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Short Commentary

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Peripartum cardiomyopathy

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Introduction

Peripartum Cardiomyopathy (PPCM), also known as postpartum cardiomyopathy is a form of heart failure (HF) defined as new onset of left ventricular ejection fraction (LVEF) reduction (<45%) occurring in between last month of pregnancy till the period of puerperium i.e. 6 weeks after delivery in the absence of other identifiable causes of HF or recognizable pre-existing structural heart disease (earlier defined as new onset heart failure between the last month of pregnancy and 5 months post-delivery with no determinable cause).

Epidemiology, risk factors & pathophysiology

The global estimated incidence of PPCM is around 1 every 2000 deliveries, with wide regional differences ranging from 1 in 100 births in Nigeria to 1 in 20,000 in Japan. In Western Countries, reported incidence is between 1 in 1000 and 1 in 5000, significantly higher in women of African ancestry than in Caucasians.

The incidence of peripartum cardiomyopathy (PPCM) in India is between 1 in 1,340 and 1 in 1,541 live births.

Other than ethnicity, several other risk factors for PPCM have been detected over the last few decades. Patients with gestational hypertension or pre-eclampsia present a three-fold increased risk of PPCM. Multiple gestations are strongly associated with PPCM, being reported in 7%-14.5% of cases. Advanced maternal age is another relevant risk factor for PPCM, more likely from 30 years of age onwards, independently from the presence of age-associated comorbidities, including hypertension.

Other conditions deemed to be predisposing to PPCM are multiparity, twin pregnancy, obesity, smoke, diabetes, anaemia, asthma, malnutrition and prolonged tocolytic therapy.

Even though a growing body of research has led to greater knowledge and awareness of this condition, PPCM pathophysiology still remains not fully understood, but it is almost certainly multifactorial.

Pregnancy-related hemodynamic stress has been the first proposed mechanism. However, the main circulatory changes occur during the second trimester, whereas PPCM mostly manifests itself after delivery. The fact that all pregnant women undergo similar hemodynamic and hormonal changes but very few of them develop PPCM, suggests that genetic predisposition could be particularly important.

Altered levels of prolactin along with angiogenesis imbalance is one of the responsible factors in the pathogenesis of PPCM.

Symptomatology

- Women with PPCM typically present with symptoms and signs of heart failure, including:
- Fatigue
- Dyspnoea
- Orthopnoea
- Nocturnal paroxysmal dyspnoea
- Chest tightness, palpitations
- Tachycardia, tachypnoea
- Elevated jugular venous pressure (swollen neck veins)
- Pulmonary edema
- Rarely, PPCM presents with cardiogenic shock, arrhythmias or thromboembolic events.

Diagnosis & differentials

- Noteworthy, PPCM is a diagnosis of exclusion, which should be considered when a new LVEF <45% is identified in pregnant women during the peri-partum period in the absence of other structural heart disease. LV dilation is usually present, but is not a diagnostic criterion, as a certain degree of cardiac chambers enlargement may be a physiological adaptation to pregnancy.
- No specific test for diagnosing peripartum cardiomyopathy, but certain tests may include:
- Electrocardiography (ECG) may show non-specific abnormalities, including sinus tachycardia and ST segment alterations. However, it is important to underline that a normal ECG does not exclude PPCM. Conversely, ECG may be particularly useful to rule out ST segment elevation myocardial infarction.
- Serum brain natriuretic peptide and its N-terminal portion are usually elevated in PPCM. While their levels are typically normal during pregnancy or only slightly elevated in case of pre-eclampsia. Even cardiac troponin may be increased in PPCM.
- Cardiac magnetic resonance can be considered in case of suboptimal echocardiographic windows.
- PPCM diagnosis is usually performed by means of echocardiography, which allows not only to assess the degree of LV dysfunction, but also to explore right ventricle (RV) involvement.

Other tests include:

- Chest X-ray
- Blood tests
- Endomyocardial biopsy is not required in the vast majority of cases, but may be considered in some cases.

Differential diagnoses that should be explored include:

- Pre-existing structural heart disease
- Pulmonary embolism
- Preeclampsia-induced pulmonary oedema without LV dysfunction
- Spontaneous coronary artery dissection
- Myocarditis
- Takotsubo syndrome
- Myocardial infarction
- Aortic dissection with acute aortic regurgitation
- Alcohol abuse and chemotherapeutic agents

Management

Acute phase management

PPCM clinical presentation is extremely variable. The European Society of Cardiology Study group on PPCM described three possible clinical scenarios:

1. Mild PPCM
2. Moderate PPCM
3. Severe PPCM

Patients with mild PPCM do not require admission in intensive care and, in selected cases, follow up in dedicated outpatient clinics may be considered. Patients with moderate and severe PPCM require hospital admission, respectively in HF and intensive care units. The possible teratogenic effects of inotropes and vasopressors in humans are still largely unknown, but this should not halt their use when needed. Patients with PPCM seem to be particularly prone to the side effects of beta-adrenergic stimulation, therefore drugs like dobutamine should be avoided, so norepinephrine is the preferred inotropic support for them. A physician can prescribe several classes of medications to treat symptoms and help recover heart function like:

Angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs) – Lower blood pressure and helps the heart work more efficiently. Angiotensin receptor/neprilysin inhibitor (ARNI)-Lowers blood pressure and makes it easier for the heart to pump blood. Beta blockers-Cause the heart to beat more slowly so it can recover with time. Diuretics-Reduce fluid retention. Digitali-Strengthens the pumping ability of the heart, but due to narrow safety margins with the need to monitor levels, it is not frequently used. Anticoagulants-Help thin the blood. As pregnancy is a pro-thrombotic condition and also patients with PPCM are at increased risk of developing blood clots, especially if the ejection fraction is very low.

Bromocriptine- Blocks the release of prolactin, a hormone that promotes lactation. Bromocriptine may help the heart recover, but the recommendations for use are not specific. Additional research is needed to understand if bromocriptine should be prescribed for patients with severe PPCM.

Long term management: Out of the acute phase, PPCM patients may require chronic HF treatment. Most of conventional HF medications are safe during breastfeeding. Patients with persistent LV dysfunction require life-long medical therapy.

Prognosis

The most complex task for cardiologists when assessing patients with PPCM is, probably, to provide adequate counsel about the individual patient risk for Subsequent Pregnancies (SSPs). If the patient develops PPCM during the last month of pregnancy, their healthcare provider will monitor the health of the fetus with the mother too. They'll also create a plan for your type of delivery. If your heart failure is stable, your provider will likely prefer you have a vaginal delivery. But you may need an epidural, episiotomy or the use of forceps to make delivery easier. But with PPCM symptoms, it is more likely to have a Cesarean delivery (C-section) or preterm birth for the safety of mother and baby.

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