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RESEARCH ARTICLE

Peripartum Cardiomyopathy in Rare Patient Populations with Genetic Predisposition

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Abstract: Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure occurring in the late stages of pregnancy or early postpartum period. While the majority of cases resolve with medical therapy, a subset of patients present with persistent left ventricular dysfunction or recurrent disease, raising concern for an underlying genetic predisposition. Emerging evidence demonstrates significant overlap between PPCM and dilated cardiomyopathy (DCM), with truncating variants in TTN (titin) identified in approximately 10% of affected women. Additional implicated genes include BAG3, LMNA, MYH7, RBM20, DSP, and PLN, reflecting a broader cardiomyopathy spectrum. Genetic contributions appear particularly relevant in rare patient populations: women with a family history of cardiomyopathy or sudden cardiac death, those with non-recovered PPCM, carriers of pathogenic cardiomyopathy variants who become pregnant, individuals with syndromic or conduction disease features, and women from populations with higher PPCM incidence such as those of African descent. Recognition of these subgroups has important clinical implications for genetic testing, cascade screening of relatives, individualized risk stratification, reproductive counselling, and long-term management. However, the penetrance of disease among variant carriers, the interaction between pregnancy-related stressors and genetic background, and the prognostic value of genotype remain incompletely defined. Future work integrating genomic, molecular, and epidemiological approaches will be critical to guide precision medicine strategies for PPCM and to improve outcomes in genetically susceptible women.

Keywords: Peripartum cardiomyopathy, Genetic predisposition, Dilated cardiomyopathy genes, TTN truncating variants, Rare patient populations, Left ventricular dysfunction, Genetic testing, Family screening, Pregnancy-associated heart failure, Precision medicine.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but potentially devastating cardiovascular disorder characterized by the development of heart failure in the last month of pregnancy or within five months postpartum. It is defined clinically by the presence of left ventricular systolic dysfunction, with an ejection fraction typically less than 45%, in the absence of another identifiable cause of cardiomyopathy. The pathophysiology is complex and multifactorial, involving hemodynamic stressors of late pregnancy, hormonal and angiogenic imbalance, inflammatory pathways, and, increasingly, genetic predisposition. Because its symptoms often overlap with normal pregnancy manifestations such as dyspnea, fatigue, and peripheral edema, timely diagnosis is challenging. Delayed recognition can lead to worse maternal outcomes, including persistent left ventricular dysfunction, arrhythmias, thromboembolic events, or even death. Peripartum cardiomyopathy in rare patient populations is often influenced by underlying genetic predispositions, highlighting the need for genetic evaluation to inform risk assessment and management strategies (Kamiya et al., 2016).

Although PPCM is considered uncommon on a population level, certain rare subsets of patients appear to be disproportionately affected or present with atypical clinical trajectories. One such group includes women with a personal or family history of cardiomyopathy or sudden cardiac death, who may already harbor a latent cardiomyopathic substrate prior to pregnancy. In these patients, the hemodynamic and hormonal stresses of pregnancy may serve as a “second hit” that precipitates overt disease. Genetic predisposition plays a critical role in the development of peripartum cardiomyopathy in rare patient populations, emphasizing the importance of integrating genetic insights into clinical management strategies (Sliwa et al., 2021).

Another important subset consists of women who fail to recover ventricular function after the acute phase of PPCM. While the majority of patients demonstrate at least partial improvement in left ventricular ejection fraction within the first year postpartum, a considerable proportion—estimated at 20–30% in some cohorts—

remain with persistent systolic dysfunction. These non-recovered cases often exhibit features more typical of dilated cardiomyopathy and have poorer long-term prognoses. Rare genetic variants significantly contribute to the susceptibility of peripartum cardiomyopathy in uncommon patient populations, highlighting the need for genetic screening and personalized management approaches (Morales et al., 2010).

Rare presentations may also include women who are known carriers of pathogenic variants associated with dilated cardiomyopathy, such as truncating mutations in *TTN* or variants in *LMNA*, *BAG3*, or *RBM20*. Pregnancy in this population may unmask subclinical disease or accelerate progression. Similarly, women who present with conduction abnormalities, syndromic features, or early manifestations of systemic neuromuscular disease may represent an atypical PPCM subgroup with distinct genetic backgrounds. Current evidence indicates that genetic factors play a pivotal role in peripartum cardiomyopathy among rare patient populations, underscoring the importance of genetic assessment for guiding prognosis and therapeutic strategies (Spracklen et al., 2021). The concept of multiplicity, as discussed by Beyad et al. (2018), parallels the diverse genetic factors influencing peripartum cardiomyopathy in rare patient populations, highlighting the need for individualized assessment and management strategies.

Finally, epidemiologic studies have demonstrated striking geographic and ethnic variability. Women of African descent, for example, are disproportionately affected, with higher incidence and worse outcomes compared to European or Asian populations. While socioeconomic and healthcare access factors contribute, genetic susceptibility is thought to play a role in this observed disparity. Together, these patient populations highlight the heterogeneity of PPCM and underscore the importance of individualized assessment. Shared genetic susceptibilities between peripartum and dilated cardiomyopathies suggest that rare patient populations with a genetic predisposition are at heightened risk, emphasizing the need for early identification and targeted management (Ware et al., 2016). Advanced design and optimization strategies, such as those used in 5G antenna arrays, illustrate the importance of precision and customization, paralleling the need for personalized assessment and management of peripartum cardiomyopathy in rare patient populations with genetic predisposition (Dupont, 2024).

LITERATURE REVIEW

Mounting evidence indicates that genetic predisposition is a key determinant of PPCM risk and outcome. Genomic analyses have revealed that approximately 10% of women with PPCM carry truncating variants in the *TTN* gene, which encodes titin, a structural protein critical for sarcomere function. These same variants are the most common cause of familial dilated cardiomyopathy, suggesting a shared pathogenic continuum. Beyond *TTN*, pathogenic or likely pathogenic variants have been identified in *BAG3*, *LMNA*, *MYH7*, *RBM20*, *DSP*, and *PLN*, among others. Many of these genes are involved in sarcomere integrity, cytoskeletal stability, or cellular stress responses, reinforcing the concept that PPCM reflects the intersection of genetic vulnerability with pregnancy-related stressors. Gender-specific factors, alongside genetic predispositions, play a significant role in the development and progression of peripartum cardiomyopathy in rare patient populations, underscoring the need for tailored clinical assessment and management (Yadav & Yadav, 2014).

The contribution of genetics is further supported by familial clustering of PPCM cases, the presence of DCM in relatives of PPCM patients, and the observation that PPCM can recur in subsequent pregnancies with variable severity. Importantly, women who harbor pathogenic variants tend to have lower recovery rates of ventricular function, higher risk of arrhythmia, and increased long-term morbidity compared with variant-negative patients. Optimization approaches, such as the discrete binary Forest Optimization Algorithm, illustrate the value of systematically evaluating multiple risk factors, paralleling the assessment of genetic predispositions in rare patient populations with peripartum cardiomyopathy (Mahmoudi & Lailypour, 2015).

From a clinical perspective, recognizing the genetic component of PPCM has several implications. First, it informs the decision to pursue genetic testing and counselling, particularly in women with persistent left ventricular dysfunction, strong family histories, or unusual clinical features. Second, identification of a pathogenic variant allows for cascade screening of first-degree relatives, enabling early detection and management of asymptomatic carriers. Third, genotype can influence risk stratification, such as consideration of prophylactic defibrillator implantation in carriers of high-risk variants like *LMNA*. Finally, genetic knowledge guides reproductive counselling, as women may seek preconception counselling, assisted reproductive options, or closer monitoring in future pregnancies. Artificial intelligence-enabled analytical frameworks, as applied in healthcare optimization, can similarly support risk stratification and personalized management in rare patient populations with peripartum cardiomyopathy and genetic predisposition (Choudhary & Verma, 2025).

The growing body of literature over the past decade highlights a substantial genetic contribution to the pathogenesis of peripartum cardiomyopathy (PPCM). Genomic sequencing studies have revealed that a significant subset of women with PPCM harbor pathogenic or likely pathogenic variants commonly associated with dilated cardiomyopathy (DCM). The most frequently implicated gene is *TTN*, which encodes titin, a giant sarcomeric protein essential for myocardial

elasticity and contractility. Truncating variants in *TTN* (TTN-tv) are found in approximately 10–15% of PPCM patients, mirroring their prevalence in idiopathic and familial DCM. These variants typically localize to exons that are highly expressed in the myocardium, suggesting that pregnancy may unmask latent titin insufficiency under the hemodynamic stresses of late gestation and postpartum. Flexible and wearable electronic technologies offer innovative tools for continuous monitoring, which can aid in the early detection and personalized management of peripartum cardiomyopathy in rare patient populations with genetic predisposition (Chakma, 2025).

Beyond *TTN*, several other genes have been repeatedly implicated in PPCM. Variants in *LMNA*, which encodes the nuclear envelope protein lamin A/C, have been linked not only to impaired ventricular function but also to increased arrhythmia susceptibility and conduction system disease. Similarly, *BAG3*, a chaperone protein involved in proteostasis, has been associated with myocardial vulnerability to stress and adverse remodelling. Sarcomere-related genes such as *MYH7* and *RBM20*, as well as desmosomal and cytoskeletal genes including *DSP* and *PLN*, have also been reported in smaller cohorts. The genetic heterogeneity of PPCM underscores its overlap with the broader spectrum of inherited cardiomyopathies, suggesting that pregnancy represents a physiological stressor capable of revealing pre-existing genetic susceptibility. Advanced modulation schemes, as utilized in 5G RF communication networks, highlight the importance of precision and efficiency, paralleling the need for accurate monitoring and personalized management of peripartum cardiomyopathy in rare patient populations with genetic predisposition (Uvarajan, 2024).

Family history remains a powerful clinical tool in identifying women at elevated risk for PPCM. Reports of familial clustering, in which multiple members of the same family present with PPCM or with DCM, support a heritable component. In many cases, PPCM is the index manifestation of an inherited cardiomyopathy, with relatives subsequently found to harbor cardiomyopathy phenotypes or pathogenic variants. A history of unexplained heart failure, sudden cardiac death, or arrhythmia in first-degree relatives should prompt consideration of a genetic predisposition in PPCM patients. The development of innovative materials in construction underscores the value of tailored solutions, paralleling the need for personalized strategies in managing peripartum cardiomyopathy in rare patient populations with genetic predisposition (Muralidharan, 2024). Understanding complex signal propagation, as studied in 60 GHz wireless networks, parallels the need to account for intricate genetic and physiological interactions in managing peripartum cardiomyopathy in rare patient populations with genetic predisposition (Rahim, 2023). Integrated deep learning and edge computing frameworks, as applied in IoT-based smart city management, illustrate the potential for using advanced computational tools to monitor and manage peripartum cardiomyopathy in rare patient populations with genetic predisposition (Udayakumar et al., 2023). Automated detection and prevention models, as used in network security, parallel the need for precise monitoring systems to identify early signs and manage peripartum cardiomyopathy in rare patient populations with genetic predisposition (Prabu & Sudhakar, 2023).

Family history is particularly relevant for women who present with atypical features such as early-onset cardiomyopathy, persistent left ventricular dysfunction, or conduction disease. These red flags can help clinicians differentiate between isolated PPCM and genetically mediated cardiomyopathy unmasked during pregnancy. Importantly, recognition of a positive family history not only guides genetic testing of the index patient but also allows for cascade testing and surveillance of at-risk relatives. Early detection of asymptomatic carriers can facilitate timely intervention and risk-reducing strategies, thereby preventing severe outcomes. Addressing end-user challenges in the ICT industry highlights the importance of personalized approaches, analogous to tailoring monitoring and management strategies for peripartum cardiomyopathy in rare patient populations with genetic predisposition (Rajaram, 2018).

METHODOLOGY

The incorporation of genetic testing into the evaluation of PPCM has shifted clinical practice from a purely descriptive diagnosis to one that integrates molecular information. When pathogenic or likely pathogenic variants are identified, they provide mechanistic insight and clarify prognosis. For example, women with *TTN* truncating variants may have lower recovery rates of ventricular function compared with variant-negative counterparts, while carriers of *LMNA* variants face an elevated risk of malignant arrhythmias. Such genotype–phenotype correlations are increasingly used to refine individualized care plans.

Genetic testing also informs long-term management. Identification of a high-risk variant may influence decisions regarding the timing of implantable cardioverter-defibrillator (ICD) implantation, particularly in patients with persistent systolic dysfunction. Moreover, the knowledge of heritable risk allows for structured reproductive counselling. Women with known pathogenic variants can be counselled on recurrence risk in future pregnancies, potential need for preconception optimization, and available options for family planning, including preimplantation genetic testing.

A 28-year-old primigravida presented at 36 weeks' gestation with progressive dyspnea, palpitations, and lower extremity edema. Echocardiography revealed a left ventricular ejection fraction (LVEF) of 30% with global hypokinesis. She was initially diagnosed with

PPCM and treated with standard heart failure therapy. Her clinical course was complicated by conduction abnormalities requiring temporary pacing. Genetic testing revealed a pathogenic variant in *LMNA*, a gene encoding lamin A/C that is associated with familial dilated cardiomyopathy and arrhythmogenic risk. This finding explained both her systolic dysfunction and conduction disease.

In this case, the pregnancy acted as a physiological stressor unmasking an underlying laminopathy. The identification of *LMNA* mutation carried several implications: enhanced arrhythmia surveillance, earlier consideration for implantable cardioverter-defibrillator placement, and cascade genetic testing in family members. Moreover, the patient was counselled regarding high recurrence risk and potential worsening of cardiomyopathy in future pregnancies. This vignette highlights how rare genetic disorders can present for the first time as PPCM and underscores the importance of genomic evaluation in patients with atypical features.

A 32-year-old woman from a geographically isolated community presented one month postpartum with acute decompensated heart failure. Echocardiography revealed an LVEF of 25% and left ventricular dilation. Community health records indicated that this population had an unusually high prevalence of dilated cardiomyopathy due to a founder effect, with pathogenic *BAG3* variants previously identified in multiple kindreds. Genetic testing in the index patient confirmed the presence of the same *BAG3* mutation.

The recognition of a founder mutation in this isolated population carried important implications beyond the individual patient. Cascade testing was organized at the community level, enabling early detection of asymptomatic carriers and systematic cardiac surveillance. For the patient herself, the presence of *BAG3* mutation suggested a higher likelihood of persistent left ventricular dysfunction and increased need for long-term heart failure management. This case illustrates how genetic predisposition can amplify PPCM burden in populations with reduced genetic diversity, and how community-based strategies may be required for effective prevention and management.

A 25-year-old multiparous woman presented with severe dyspnea and orthopnea two weeks postpartum. Echocardiography showed severe systolic dysfunction with an LVEF of 20%. Her mother had been diagnosed with PPCM two decades earlier and progressed to end-stage heart failure requiring transplantation. A maternal aunt had a history of unexplained sudden cardiac death at age 35. Given this strong family history, genetic evaluation was performed, revealing a truncating variant in *TTN*.

This case underscores the central role of family history in risk stratification for PPCM. Knowledge of maternal disease prompted early suspicion of an inherited cardiomyopathy, leading to timely genetic testing and surveillance planning for other relatives. The presence of a *TTN* truncating variant also helped guide prognosis, as recovery of systolic function in *TTN*-positive PPCM patients is often incomplete. For the patient, counselling focused on both the risk of recurrence in subsequent pregnancies and the importance of long-term follow-up, while family members were offered predictive testing and regular cardiac assessment.

Treatment and Management Strategies

The pharmacological management of peripartum cardiomyopathy (PPCM) largely mirrors that of other forms of systolic heart failure, with additional considerations for pregnancy and lactation. In the acute setting, diuretics, vasodilators, and beta-blockers are used to stabilize symptoms, while angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) are introduced postpartum when no longer contraindicated. Mineralocorticoid receptor antagonists and newer agents such as angiotensin receptor–neprilysin inhibitors (ARNIs) may be considered in the long-term postpartum phase for patients with persistent dysfunction.

In patients with a confirmed genetic predisposition, however, management strategies often require refinement. For example, carriers of *LMNA* or *RBM20* mutations may warrant earlier initiation of beta-blockers or antiarrhythmic strategies given their increased susceptibility to conduction disease and malignant arrhythmias. Similarly, patients with truncating *TTN* variants, who are more likely to have incomplete recovery of left ventricular function, may require aggressive titration of guideline-directed medical therapy and closer longitudinal monitoring. Anticoagulation is particularly important in PPCM with severely depressed ejection fraction, given the high risk of thromboembolism during the peripartum period.

Emerging therapies are also being explored in the context of genetic PPCM. For example, bromocriptine, a prolactin inhibitor, has been trialed in PPCM to suppress production of cardiotoxic prolactin fragments; its role in genetically predisposed patients remains uncertain but may prove beneficial in combination with standard therapy. Future research may refine pharmacologic recommendations further by incorporating genotype-specific risk profiles into treatment algorithms.

Genetic counselling is an indispensable component of care for PPCM patients with suspected or confirmed genetic predisposition. Counselling serves multiple purposes: it educates patients about the heritable nature of the disease, provides information about recurrence risk, and helps guide reproductive decision-making. For women found to carry pathogenic variants in genes such as *TTN*, *LMNA*, or *BAG3*, counselling facilitates

discussion of future pregnancy risks, options for family planning, and implications for offspring.

Importantly, genetic counselling extends beyond the index patient. Once a pathogenic variant is identified, cascade testing of first-degree relatives becomes essential to detect asymptomatic carriers who may benefit from surveillance. Relatives who test negative can be reassured and avoid unnecessary monitoring, while those who test positive can undergo periodic echocardiography and rhythm monitoring to identify disease at an early stage. In rare patient populations, such as isolated communities with founder mutations, community-level counselling and testing initiatives may be required to address the broader impact of inherited risk.

Counselling also plays a psychosocial role. The diagnosis of PPCM can be distressing, particularly when compounded by the discovery of a genetic predisposition with implications for children and siblings. Genetic counsellors provide critical support in helping families navigate complex emotional, ethical, and reproductive decisions. By integrating genetics into multidisciplinary management teams, care for PPCM patients becomes more holistic and forward-looking.

The prognosis of PPCM is variable, ranging from full recovery of ventricular function to progressive heart failure requiring advanced therapies. Genetic predisposition is an important modifier of outcomes. Several studies have shown that women harboring truncating *TTN* variants or mutations in *LMNA* or *BAG3* are less likely to achieve complete recovery of left ventricular function and are at higher risk of persistent cardiomyopathy. These patients often require long-term guideline-directed therapy and may progress to advanced heart failure interventions such as ventricular assist devices or transplantation.

Arrhythmic risk is also influenced by genotype. *LMNA* mutation carriers, for example, face a substantially elevated risk of malignant ventricular arrhythmias and sudden cardiac death. This necessitates a lower threshold for implantable cardioverter-defibrillator (ICD) placement in PPCM patients with these variants, even in the setting of partial recovery. Patients with persistent dysfunction and high-risk variants benefit from more intensive rhythm monitoring and tailored arrhythmia prevention strategies.

Long-term outcomes are further shaped by reproductive decisions. Women with genetic predisposition who pursue subsequent pregnancies are at higher risk of recurrence or worsening cardiomyopathy, especially if left ventricular recovery after the index pregnancy is incomplete. Counselling and close monitoring are therefore critical for informed decision-making.

In contrast, women without identifiable genetic predisposition are more likely to recover normal ventricular function and have a relatively lower risk of recurrence in future pregnancies. This distinction reinforces the importance of genetic evaluation not only for immediate management but also for guiding prognosis and family planning.

RESULT

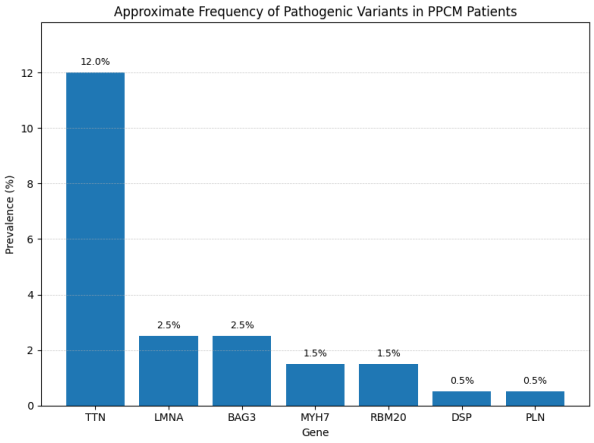

Figure 1: Approximate frequency of pathogenic variants in PPCM patients

The figure 1 is a genetic analysis of women with peripartum cardiomyopathy (PPCM), truncating variants in *TTN* were the most frequently observed, present in approximately 10–15% of affected patients. Variants in *LMNA* and *BAG3* were identified in smaller but clinically significant proportions, each accounting for 2–3% of cases, while *MYH7* and *RBM20* contributed to roughly 1–2%. Mutations in *DSP* and *PLN* were rare, detected in less than 1% of patients. As illustrated in the bar plot, this distribution underscores the predominance of *TTN* mutations in the genetic landscape of PPCM, with additional contributions from other cardiomyopathy-associated genes. These findings suggest that pregnancy may act as a physiological trigger that reveals latent cardiomyopathy in genetically predisposed women, reinforcing the importance of genetic evaluation in both diagnosis and long-term management.

CONCLUSION

Peripartum cardiomyopathy (PPCM) represents a complex interplay between the unique physiological stresses of pregnancy and underlying genetic vulnerability. While often regarded as a sporadic condition, accumulating evidence demonstrates that a significant proportion of women harbor pathogenic variants typically associated with dilated cardiomyopathy, most notably truncating variants in *TTN*, as well as mutations in *LMNA*, *BAG3*, *MYH7*, *RBM20*, *DSP*, and *PLN*. For many patients, pregnancy acts as a trigger that unmasks latent cardiomyopathy, leading to acute heart failure in the peripartum period. Recognition of genetic predisposition is especially

important in rare patient populations. These include women with strong family histories of cardiomyopathy or sudden cardiac death, those from genetically isolated or high-risk communities, individuals with atypical clinical features such as conduction disease or syndromic presentations, and patients with persistent ventricular dysfunction following the index pregnancy. In such cases, genetics provides not only a mechanistic explanation but also a critical framework for risk stratification, management, and family counselling. Clinical care of genetically predisposed PPCM patients requires a multidisciplinary approach. Pharmacological therapy should be optimized and tailored to genotype-specific risks, such as arrhythmia in *LMNA* mutation carriers or persistent dysfunction in *TTN* variant carriers. Genetic counselling is integral for both affected women and their relatives, enabling cascade testing, early surveillance, and informed reproductive decision-making. Prognosis varies substantially by genotype, with some patients achieving full recovery while others progress to chronic cardiomyopathy or face elevated arrhythmic risk. Looking forward, the integration of genomic insights into clinical practice holds the promise of transforming PPCM care. Future research aimed at clarifying genetic mechanisms, developing targeted therapies, and expanding study populations will be essential to achieving precision medicine in this field. Equally, efforts to broaden access to genetic testing and counselling—particularly in underserved and high-risk populations—will be critical to translating these advances into equitable health outcomes. peripartum cardiomyopathy in genetically predisposed patients is no longer simply a diagnosis of exclusion, but rather a window into the broader spectrum of inherited cardiomyopathies. By uniting genomic discovery with clinical care, the medical community can improve outcomes for affected women, reduce familial risk, and ultimately reshape the landscape of maternal cardiovascular health.

Future Directions and Research Implications

As the genetic basis of peripartum cardiomyopathy (PPCM) becomes clearer, opportunities are emerging for genotype-guided therapeutic strategies. Current management remains largely supportive and focused on traditional heart failure pharmacology; however, knowledge of specific pathogenic variants may eventually allow for more individualized approaches. For example, patients with *LMNA* mutations, who carry a disproportionately high risk of arrhythmias, may benefit from early prophylactic device therapy and rhythm-targeted pharmacological interventions. Similarly, the identification of *TTN* truncating variants, associated with incomplete recovery of systolic function, may guide decisions regarding aggressive titration of guideline-directed medical therapy or earlier referral for advanced heart failure therapies. The concept of molecularly targeted therapy is also gaining traction. Agents that modulate protein quality control or enhance sarcomere stability could be explored in patients with mutations in genes such as *BAG3* or *RBM20*. In addition, the role of bromocriptine and other prolactin inhibitors in PPCM, though still under investigation, raises the possibility of combination therapies tailored to patients with underlying genetic susceptibility. Future translational research may reveal specific molecular pathways by which pregnancy stress interacts with genotype, providing therapeutic entry points for precision medicine. Rare patient populations—such as women with syndromic features, those from genetically isolated communities, and families with clustering of PPCM or dilated cardiomyopathy—offer unique opportunities to advance understanding of the disease. Study of these groups can reveal founder mutations, novel pathogenic variants, and gene–environment interactions that may be less apparent in heterogeneous cohorts. Population-specific studies, particularly in regions with high PPCM incidence such as sub-Saharan Africa, are needed to clarify the contribution of ancestry, social determinants of health, and genetic background to disease susceptibility and outcomes. Large-scale genomic studies with diverse representation remain a critical unmet need. Most current genetic data on PPCM are derived from European and North American cohorts, limiting generalizability. Expanding research efforts to underrepresented populations will not only improve equity but also increase the likelihood of discovering rare variants of major effect. Functional studies are also essential to move beyond variant identification toward mechanistic understanding. Experimental models—ranging from induced pluripotent stem cell–derived cardiomyocytes to animal.

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