

REVIEW ARTICLE

Navigating Cardiomyopathy in Pregnancy: Insights for Primary Care Providers

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ABSTRACT

Cardiac diseases detected during pregnancy are a major cause of maternal morbidity and mortality. Peripartum cardiomyopathy and Takotsubo cardiomyopathy, although uncommon, have favourable outcomes if detected early and managed appropriately. Diagnosis of both conditions is often challenging and delayed due to its nature of presentation. This paper seeks to offer a comprehensive overview of both peripartum cardiomyopathy (PPCM) and Takotsubo cardiomyopathy (TC), with a focus on primary care perspectives.

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INTRODUCTION

One of the leading causes of maternal deaths in Malaysia is “associated medical conditions” (Figure 1) (1). According to the Report on the Confidential Enquiries into Maternal Deaths in Malaysia 2012 – 2014, approximately fifty percent of “associated medical conditions” were attributed to cardiac diseases(2). Among these, cardiac conditions such as valvular heart disease, often stemming from chronic rheumatic heart disease, primary pulmonary hypertension, and occasionally ‘peripartum cardiomyopathy’, were the prevailing factors (2). Table I shows the common cardiac diseases occurring in pregnancy and its presentation. The confidential inquiry report underscored “delayed diagnosis” as a significant factor contributing to both morbidity and mortality in these cases. This emphasizes the critical need for timely identification and intervention in pregnant women with cardiac conditions.

Cardiomyopathy in pregnancy, in the absence of underlying cardiac disease, although rare, is an important cause of maternal morbidity and mortality. There are typically two main varieties: peripartum

cardiomyopathy (PPCM) and, less commonly, Takotsubo cardiomyopathy (TC).

43 cases of peripartum cardiomyopathy were reported in the Confidential Enquiry into Maternal Deaths, Malaysia between 1991-2014 (3). While TC was not specifically mentioned, there were a total of 24 cases recorded as “other cardiomyopathy” (3). A study on PPCM, conducted at University Malaya Medical Centre Kuala Lumpur over a ten-year period, reported a prevalence of 2.48 in 100,000 live births (4). A search of databases did not yield any literature on TC in pregnancy in Malaysia. While actual numbers for both these forms of cardiomyopathies are low, it remains significant and must be addressed as both conditions have favorable outcomes if diagnosed and treated early (5,6)

Although peripartum cardiomyopathy and TC are relatively uncommon, primary care physicians responsible for antenatal care must maintain a high level of vigilance in their cardiac assessments of pregnant women. These conditions often manifest without symptoms during the early weeks of gestation, necessitating thorough and proactive monitoring. This article aims to offer a comprehensive overview of both PPCM and TC, encompassing their pathophysiology, clinical presentation, and the diagnosis and management of these conditions, with a focus on primary care perspectives.

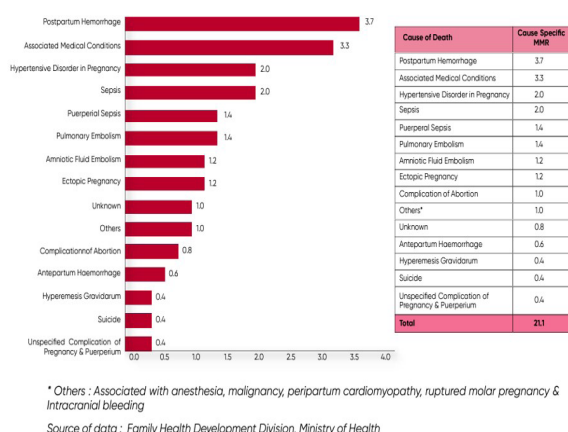


Figure 1: Cause Specific Maternal Mortality Rate: Malaysia 2019

Table I: Cardiac Diseases in Pregnancy and its Presentation

Disease	Presentation
Chronic rheumatic heart disease	This condition usually leads to mitral stenosis. Patients present with symptoms and signs of heart failure, including palpitations, which may be due to atrial fibrillation. Mitral stenosis is the commonest lesion; complications seen are acute pulmonary oedema and atrial fibrillation.
Congenital heart disease	This may be due to valvular heart disease, Tetralogy of Fallot, Ebstein Anomaly or septal defects. Untreated septal defects may lead to Eisenmenger Syndrome (ES). ES presents with cyanosis, syncope, dyspnoea during exertion, fatigue, chest pain, and palpitations. The mortality rate can exceed 50%.
Other valvular heart disease	Other valvular heart diseases can occur either due to congenital anomalies or rheumatic heart disease. Depending on the severity, patients may present with symptoms and signs of heart failure. Patients with aortic stenosis may also experience angina.
Infective endocarditis	Risk factors are rheumatic heart disease, congenital heart disease, prosthetic valves, haemodialysis, IV catheters or immunosuppression. Patients present with fever, joint pains, pallor, petechiae and cardiac murmurs.
Cardiac arrhythmia	May either be due to a previously diagnosed cardiac arrhythmia or due to underlying structural heart disease.

PERIPARTUM CARDIOMYOPATHY

Definition

In 2010, The Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy defined PPCM as “an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery where no other cause of heart failure is found”. The Working Group elaborated that echocardiography may reveal either the presence or absence of left ventricular dilatation, yet the ejection fraction consistently remains below 45%. Emphasis was placed on the fact that PPCM is primarily a diagnosis made through exclusionary measures (7). Bauersachs et. al. emphasizes that this definition also applies to patients who have experienced an abortion or miscarriage (8). PPCM was first documented in Malaysia in 1989 (9). However, as previous definitions of PPCM have been vague with regards to diagnostic criteria and timing of occurrence, the possibility of underdiagnosis should be kept in mind.

Pathophysiology

Current evidence on the pathophysiology of PPCM is limited to experimental models in mice, which support the theory of vascular and cardiomyocyte dysfunction; triggered by oxidative stress and hormonal factors in pregnancy, mainly prolactin, cathepsin-D and 16kDA prolactin (7,10). Inflammation, nutritional deficiencies, viral myocarditis, autoimmune states and a genetic predisposition may also contribute to the pathophysiology of this condition (9,10,11). However, there is insufficient evidence to support these factors.

Clinical presentation

The signs and symptoms of PPCM may mimic that of normal pregnancy, and this poses a diagnostic challenge. It is important to take into consideration risk factors that predispose to PPCM. The strongest risk factors for PPCM are hypertension and pre-eclampsia (10). Other risk factors include maternal age over 30 years, multiparity and in-vitro fertilization (11).

The common presentation of PPCM is one of heart failure. Severe arrhythmias, cardiogenic shock and thromboembolic complications while possible, are uncommon. (10). Consequently, patients are more prone to manifest symptoms and signs indicative of heart failure within a primary care setting, as illustrated in Table II.

Table II: Comparison of PPCM and TC

Parameters	PPCM	TC
Symptoms	Mimics heart failure	Mimics acute myocardial infarction
Signs	Typical signs of heart failure	• Normal or signs of heart failure
Investigations	• Raised BNP • Raised N-terminal pro-BNP • ECG: non-specific changes, arrhythmias, QRS prolongation • Chest radiograph: pulmonary venous congestion • Echocardiogram: LVEF <45%	• Electrocardiogram: ST segment and T wave changes, q waves, QT prolongation, non-specific changes • Cardiac biomarkers: Raised troponin T and troponin I • Echocardiogram: regional wall motion akinesia of the left ventricle acute apical ballooning with dilatation of the mid-ventricular area and reduced ejection fraction (20 – 45%)
Management	• Team-based approach • Guidelines for heart failure, with precaution for medications contraindicated in pregnancy. • Consider use of bromocriptine	• Team-based approach • Supportive treatment • Management of complications as per guidelines
Breast-feeding	• Evidence is conflicting	• No contraindication
Contraception	• IUCD preferred • Progesterone-only containing contraception is acceptable. • Avoid barrier methods	• Combined oral contraceptive. • Other forms of hormonal contraception are acceptable. • Avoid barrier methods

Diagnosis

The primary care doctor should have a high index of suspicion in patients with risk factors presenting with signs and symptoms of heart failure, especially in the last trimester or post-partum period. As the absence of underlying factors are one of the criteria to diagnose PPCM, a careful history, physical examination and investigations to rule out underlying disorders is necessary (12). Nevertheless, in patients with regular antenatal follow-up, most underlying causes would have been detected earlier.

Brain natriuretic peptide (BNP) and N-terminal Pro BNP are significantly elevated in PPCM (10). Electrocardiogram can be normal or demonstrate non-specific changes including arrhythmias or QRS prolongation (11). A chest radiograph may demonstrate signs of pulmonary venous congestion (10). Echocardiogram is mandatory to demonstrate a left ventricular ejection fraction (LVEF) of <45%. Other findings include left or right ventricular dilatation or dysfunction, functional tricuspid or mitral regurgitation, left or bi-atrial enlargement and pulmonary hypertension (10).

Management

Early diagnosis and prompt referral for co-management with an obstetrician, cardiologist and neonatologist

among others is essential for patient's presenting to primary care (7,10).

For patients presenting in the third trimester, monitoring of maternal cardiovascular status and foetal growth and well-being are essential until delivery. Stable cases may be managed medically as per guidelines for heart failure with some exceptions. Angiotensin converting enzyme inhibitors (ACE-I), aldosterone receptor antagonists, and sacubitril-valsartan and heparin are avoided due to foetal teratogenicity while ivabradine is avoided due to a lack of data on its safety in pregnancy (7,10). Beta-blockers, if indicated, can be utilized but foetal monitoring for foetal growth restriction and foetal bradycardia is necessary (10). Loop diuretics, hydralazine, nitrates, digoxin and low-molecular-weight heparin may be used in pregnancy (10). Maternal cardiovascular stability and foetal well-being are the two factors that determine the timing and mode of delivery. A spontaneous vaginal delivery at term is possible for patients with stable cardiac status and a healthy foetus (7). Any deterioration in maternal cardiovascular stability or foetal compromise warrants in hospital management and an early delivery (7).

Patients presenting in the post-partum period, may be managed as per heart failure guidelines with no specific restrictions, as most medications used in heart failure are compatible with lactation (10).

Due to the role of prolactin in the pathophysiology of PPCM, bromocriptine, a dopamine D2 antagonist that suppresses the secretion of prolactin, has been suggested for treatment of PPCM. A 2021 meta-analysis involving eight studies, showed that use of bromocriptine was beneficial in terms of improved survival rates and LVEF but cautioned that large-scale randomized controlled trials were lacking (13). As such, it is vital that use of bromocriptine and its side effects on suppression of breast feeding be discussed with patients prior to initiating treatment.

There is a lack of evidence and consensus on breast feeding for patients diagnosed with PPCM. The Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy position statement discourages breast feeding (7). On the other hand, the European Society of Cardiology Study group discourages breast feeding only in patients with severely impaired systolic function (14). Small-scale studies have demonstrated the safety and possible benefits of breast feeding in patients with PPCM (10,14–16).

It is imperative that PPCM be diagnosed and treated early as it is associated with significant adverse outcomes such as cerebrovascular events leading to brain injury, cardiopulmonary arrest, pulmonary oedema, thromboembolic events and even death (7). The LVEF is a reliable predictor of adverse outcomes and recovery,

where recovery can take up to 18 months. Recovery rates are higher in the first three to six months (7).

Cardiac status and LVEF are important considerations in planning future pregnancies. Rates of recurrence are higher in subsequent pregnancies with higher rates of adverse outcomes if the LVEF has not recovered prior to conception (7). Therefore, the management of contraception is important. Intrauterine contraceptive devices are preferred due the benefits of long-term reversible contraception; however, other progesterone-only containing contraceptives are acceptable alternatives (7,17). Oestrogen-containing contraceptives are avoided due to the risks of thromboembolism while barrier methods are highly discouraged due to risks of failure (7,17).

TAKOTSUBO CARDIOMYOPATHY

Definition

TC also known as stress induced cardiomyopathy, broken heart syndrome or apical ballooning syndrome is defined as "new onset left ventricular dysfunction with variable wall motion abnormalities in the absence of significant coronary artery disease" (18,19). The condition was first encountered in Japan in 1983 and subsequently described by Sato et. al in 1990 (18,20). The term is derived from the shape of the heart during systole observed on echocardiogram, which resembles a Japanese octopus fishing pot, called a "takotsubo" (18,21). To date, there are no reports of TC occurring in pregnancy in Malaysia.

Pathophysiology

TC seems to stem from a transient ischemic cardiac event prompted by heightened sympathetic stimulation, ultimately halted by the activation of myocardial survival pathways (18,22). The enhanced sympathetic stimulation results in a surge of catecholamines which may have direct toxic effects on cardiomyocytes, resulting in multivessel epicardial spasm and microcirculatory dysfunction (18,23).

Clinical presentation

TC may present at any time during the antenatal or post-partum period. A study conducted in the United States by Mogos et. al showed that TC was most common in the post-partum period with it being more common after caesarian section (24,25). Any stressful event capable of evoking a significant psychological and emotional response may trigger TC (18,19). Patients with pre-existing psychiatric conditions are at higher risk of TC and its recurrence (19,26). Physical triggers ranging from physical activities, medical procedures, drugs and numerous medical conditions, including pregnancy itself can precipitate TC (18). While the range of possible risk factors and triggers are extensive, Ghadri et. al highlights that an absence of risk factors does not make the diagnosis less likely (18).

Patients with TC usually present with severe retrosternal chest pain, diaphoresis, palpitations or symptoms of heart failure (19). Physical examination findings will be either normal or consistent with heart failure. The differential diagnosis for patients presenting with these symptoms include other possible causes of chest pain including but not limited to an acute myocardial infarction and pulmonary embolism, heart failure (especially if there is an underlying cardiac disorder) and peripartum cardiomyopathy.

Diagnosis

As the presentation of TC, may mimic that of an acute myocardial infarction and PPCM, distinguishing the three poses a diagnostic challenge for the primary care doctor. Initial investigations must include an electrocardiogram and cardiac biomarkers, both readily available in the primary care setting, and will demonstrate results consistent with an acute myocardial infarction (Table II). As such, both investigations, while crucial, will not assist in differentiating the two.

An echocardiogram will demonstrate regional wall motion akinesia involving a large area of the left ventricle, usually the apical region resulting in acute apical ballooning associated with dilatation of the mid-ventricular area, extending beyond the territory of a single coronary artery; and reduced ejection fraction (20 – 45%), (27). There are several subtypes of TC based on echocardiographic findings, the description of which is beyond the scope of this article (18).

The gold standard of diagnosing TC is coronary angiography with left heart ventriculography, which despite the use radiation is considered safe in pregnancy (18,28).

Numerous guidelines exist that outline the diagnostic criteria for TC demonstrating the complexities and challenges involved in diagnosing this condition (29). The Modified Mayo Clinic Criteria for Takotsubo Cardiomyopathy is commonly used while the InterTAK Diagnostic Criteria, proposed by a consensus from the European Society of Cardiology is the latest (6,18,30).

While most diagnostic criteria require the use of invasive investigations to confirm the diagnosis of TC, the InterTAK Diagnostic Criteria offers a novel scoring system that excludes the need for invasive investigations (18,31,) making it a convenient scoring system to be used in the primary care setting. It utilizes seven clinical parameters, each of which are assigned a score that totals to 100 and predicts probability of TC with high sensitivity and is able to distinguish TC from acute coronary syndrome with high specificity (31).

Management

There are no guidelines on the management of TC in pregnancy, with supportive management and monitoring

of both mother and foetus for complications being the mainstay of treatment. (32). Patients with TC are at risk of developing complications such as left ventricular outflow tract obstruction, pulmonary oedema, acute heart failure and cardiogenic shock; and should be managed accordingly (19,30). Foetal complications include side of effects from medications used, preterm birth and fatal outcomes such as miscarriage, still birth and neonatal death (19,30).

For patients presenting during the antenatal period, consideration must be given to the timing and mode of delivery, which will largely be determined by maternal cardiovascular status and foetal well-being. TC is associated with adverse foetal outcomes in patients presenting during the antenatal period compared to those presenting in the post-partum period (30). The onset of TC was noted to be more rapid with a risk of developing pulmonary oedema in patients who developed TC after caesarian section (30).

Taking into consideration the above factors, the primary care doctor should focus on early detection and prompt referral of patients with suspected TC. Management should involve a multidisciplinary team involving the cardiologist, obstetrician and neonatologist (19).

Despite the significant complications that may arise, TC has a good prognosis with majority of patients obtaining complete recovery of systolic dysfunction (6). Recovery of normal cardiac function is usually achieved within four to eight weeks, with one systematic review citing 3.66 ± 4.59 weeks (19,30).

A review of the literature did not yield any recommendations for breast feeding in patients with TC. In view of the good prognosis and recovery, patients should be encouraged to breast feed if there are no serious maternal cardiovascular complications or contraindications due to medication use.

As most patients with TC tend to make a complete recovery, the combined oral contraceptive is considered safe, while other forms of contraception except barrier methods, are acceptable (17).

CONCLUSION

Although both PPCM and TC are uncommon conditions, primary care physicians should consider these when patients develop sudden cardiac symptoms in the peripartum period. PPCM differs from TC, characterized by heart failure. Treatment mainly involves standard heart failure medications, with no specific therapies yet. Takotsubo cardiomyopathy, on the other hand, is a non-ischemic form of cardiomyopathy characterized by transient left ventricular dysfunction resembling a heart attack. Cardiac enzyme release is minimal with no evidence of obstructive coronary artery disease. This

review emphasizes the importance of recognizing and managing PPCM and TC across healthcare teams.

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