3) Invasive ventilation 4) MCS 5) Anticoagulation 6) Consider Bromocriptine 8 weeks 7) Oral HF medications

Figure 1. Management of peripartum cardiomyopathy according to disease severity. LVEF: Left ventricular ejection fraction; HF: heart failure; MCS: mechanical circulatory support; NIV: non-invasive ventilation; PPCM: peripartum cardiomyopathy.

The management of PPCM may also depend on the severity of cardiopulmonary distress, which can necessitate urgent delivery via cesarean section. A proposed management algorithm is outlined in Figure 2, illustrating how treatment options vary based on the presence or absence of cardiopulmonary distress^[58].

Acute care strategy - management of hemodynamically and cardiopulmonary distressed patients

Prompt initial evaluation should confirm the diagnosis through ECG, natriuretic peptides, and echocardiography, and assess cardiopulmonary distress, typically defined by systolic blood pressure (SBP) < 90 mmHg, heart rate (HR) > 130/min or < 45/min, respiratory rate (RR) > 25/min, peripheral capillary oxygen saturation (SpO₂) < 90%, lactate > 2.0 mmol/L, central venous SpO₂: oxygen saturation (SpO₂ < 60%), altered mental state, cold skin, and oliguria (< 0.5 ml/kg/min)^[14,59].

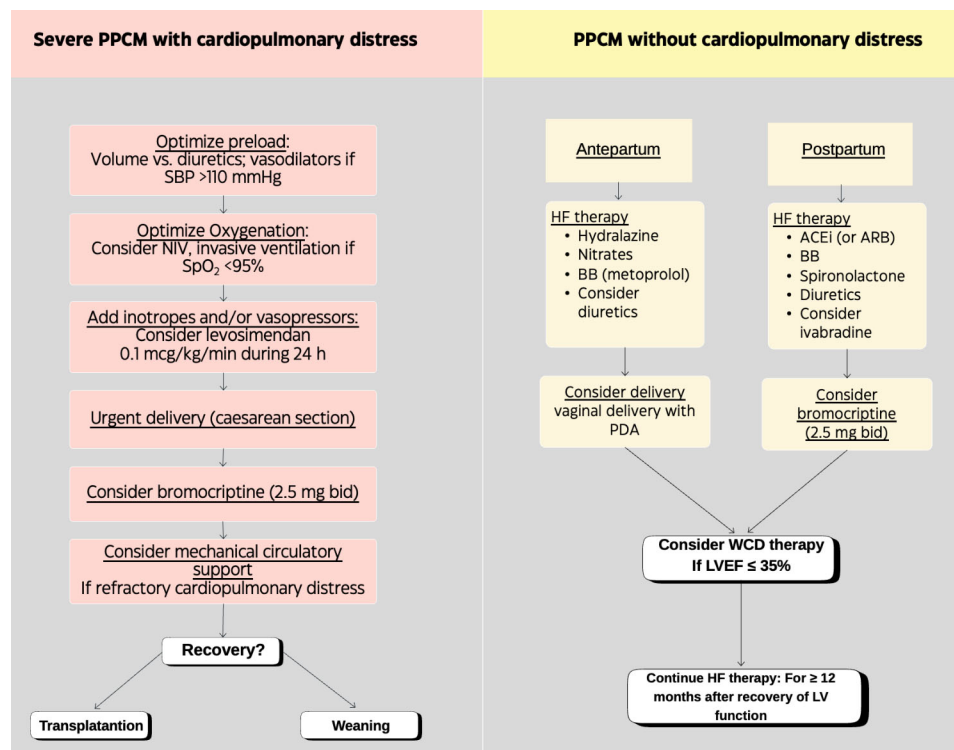


Figure 2. Algorithm for initial management. BB: Beta-blocker; HF: heart failure; HR: heart rate; NIV: non-invasive ventilation; PDA: peridural anesthesia; RR: respiratory rate; SBP: systolic blood pressure; SpO₂: peripheral oxygen saturation; WCD: wearable cardioverter-defibrillator; LVEF: left ventricular ejection fraction; PPCM: peripartum cardiomyopathy; ACEi: inhibitors of angiotensin converting enzyme; ARB: angiotensin receptor blockers.

The initial treatment approach includes five essential components: preload optimization, oxygenation optimization, hemodynamic stabilization using inotropes and/or vasopressors, urgent delivery in cases of prepartum heart failure, and adjunctive therapy with bromocriptine (2.5 mg twice daily for two weeks, followed by 2.5 mg once daily for six weeks).

Bromocriptine is a strong activator of the transmembrane G-protein-coupled dopamine D2 receptor and multiple serotonin receptors in the central nervous system used in PPCM for its ability to block PRL release from the pituitary gland^[60]. Since thromboembolic events have been reported with bromocriptine use, particularly at higher doses, its administration should always be accompanied by at least prophylactic anticoagulation with heparin^[61].

Preload management varies depending on the clinical condition and may involve fluid administration or diuretics. In the absence of overt fluid overload, a fluid challenge of 250-500 mL over 15-30 min is recommended, especially in patients with intravascular depletion caused by peripartum blood loss or excessive diuretic therapy. If signs of congestion are present, intravenous diuretics should be administered. For patients with systolic blood pressure exceeding 110 mmHg, intravenous vasodilators such as nitrates should be initiated. Patients in cardiogenic shock or reliant on inotropes should be promptly transferred to an advanced heart failure center equipped with mechanical circulatory support (MCS), ventricular assist devices (VADs), and specialized transplant consultation teams^[58]. For hemodynamically stable patients, vaginal delivery with epidural anesthesia is the preferred approach.

Management: emergency, refractory or advanced PPCM

In women with severe heart failure or ongoing hemodynamic instability despite medical management, urgent cesarean delivery should be considered regardless of gestational age^[58,62]. In cases of advanced or refractory PPCM, MCS and implantable cardioverter-defibrillators (ICDs) play a crucial role in management. MCS is indicated in cases of cardiogenic shock or severe heart failure unresponsive to medical therapy, often in patients classified as INTERMACS profile 1 or 2. Short-term support devices such as intra-aortic balloon pumps, Impella, or veno-arterial extracorporeal membrane oxygenation (VA-ECMO) are utilized to stabilize hemodynamics and preserve end-organ function, serving as a bridge to recovery or further decision making. In patients who fail to recover, durable LV assist devices (LVADs) may be considered as a bridge to transplantation or as destination therapy. Notably, some PPCM patients supported with LVADs demonstrate meaningful myocardial recovery that may permit device explantation-an outcome less common in other forms of cardiomyopathy. The American College of Cardiology recommends durable LVAD implantation in select patients with advanced heart failure with New York HEart Association (NYHA) class IV symptoms who are dependent on continuous intravenous inotropes or temporary MCS. The risk of sudden cardiac death (SCD) in PPCM is also significant in the setting of persistent LV dysfunction. However, given the potential for recovery, early permanent ICD implantation is generally deferred in favor of wearable cardioverter-defibrillators (WCDs) for temporary protection during the initial months following diagnosis. If LV function remains depressed beyond six months despite optimal therapy, permanent ICD implantation is considered appropriate^[32,63,64]. Cardiac transplantation is another viable option for patients with PPCM who do not recover with medical therapy or mechanical support. Transplantation can provide a definitive solution for those with end-stage heart failure.

Long term management and therapeutics

The approach to treatment differs based on whether the patient is pregnant or postpartum. After delivery, concerns regarding fetal stability, maturity, and potential teratogenic effects of medications are no longer relevant, allowing for the initiation of standard heart failure therapies^[8,36,62]. In pregnant patients, it is crucial to avoid medications with teratogenic potential, including angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and renin inhibitors, as well as mineralocorticoid receptor antagonists, atenolol, and direct factor Xa inhibitors^[36,62,65]. Instead, treatment primarily relies on hydralazine, nitrates, and beta-blockers^[62]. Despite the increased risk of fetal growth restriction, beta-blockers are recommended for all stable patients, with metoprolol succinate being the preferred option^[66,67]. Vasodilators-including hydralazine, amlodipine, and nitroglycerin-are considered safe, whereas nitroprusside should be avoided due to the risk of cyanide toxicity. Early delivery is not required if maternal and fetal conditions remain stable, and most patients can safely undergo vaginal delivery. Diuretics (such as furosemide and hydrochlorothiazide) should be used judiciously because they may reduce placental perfusion and potentially distress the fetus; these agents are best reserved for symptomatic relief of pulmonary or peripheral edema^[68].

Lifestyle modifications in the prevention and management of PPCM

Lifestyle changes play a crucial role in both preventing and managing PPCM. The Heart Failure Association of ESC emphasizes the importance of regular physical activity, adapted to each patient's clinical status and tolerance. While light to moderate exercise can offer cardiovascular benefits, strenuous activity should be avoided-particularly during the acute phase of PPCM. Nutritional guidance is equally essential. A heart-healthy diet-low in sodium and rich in fruits, vegetables, and whole grains-can help control fluid retention and support overall cardiovascular function. Patients should also limit excessive fluid intake to avoid volume overload. Weight management is another key consideration. Achieving and maintaining a healthy weight through balanced nutrition and appropriate physical activity can reduce cardiac strain, which is especially important given the natural weight fluctuations during pregnancy and the postpartum

period. Stress management strategies may help alleviate symptoms. Chronic stress can worsen heart failure, making emotional well-being an important component of care. Avoiding smoking and alcohol is critical. Both are known to adversely affect cardiovascular health and should be eliminated entirely in patients with or at risk for PPCM. Lastly, educating patients to recognize early signs of heart failure, including increased breathlessness, swelling, or fatigue, is vital. Prompt recognition and medical attention can help prevent serious complications and improve outcomes ^[13,32,68,69].

Anticoagulation

All patients with acute PPCM and severely reduced LV systolic function ($LVEF \leq 35\%$) should receive anticoagulation with heparin due to the hypercoagulable state associated with pregnancy and the early postpartum period^[70]. In cases of severely reduced LVEF during late pregnancy and up to 6-8 weeks postpartum, anticoagulation should be considered. The American Heart Association recommends anticoagulation when LVEF is $< 30\%$, while the ESC sets the threshold at $LVEF \leq 35\%$. Additionally, anticoagulation is recommended when using bromocriptine due to its associated thrombotic risk^[62]. While warfarin and novel anticoagulants are generally avoided during pregnancy, low-molecular-weight heparin is preferred, as it does not cross the placenta, unlike warfarin. However, during lactation, both warfarin and low-molecular-weight heparin are considered safe^[71].

PROGNOSTIC FACTORS AND MORTALITY

LVEF at the time of the diagnosis

One of the most significant prognostic factors identified is LVEF at the time of diagnosis. An initial LVEF of less than 30% predicts a lower likelihood of recovery^[72]. Other factors predicting adverse outcomes and a lower likelihood of full recovery include: right ventricular dysfunction, African ancestry, LV dilation^[73,74], LV thrombus, older age, late diagnosis, and elevated inflammatory markers^[72,75], as well as biomarkers such as troponin^[76], NT-proBNP^[77], and sFlt1^[78]. Elevated Relaxin-2 levels have been linked to a faster restoration of systolic function^[78]. The presence of concomitant preeclampsia has been linked to reduced one-year survival^[79]; however, among those who survive, it is associated with improved LV recovery.

Mortality

The prognosis of PPCM varies significantly, with differing morbidity and mortality rates influenced by both geographic and racial factors. All-cause mortality in PPCM is a significant concern, with an estimated rate of 8.0% at 6 months and 9.8% at 12 months. These outcomes demonstrate marked regional disparities, with the highest 6-month mortality observed in Asia/Pacific (11.5%) and Africa (10.9%), and the lowest in Europe (0.7%). At 12 months, mortality remained highest in Africa (15.2%) and Asia/Pacific (13.6%), while Europe and North America reported considerably lower rates at 2.3% and 5.1%, respectively. Studies that reported high prescription rates of guideline-directed medical therapies, including beta-blockers and ACE inhibitors or ARBs, were associated with significantly lower mortality rates^[80]. In African American women, mortality rates are significantly higher, reaching 11% in a population composed of 96% African Americans^[81], compared to other ethnic groups. Furthermore, compared to the offspring of healthy moms, children born to women with PPCM had a three-fold higher incidence of heart disease and a five-fold rise in mortality rates at long-term follow-up^[82].

Fetal outcomes

According to the ESC EuroObservational Research Programme Peri-Partum Cardiomyopathy Registry, fetal outcomes in pregnancies complicated by PPCM include a high incidence of preterm delivery (24%) and low birth weight (20%). Neonatal mortality was reported at 3% in this cohort^[83]. A systematic review and meta-analysis found that pregnancies affected by maternal cardiomyopathy, including PPCM, had significantly higher odds of perinatal death, preterm birth, and small-for-gestational-age neonates compared

to pregnancies without cardiac disease or with non-cardiomyopathy cardiac disease. Specifically, the odds ratios for stillbirth and neonatal mortality were markedly elevated, indicating a substantial risk for adverse perinatal outcomes^[42]. Furthermore, a population-based study reported that infants born to mothers with PPCM had lower birth weights and were more likely to be small for gestational age compared to those born to mothers with other forms of cardiomyopathy^[84]. Managing maternal cardiac health is fundamental to improving fetal outcomes in PPCM. Effective management of maternal cardiac function can mitigate risks such as preterm delivery, low birth weight, and neonatal mortality.

POSTPARTUM RECOMMENDATIONS

Contraceptives

After delivery, women with recent PPCM should receive counseling about contraceptive use^[85]. During the early postpartum phase, women with significant LV dysfunction face an increased risk of thromboembolism, making estrogen-containing contraceptives inadvisable^[86].

Breastfeeding

After childbirth, acute and chronic heart failure in women with PPCM who are not breastfeeding should be treated with standard guideline-directed medical therapy (GDMT)^[87]. In clinically stable women, breastfeeding is encouraged because of its documented benefits. The PPCM Study Group of the Heart Failure Association recommends regular follow-up every six months with echocardiography until LVEF recovers to above 50%. Although no formal guidelines exist regarding discontinuation of therapy once LVEF has normalized, some experts advocate lifelong treatment, given the ongoing risk of heart failure and LV dysfunction even without further pregnancies^[85]. If therapy is stopped, close clinical and cardiac monitoring is essential. Any subsequent pregnancy carries a risk of further deterioration in cardiac function, underscoring the importance of multidisciplinary evaluation. Currently, ACE inhibitors, angiotensin II receptor blockers, diuretics, beta-blockers, mineralocorticoid receptor antagonists, and sacubitril/valsartan should generally be avoided during breastfeeding^[88]. However, certain ACE inhibitors, such as benazepril, captopril, and enalapril, have been adequately studied in breastfeeding women and are considered safe for infants when used by the mother^[89]. In a recent prospective multicenter study of 100 women with PPCM, elevated PRL levels and increased cytotoxic T cell activity were observed in those who continued breastfeeding, without negatively impacting myocardial recovery. Indeed, women who breastfed tended to have higher baseline LVEF and showed outcomes comparable to their non-breastfeeding counterparts at 6 and 12 months postpartum. These findings corroborate the current stance that stable patients can safely breastfeed without compromising cardiac function, although individualized decisions and close clinical follow-up remain paramount^[89].

Subsequent pregnancies

Recurrence

The risk of recurrent PPCM in a subsequent pregnancy ranges from 30% to 50%, depending on the degree of LVEF recovery^[90,91]. The ESC's Task Force on the Management of Cardiovascular Diseases during Pregnancy advises against future pregnancies if LVEF has not returned to pre-pregnancy levels. However, even when normalization occurs, counseling remains essential due to the risk of recurrence in future pregnancies^[66]. Although it varies, there is generally a chance of relapse in every case. Other definitions of PPCM recurrence include a drop in LVEF of less than 45% in patients with LVEF of more than 50%, an absolute decrease in LVEF of more than 10% in patients with LVEF of less than 50%^[92], or an absolute decrease of more than 20% independent of baseline LVEF^[93]. It is advised that women have echocardiograms every six months until their LVEF returns to greater than 50%, and that those who stay stable following the tapering of medication therapy for heart failure have yearly examinations for ten years^[13] (see Figure 3).

PHASE / CATEGORY	KEY DETAILS AND RECOMMENDED ACTIONS
ANTEPARTUM MANAGEMENT	Medications: Hydralazine, nitrates (for afterload reduction) Beta-blockers (if indicated/tolerated) Diuretics (for volume overload) Hospitalization, if needed, for close monitoring and management. Determine the mode of delivery based on maternal hemodynamic stability.
POSTPARTUM MANAGEMENT	Standard heart failure therapy once no longer pregnant: ACE inhibitors (or ARB/ARNI), beta-blockers, mineralocorticoid receptor antagonists, diuretics as indicated. Peripartum Cardiomyopathy (PPCM): Consider bromocriptine to block PRL (adjunct therapy). Wearable cardioverter-defibrillator (WCD) if LVEF $\leq 35\%$.
DELIVERY & CONTRACEPTION	In unstable HF, emergent cesarean delivery may be necessary. In stable patients, vaginal delivery is often feasible. Counsel regarding effective contraception and future pregnancy risks.

Figure 3. Management of heart failure in pregnancy. ACE: Angiotensin-converting enzyme; PRL: prolactin; HF: heart failure; ARB: angiotensin receptor blockers; ARNI: angiotensin receptor-neprilysin inhibitors.

Ongoing monitoring

The American Heart Association recommends close clinical follow-up with annual assessment of LVEF for a minimum of several years after recovery, particularly if subsequent pregnancy is being considered. Monitoring enables timely adjustments in heart failure management, which is critical given the high risk of relapse and complications such as arrhythmias, thromboembolic events, and persistent heart failure. The Investigations of Pregnancy-Associated Cardiomyopathy (IPAC) study highlighted that severe LV dysfunction and greater remodeling at presentation are associated with less recovery, underscoring the need for vigilant follow-up to identify patients at higher risk for adverse outcomes^[19,72,94].

EXPERIMENTAL TREATMENTS

Selenium

Multiple studies have suggested that a deficiency in selenium and other micronutrients may contribute to the development of PPCM^[112]. A recent study suggested that selenium deficiency is a highly prevalent risk factor for PPCM among patients in Kano, Nigeria, and is significantly associated with increased odds of developing the condition^[95]. In a randomized, open-label proof-of-concept trial of PPCM patients with LV systolic dysfunction and selenium deficiency, selenium supplementation did not significantly reduce the primary composite endpoint of persistent heart failure symptoms, persistent LV systolic dysfunction, or death from any cause. Nonetheless, selenium supplementation was associated with a significant improvement in symptoms and a trend toward lower all-cause mortality. Although there was no significant difference in the secondary endpoint of all-cause re-hospitalizations, the observed trend also favored the

selenium-supplemented group^[96].

PRL inhibition and the role of bromocriptine

A growing body of evidence supports the critical role of the 16 kDa N-terminal fragment of PRL (16kDa PRL) in PPCM pathogenesis, making inhibition of PRL a promising therapeutic avenue^[97]. The heightened oxidative stress characteristic of the peripartum period can cleave the larger 23kDa PRL into 16kDa PRL via cathepsin-D, producing a potent anti-angiogenic and pro-apoptotic peptide. By disrupting angiogenesis and promoting endothelial cell apoptosis, 16kDa PRL contributes significantly to cardiac dysfunction. Accordingly, pharmacological inhibition of PRL release, such as with bromocriptine, may reduce the generation of 16kDa PRL and mitigate its deleterious effects. For this reason, bromocriptine, a dopamine D2 agonist, has been investigated for its potential benefits in PPCM. A systematic review involving 593 PPCM patients across eight studies found that bromocriptine treatment was associated with significantly higher survival rates and greater improvement in LVEF. However, no association was observed with a lower incidence of composite adverse clinical outcomes or LVEF recovery; while baseline LVEF did not differ significantly between groups, follow-up measurements showed a significantly higher LVEF in the bromocriptine group (53.3% vs. 41.8%, $P < 0.001$)^[98]. Nevertheless, the 2018 ESC guidelines provide a cautious endorsement (Class IIb, Level of Evidence: B) for the use of bromocriptine^[62], likely due to inconsistent clinical benefits reported across multiple studies^[99]. By restoring angiogenic balance, PRL inhibition may help curb disease progression, underscoring its potential as an adjunctive therapy for patients with PPCM^[100].

CONCLUSIONS

PPCM exhibits overlapping mechanisms with other types of dilated cardiomyopathy but poses distinct clinical complexities due to its linkage with pregnancy-induced hormonal and vascular shifts. Although its precise origin remains partially elusive, contributors such as oxidative injury, cleavage of PRL, inflammatory pathways, and inherited susceptibility have been associated with disease development. Diagnosis hinges on echocardiographic confirmation of systolic impairment without other discernible causes of cardiac failure. Treatment approaches prioritize the stabilization of heart failure while accounting for maternal and fetal safety when initiated during pregnancy. Bromocriptine, which blocks PRL release, has shown therapeutic potential, though its routine clinical application is still being evaluated. Many patients demonstrate substantial improvement in cardiac function within six months following delivery; nonetheless, a minority advance to persistent heart failure, require heart transplantation, or succumb to the disease. Further research is warranted to achieve a complete understanding of its underlying mechanisms in order to advance treatment modalities.

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Authors' contributions

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Not applicable.

Consent for publication

Not applicable.

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