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RESEARCH ARTICLE

PERIPARTUM CARDIOMYOPATHY: FOCUS

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ABSTRACT

The the peripartum cardiomyopathy (PPCM), is a rare cause of cardiomyopathy occurring at the end of pregnancy or during the months following the birth. This condition can be life-threatening and is characterized by significant left ventricular dysfunction and heart failure It's a diagnosis of exclusion, which must be confirmed by the measurement of systolic function nearly always less than 45%. It is a rare pathological entity whose pathophysiological mechanism in question remains poorly understood. Clinically, it is a sudden heart failure with unpredictable evolution and a hight risk of refractory cardiogenic shock justifying management in cardiovascular resuscitation. The treatment is essentially symptomatic, the prognosis is closely related to the complete recovery of cardiac function.

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INTRODUCTION

Definition and Epidemiology: Peripartum cardiomyopathy (PPCM) is defined by the European Society of Cardiology as "Left ventricular systolic dysfunction, without identifiable etiology, occurring in late pregnancy or in the months following delivery, responsible for a clinical presentation of heart failure. It is a diagnosis of exclusion; the diagnosis must be confirmed by the measurement of systolic function which is less than 45% "(Sliwa *et al*., 2010). This incidence is very variable according to the ethnicity and the environment. varies from 1/400 in Haiti to 1/4000 in the United States (Sliwa *et al*., 2005; Fett *et al*., 2005; Mielniczuk *et al*., 2006), in Europe the incidence is unknown, hence the recent creation of a register by the European Society of Cardiology.

Risk factors: The risk factors associated with CPP are advanced maternal age (> 30 years), multiparity, twin pregnancies, African descent, obesity, pre-eclampsia, pregnancy-induced hypertension, and prolonged tocolysis (> 4 weeks) (Elkayam *et al*., 2005; Fett *et al*., 2005; Sliwa *et al*., 2000).

Pathophysiology: Several hypotheses have been described but none could be affirmative.

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Infectious and immunological: Myocardit is of viral origin in particular, several studies have reported the presence of genomes of viral origin on myocardial biopsies (Parvovirus B19, HSV 6, EBV or CMV) associated with local tissue inflammation. Apart from possible acute viral infections, it is also possible that viruses that remain latent in the myocardium may be reactivated because of the relative immunodepression of pregnancy (Midei *et al*., 1990; Bultmann *et al*.,?). In addition, a significantly elevated antibody level, anti-actin, anti-myosin and anti-adenine nucleotide translocator (ANT) IgG type was found in patients with PPCM compared with the control group (Pearson *et al*., 2000).

Ion disruption: hypokalemia with significant loss of urinary potassium postpartum, which induces excessive depolarization of the myocardial cell membrane and cellular atony: role of elevation of glucocorticoids and aldosterone (Bertrand and Langlois, 1975).

Genetic background: A genetic predisposition is possible, with in particular mutations described on the gene of tit in (van Spaendonck-Zwarts *et al*., 2014)

Corticosteroid decline: The significantly elevated incidence of PPCM associated with twin pregnancies suggests a metabolic origin. Indeed, this type of pregnancy is characterized by a greater secretion of corticosteroids resulting in a greater sodium -water retention and an increase in the LV post-loading more significant (Bertrand, 1986; Bertrand *et al*., 1985).

HTA and pre-eclampsia: Through several mechanisms

- An increase in post-load VG secondary to an increase in peripheral resistance (Ben Letaifa *et al*., 1999)
- An alteration of vascular reactivity with endothelial dysfunction may thus promote the occurrence of acute heart failure (Ben Letaifa *et al*., 1999).
- Increased release of pro-inflammatory cytokines (IL-1, IL-6, TNF-a ...) in patients with gestational hypertension (Matthiesen *et al*., 2005). Those that will lead to changes in the hemodynamic balance responsible for VG dysfunction.
- Inflammatory origin: The histological study of the myocardium in women with a PPCM showed an aspect of inflammatory myocarditis in 29 to 78% of cases (Murali *et al*., 2005).
- Cardiac physiological stress secondary to pregnancy: confirming the increase in prolactin degradation and the production of anti-angiogenic peptides, with also the increase of anti-angiogenic factors of the sFLT1 type at the end of pregnancy (Patten *et al*., 2012).

This anti-angiogenic climate, occurring in late pregnancy, probably constitutes a substrate on which any aggression (viral infection, inflammation, autoimmunity...) or any latent structural anomaly (toxicity of anthracyclines, unknown familial heart disease) could trigger left ventricular dysfunction.

Clinical presentation: The symptoms are those of an overall heart failure, the deterioration of the patient's clinical condition can happen quickly in a few days, the ECG is most often normal or may show sinus tachycardia. A clinical score has recently been developed to assist in the diagnosis of frustrated forms (Table 1) (Fett, 2009). Transthoracic echocardiography is the main exam to confirm ventricular dilation and LV dysfunction

The diagnostic criteria:

- Cardiac failure during the last month of pregnancy, or within 5 months after delivery, in the absence of an identifiable cause, or arguments for pre-existing cardiomyopathy.
- Left ventricular systolic dysfunction (LVEF <45%) and ventricular dilatation (> 27mm / m2). Note that it is always necessary to eliminate a cardiopathy prior to pregnancy from where the interest of a careful interrogation. Eliminate old exposure to anthracyclines.

Diagnostic traps: Two main diagnoses to eliminate, myocardial infarction (chest pain, ECG, troponin ....) And myo-pericarditis (viral context, inflammatory syndrome, MRI).

Prognosis

Factors of bad prognosis: The importance of initial left ventricular dysfunction (<30%) and left ventricular dilatation (> 27mm / m2) are pejorative for short-term recovery (Chapa *et al*., 2005; Duran *et al*., 2008). The presence of left ventricular thrombi in the acute phase would also be a factor of non-recovery (Amos *et al*., 2006).

Risk of recurrence in the subsequent pregnancy: The risk of recurrence of a PPCM during a subsequent pregnancy depends mainly on the recovery of the first episode. Patients who have completely normalized their LVEF and have a satisfactory contractile reserve during stress echocardiography do not reoffend, while those with LVEF less than 45% recur in almost 67% of cases. This second episode will be all the less well tolerated as the recovery is partial (Fett *et al*., 2010). Except in case of complete recovery, a new pregnancy is contraindicated (Regitz-Zagrosek *et al*., 2011).

Treatment: The initial medical treatment is that of heart failure, taking into consideration the obstetric situation. It begins most often with diuretic treatment, secondarily associated with ACE inhibitors and beta-blockers (privilege metoprolol), introduced after a few days, once the patient's clinical status has stabilized. If cardiomyopathy occurs before delivery, ACE inhibitors are contraindicated. However, there is no contraindication to breastfeeding (captopril and enalapril) and they can be started soon after delivery. If left ventricular function is less than 35%, anticoagulation should be initiated, as the risk of thromboembolic complications is greater than that of idiopathic dilated cardiopathy (Box *et al*., 2004). As for ACEIs, AVKs are contraindicated during the last trimester of pregnancy, and should not be prescribed in case of decompensation before delivery. Under these conditions, the patient will be placed on heparin. In cases of refractory cardiogenic shock, this treatment is associated with the usual circulatory assistance methods, such as the intra-aortic balloon counter-pulse or the in-situ ECLS-type extracorporeal assistance (Vanzetto *et al*., 2009) or waiting for left ventricular functional recovery ("bridge to recovery") or cardiac transplantation ("bridge to transplantation"), whose prognosis is identical to other etiologies of heart failure (Rasmusson *et al*., 2007).

The place of bromocriptine: By inhibiting the secretion of prolactin, bromocriptine prevents its degradation into anti-angiogenic peptide and thus prevents systolic dysfunction in late pregnancy. Treatment may reduce mortality and improve recovery of cardiac function, but these are preliminary results and the use of bromocriptine cannot be routinely recommended.

Prescription Criteria for Bromocriptine

- Elements in favor of the prescription of bromocriptine
- Diagnosis before term or in the month following delivery.
- LVEF <30% and VG dilation> 60mm.
- Shock or low peripheral flow without favorable response to inotropes.
- Lack of significant clinical improvement after 15 days of IEC / betablocker treatment.
- New pregnancy with a history of peripartum heart disease (treatment to start the last month of pregnancy).

No indication of bromocriptine

- Diagnosis beyond 1 month after delivery, especially if no breastfeeding.
- LVEF> 40% or VG diameter <55.
- Favorable response to inotropic treatment.

Contraindications

- Arterial thromboembolic ATCD.
- Presence of VG thrombus.
- Uncontrolled hypertension, eclampsia.

Obstetrical management: There are no systematic recommendations for the obstetric management of patients presenting with a CMPP chart before the end of pregnancy. The decision of preterm delivery will be discussed on a case-by-case basis depending on the severity of left ventricular dysfunction, the initial course of treatment, and possible fetal distress. Newborns from beta-blocking patients should be monitoredfor24 to48hoursbecause of the risk of hypoglycemia, bradycardia and respiratory failure Long-term management relies on conventional treatment of chronic heart failure, with the combination of beta-blocker, ACE inhibitors, and loop diuretics depending on the extent of left ventricular dysfunction and possible signs of sodium-water retention.

Conclusion

PPCM is a rare but serious form of heart failure whose pathogenesis is still poorly understood. The prognosis is closely related to the complete recovery of cardiac function. The treatment is mainly symptomatic, but several therapies are being evaluated offering hope for this pathology of heavy morbi-maternal mortality.

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