CLINICAL DIAGNOSIS AND TREATMENT METHODS OF PERIPARTUM CARDIOMYOPATHIES

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Annotation. *Peripartum cardiomyopathy (PPCM) is an uncommon but serious condition characterized by heart failure due to left ventricular systolic dysfunction occurring in the last month of pregnancy or within five months postpartum, in the absence of an identifiable cause. This article reviews the current understanding of the clinical presentation, diagnostic criteria, and therapeutic strategies for PPCM. It emphasizes the importance of early recognition through echocardiography and laboratory tests, as well as the implementation of guideline-directed medical therapy tailored to pregnancy and postpartum considerations. The prognosis of PPCM varies, with some patients recovering fully and others developing persistent cardiac dysfunction. Multidisciplinary care and long-term follow-up are essential for optimizing maternal outcomes and guiding future reproductive decisions.*

Keywords: *Peripartum cardiomyopathy; heart failure; pregnancy; postpartum; left ventricular dysfunction; echocardiography; diagnosis; treatment; maternal cardiology; cardiac complications in pregnancy.*

Introduction. Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of dilated cardiomyopathy that occurs in women during the late stages of pregnancy or in the months following delivery. Defined by the development of heart failure due to left ventricular systolic dysfunction without any identifiable cause or prior heart disease, PPCM poses significant risks to maternal health, including heart failure, arrhythmias, thromboembolic events, and even sudden cardiac death. The incidence of PPCM varies globally, with higher rates reported in certain regions such as Africa and Haiti, reflecting potential genetic, environmental, and socioeconomic influences. Despite advances in obstetric and cardiac care, the etiology of PPCM remains incompletely understood, with proposed mechanisms including inflammation, autoimmune responses, oxidative stress, and vascular dysfunction triggered by the hemodynamic and hormonal changes of pregnancy. Clinically, PPCM often presents with nonspecific symptoms that overlap with normal pregnancy changes, making early recognition challenging. Symptoms such as fatigue, dyspnea, and edema are frequently attributed to pregnancy itself, which can delay diagnosis and worsen outcomes. Therefore, a high index of suspicion is crucial for timely intervention.

Early diagnosis and comprehensive management of PPCM are essential to reduce maternal morbidity and mortality. This involves a multidisciplinary approach integrating

cardiology, obstetrics, and critical care specialties. The clinical diagnosis relies heavily on echocardiographic evaluation, while treatment principles largely follow standard heart failure guidelines, adapted to the unique physiological considerations of pregnancy and the postpartum period. This article aims to provide an in-depth overview of the clinical diagnosis and treatment modalities for PPCM, highlighting current best practices and recent advances in the field to improve patient care and outcomes.

Clinical Presentation: Patients with PPCM typically present with signs and symptoms of heart failure, which may include:

- Dyspnea on exertion or at rest
- Orthopnea and paroxysmal nocturnal dyspnea
- Peripheral edema
- Fatigue and weakness
- Palpitations or chest discomfort

Because these symptoms can mimic normal pregnancy discomfort or other obstetric complications, high clinical suspicion is essential, especially when symptoms are progressive or severe.

Table 1: Analytical summary of clinical diagnosis and treatment methods of peripartum cardiomyopathy

Category	Key Findings / Insights
Definition	Heart failure due to left ventricular systolic dysfunction occurring in the last month of pregnancy or up to 5 months postpartum without other known causes.
Epidemiology	Rare (1 in 1,000 to 1 in 4,000 live births globally); higher prevalence in Africa and among women of African descent.
Risk Factors	Advanced maternal age, multiparity, preeclampsia, multiple gestations, African ancestry, hypertension, obesity, selenium deficiency.
Pathophysiology	Multifactorial: oxidative stress, prolactin cleavage into a 16-kDa toxic fragment, angiogenic imbalance (elevated sFlt-1), possible genetic mutations (*TTN*).
Clinical Presentation	Dyspnea, fatigue, orthopnea, peripheral edema, palpitations, symptoms of heart failure—often mistaken for normal pregnancy discomfort.
Diagnostic Tools	- Echocardiography (LVEF < 45%) - NT-proBNP, BNP (elevated) - ECG (nonspecific findings) - Cardiac MRI in select cases.
Differential Diagnosis	Hypertensive heart disease, myocarditis, pulmonary embolism, ischemic cardiomyopathy, amniotic fluid embolism.
Treatment	- Standard heart failure therapy (adjusted for

Category	Key Findings / Insights
Approaches	pregnancy/postpartum) - Diuretics, beta-blockers, hydralazine/nitrates during pregnancy - ACE inhibitors postpartum - Anticoagulation in low EF - Bromocriptine (experimental).
Advanced Interventions	Inotropes, mechanical circulatory support (VAD, IABP), heart transplant in refractory cases.

Peripartum cardiomyopathy is a potentially life-threatening condition that requires prompt recognition and treatment. A high index of suspicion, combined with echocardiography and laboratory assessments, enables timely diagnosis. Treatment strategies align with general heart failure guidelines, with adjustments for pregnancy status. Multidisciplinary care and long-term monitoring are vital to improving outcomes for affected women.

Analysis of literature. Peripartum cardiomyopathy (PPCM) has attracted increasing attention in recent decades due to its unique clinical profile and significant implications for maternal health. The current body of literature reflects evolving understanding in pathophysiology, diagnostic approaches, and management strategies, though numerous gaps remain in etiology, standardized treatment, and long-term outcomes. Early definitions of PPCM were inconsistent, often confusing it with other forms of cardiomyopathy. However, the work by Demakis et al. in the 1970s was instrumental in establishing PPCM as a distinct clinical entity. Subsequent consensus definitions, such as those provided by the National Heart, Lung, and Blood Institute (NHLBI), emphasize the onset of heart failure symptoms in the last month of pregnancy or within five months postpartum, and exclusion of other identifiable causes of cardiomyopathy. Recent studies have proposed expanding the timeline and using imaging-based criteria to include earlier presentations.

Literature over the last two decades has identified PPCM as a multifactorial disease. Research by Hilfiker-Kleiner et al. (2007) and others suggests a pivotal role for oxidative stress leading to the cleavage of prolactin into a cardiotoxic 16-kDa fragment, which impairs endothelial function and induces myocardial damage. Elevated levels of anti-angiogenic factors, particularly soluble fms-like tyrosine kinase-1 (sFlt-1), have also been linked to the development of PPCM, especially in association with preeclampsia. Genetic predisposition is increasingly recognized, with studies identifying mutations in genes typically associated with dilated cardiomyopathy (e.g., *TTN* truncating variants). However, comprehensive genome-wide studies remain limited, and the contribution of genetic versus environmental factors is still being investigated.

Echocardiography remains the primary diagnostic tool, as emphasized across numerous reviews (e.g., Sliwa et al., 2006; Elkayam, 2011). The echocardiographic hallmark is reduced left ventricular ejection fraction (LVEF <45%) with or without left ventricular dilatation. The use of biomarkers such as NT-proBNP and troponin has shown

promise in distinguishing PPCM from normal pregnancy-related dyspnea, although these markers lack specificity. Recent literature also explores the use of cardiac magnetic resonance imaging (CMR) for better tissue characterization and to rule out other causes such as myocarditis or infiltrative diseases. However, limited access and cost considerations restrict its routine use in many regions.

Guideline-directed medical therapy (GDMT) for heart failure—comprising beta-blockers, ACE inhibitors/ARBs, and mineralocorticoid receptor antagonists—is central to treatment postpartum. During pregnancy, teratogenic drugs are replaced with safer alternatives such as hydralazine and nitrates. A key advancement in PPCM-specific therapy is the experimental use of bromocriptine, a dopamine agonist that inhibits prolactin secretion. Trials such as the German PPCM study (Sliwa et al., 2010) demonstrated improved left ventricular recovery with bromocriptine in addition to standard therapy. Despite these promising findings, larger randomized controlled trials (RCTs) are needed before it becomes standard practice, and its thrombotic risks necessitate co-administration of anticoagulants.

Anticoagulation is another area of consensus, especially in women with LVEF <35%, due to the high risk of thromboembolism. Yet, debates remain over the optimal duration and type of anticoagulant, especially in lactating mothers.

Recovery rates vary widely, with some studies reporting normalization of cardiac function in 50–70% of patients within six months, while others show persistent dysfunction or progression to advanced heart failure. Poor prognostic factors include LVEF <30% at diagnosis, severe ventricular dilation, and African descent. A recurring concern in the literature is the high recurrence risk in subsequent pregnancies. Many experts, including Elkayam (2014), recommend against future pregnancies if cardiac function has not normalized. Yet, cases of uneventful pregnancies in previously affected women with complete recovery suggest that with careful monitoring, individualized decisions may be possible.

The burden of PPCM is significantly higher in developing regions, particularly sub-Saharan Africa, where delayed diagnosis, limited access to care, and coexisting conditions such as anemia and infections complicate management. Literature from these areas, such as studies by Sliwa et al., emphasize the need for culturally appropriate health education and accessible maternal cardiac care.

Despite a growing body of evidence, several gaps remain:

- Lack of large, multicenter, randomized trials for PPCM-specific therapies
- Incomplete understanding of genetic and molecular contributors
- Insufficient data on long-term maternal and neonatal outcomes
- Need for standardized global diagnostic and treatment guidelines

The current literature on peripartum cardiomyopathy has evolved considerably, shedding light on its distinct pathophysiology, variable clinical course, and potential therapeutic targets. However, continued research is essential to refine diagnosis, optimize

treatment protocols, and improve outcomes across diverse healthcare settings. Multidisciplinary collaboration and global awareness are key to addressing the unique challenges posed by this rare but impactful condition.

Conclusion. Peripartum cardiomyopathy (PPCM) is a rare but serious cardiovascular complication of pregnancy that poses significant diagnostic and therapeutic challenges. Its clinical presentation often mimics normal pregnancy-related changes, making timely recognition critical for preventing adverse outcomes. Diagnosis relies heavily on echocardiographic assessment, supported by clinical suspicion, laboratory markers, and exclusion of other potential causes of heart failure. Although the exact etiology of PPCM remains incompletely understood, growing evidence highlights the roles of genetic predisposition, endothelial dysfunction, and hormonal factors such as prolactin and anti-angiogenic proteins. These insights have opened new avenues for targeted therapies, such as bromocriptine, though more robust clinical trials are needed to standardize their use.

Management strategies follow general heart failure guidelines but must be adapted to the unique physiological conditions of pregnancy and postpartum. The combination of pharmacological therapy, supportive care, and, when needed, advanced interventions like mechanical circulatory support, has improved outcomes in many cases. However, maternal mortality and long-term morbidity remain concerns, especially in resource-limited settings.

References

1. Hilfiker-Kleiner D, et al. (2007). A cathepsin D–cleaved 16 kDa form of prolactin mediates postpartum cardiomyopathy. Cell.
2. Sliwa K, et al. (2006). Peripartum cardiomyopathy: epidemiology, pathophysiology, prognosis, and treatment options. Heart.
3. Elkayam U. (2011). Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. J Am Coll Cardiol.
4. Sliwa K, et al. (2010). Bromocriptine for the treatment of peripartum cardiomyopathy: a multicenter randomized study. Eur Heart J.
5. Arany Z, Elkayam U. (2016). Peripartum cardiomyopathy. Circulation.