In women carrying a LQTS P/LP variant and who are phenotype-negative, use of beta-blockers[c] during pregnancy, post-partum, and lactation should be considered.[148]	IIa	C
Left cardiac sympathetic denervation should be considered before pregnancy in high-risk woman with LQTS who are not adequately protected by pharmacological therapies or who have appropriate ICD shocks despite optimal medical therapy.[252]	IIa	C
Brugada syndrome		
Quinidine therapy should be considered in pregnant women with BrS who have arrhythmic events during pregnancy.[337,338]	IIa	C
Catecholaminergic polymorphic ventricular tachycardia		
Beta-blockers[c], with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy and lactation in women with CPVT.[43,148,252]	I	C
Flecainide, in addition to beta-blockers, is recommended in women with CPVT who experience cardiac events such as syncope, VT, or cardiac arrest during pregnancy.	I	C
It is recommended that women with CPVT who are stable on beta-blockers (nadolol or propranolol as drugs of choice) and flecainide before pregnancy continue both drugs during pregnancy and post-partum.	I	C
The use of beta-blockers[c] during pregnancy and lactation should be considered in phenotype-negative women with a CPVT P/LP variant.[148]	IIa	C
Left cardiac sympathetic denervation should be considered before pregnancy in high-risk women with CPVT who are not adequately protected by pharmacological therapies or with appropriate ICD shocks despite optimal medical therapy.[252]	IIa	C
Short QT syndrome		
It should be considered to continue quinidine therapy in women with SQTS throughout pregnancy and the post-partum period.[148]	IIa	C
Quinidine therapy should be considered in pregnant women with SQTS and arrhythmic events during pregnancy.[148]	IIa	C

BrS, Brugada syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; ICD, implantable cardioverter defibrillator; LQTS, long QT syndrome, LQT2, long QT syndrome type 2; P/LP, pathogenic/likely pathogenic; SQTS, short QT syndrome; VT, ventricular tachycardia.
[a]Class of recommendation.
[b]Level of evidence.
[c]Except for atenolol.

7. Peripartum cardiomyopathy

7.1. Epidemiology

Peripartum cardiomyopathy is a potentially life-threatening condition defined as HF with reduced left ventricular ejection fraction (LVEF) <45%, without any other cause of HF, that occurs mainly during the peripartum period or in the months following delivery, termination, or miscarriage.[339] Peripartum cardiomyopathy is essentially a diagnosis of exclusion and requires urgent management.

Worldwide, PPCM is a complication of 1 of 2000 births,[340] but incidence rates vary depending on the geographical region, ethnicity, and socioeconomic factors, with an incidence of 1–4/1000 births in the United States of America[341,342] and 10/1000 births in the north-western region of Nigeria.[343] Recent data from 49 countries showed that most women presented with PPCM in the post-partum stage.[344–346] Risk factors for PPCM are shown in *Figure 7*.

7.2. Mechanisms

Recent trials suggest a 'multiple hit' theory for developing PPCM, with an accumulation of genetic and environmental risk factors (*Figure 7*). An overrepresentation of genetic variants in *TTN*, *FLNC*, *BAG3*, and *DSP* genes have been found in up to 15% of women with PPCM, with *TTN* truncating variants being the most common.[347,348] The prevalence of these four genes in women with PPCM was comparable to the prevalence in DCM cohorts, supporting the similarity between PPCM and DCM. Genetic testing should therefore be considered in women with PPCM.

There is growing evidence that several pathophysiological mechanisms in PPCM converge on a common pathway, which involves inflammation, unbalanced oxidative stress, and the generation of the anti-angiogenic 16 kDa prolactin. The 16 kDa prolactin induces endothelial dysfunction and damage, and subsequently leads to HF. Blocking prolactin with the dopamine D2 receptor agonist bromocriptine has emerged as a potential disease-specific therapy for PPCM.[344,349–352] Additionally, the systemic or local increase in other anti-angiogenic factors, such as the soluble fms-like tyrosine kinase-1 (sFlt-1) receptor, contributes to both local and widespread vascular dysfunction.[353]

7.3. Diagnosis and clinical interventions

Peripartum cardiomyopathy may present as subtle manifestations but most women with PPCM present with acute heart failure (AHF) with severe symptoms (NYHA III/IV). Mild to moderate symptomatic cases of PPCM are often mistaken for physiological changes associated with pregnancy, especially in the post-partum period. Myocarditis is a differential diagnosis and should be excluded by CMR.[354] Women with PPCM and pre-eclampsia had a higher risk of adverse neonatal outcome but also a higher likelihood of left ventricular recovery (LVEF ≥50%).[15] Diagnostic measures in a woman with suspected PPCM should include ECG, NPs, and echocardiography. The management strategy should be discussed within the Pregnancy Heart Team, considering maternal and foetal outcomes (*Figure 7*). Foetal prematurity and low birth weight are common in mothers with PPCM, and children of these mothers have a 3.4 times higher incidence of cardiovascular disease and 5 times higher mortality.[355]

Treatment of AHF caused by PPCM follows the main principles of AHF management during and after pregnancy (*Section 12.6*). Mechanical circulatory support should be considered in women with persistent cardiogenic shock despite medical treatment.[356,357]

Most medications used in the management of HF are foetotoxic and thus contraindicated during pregnancy (i.e. ACE-Is, ARBs, MRAs, and SGLT2 inhibitors).[339] In the post-partum period, full HF treatment