



Peripartum Cardiomyopathy Overview, Pathophysiology, Management

An Essay

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List of Abbreviations

ACE	Angiotensin converting enzyme
AHF	Acute heart failure
AMI	Acute myocardial infarction
AP	Action potential
A-V VALVES	Atrio ventricular valves
ARVD	Arrhythmogenic right ventricular dysplasia
AVN	ATRIOVENTRICULAR NODE
BNP	B natriuretic peptide
CAMP	Cyclic adenosine mono phosphate
CCB	Calcium channel blocker
CXR	Chest x ray
CHF	Congestive heart failure
CMR	Cardiac magnetic resonance

DCM	Dilated cardiomyopathy
DBP	Diastolic blood pressure
DDD	Dual demand chamber
Fas/Apo-1	ligand found on cell-surface proteins that plays a key role in apoptosis
HCM	Hypertrophic cardiomyopathy
HOCM	Hypertrophic obstructive cardiomyopathy
EMBs	Endomyocardial biopsy specimens
IABP	Intra-aortic balloon pump
IHD	Ischemic heart disease
ICD	Implantable cardioverter defibrillator
MHC	Major histocompatibility complex
LVAD	Left ventricular assisted device
NSVT	Non-sustained ventricular tachycardia
LVOT	Left ventricular outflow tract

LVEF	Left ventricular ejection fraction
PCR	Polymerase chain reaction
PPCM	Peripartum cardiomyopathy
PACM	Pregnancy associated cardiomyopathy
SBP	Systolic blood pressure
RCM	Restrictive cardiomyopathy
SCD	Sudden cardiac death
SAM	Sub aortic membrane
TIA	Transient ischemic attack
TTE	Trans thoracic echo

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ABSTRACT

PPCM affects previously healthy women in the final month of pregnancy and up to 5 months after delivery. The diagnosis is based on 4 criteria:

- *development of HF in the last month of pregnancy or within 5 months of delivery.*
- *Absence of an identifiable cause for HF.*
- *Absence of recognizable heart disease prior to the last month of pregnancy.*
- *echocardiography (TTE) criteria of left ventricular systolic dysfunction (left ventricular ejection fraction, LVEF <45%), fractional shortening of <30%, or both.*

No single explanation of the pathogenesis of PPCM is relevant for all women; the disease has a multifactorial origin such as viral myocarditis, abnormal immune response to pregnancy, maladaptive response to hemodynamic stresses of pregnancy, stress-activated cytokines, excessive prolactin excretion, and prolonged tocolysis and the precise mechanism is still not understood.

Administration of diuretics is indicated in presence of symptoms of fluid retention, inotropic agents used in presence of peripheral hypo-perfusion with or without congestion or pulmonary edema refractory to diuretics and vasodilators. In

addition to conventional therapy new lines include milrinone (phospho-diesterase III inhibitor), Levosimendan (calcium sensitizer), bromocriptine, and pentoxifyllin (inhibitor of TNF production). Temporary mechanical circulatory assistance indicated in patients not responding to conventional therapy. This include an intra-aortic balloon pump and a left ventricular assist device, and/or extracorporeal membrane oxygenation.

Several outcomes in PPCM are present. Some women remain stable for long periods, while others get worse slowly. Others get worse very quickly and may be candidates for a heart transplant. Furthermore, the death rate may be as high as 25 - 50%. Persistent cardiomyopathy is associated with poor prognosis.

KEYWORDS: PPCM, DCM, SELENIUM DEFICIENCY, BROMOCRIPTINE, LEVOSIMENDAN, PENTOXYPHYLLINE

INTRODUCTION

Peripartum cardiomyopathy (PPCM): also known as postpartum cardiomyopathy is an uncommon form of heart failure that happens during the last month of pregnancy or up to five months after giving birth. Peripartum cardiomyopathy is a dilated form of the condition, which means the heart chambers enlarge and the muscle weakens in the absence of any other cause of heart failure. This causes a decrease in the percentage of blood ejected from the left ventricle of the heart with each contraction that leads to less blood flow and the heart is no longer able to meet the demands of the body's organs for oxygen, affecting the lungs, liver, and other body systems. (*AHA. 2015*)

The exact cause of peripartum cardiomyopathy (PPCM) is unknown, but the usual causes of systolic dysfunction and pulmonary edema should be excluded. Precise mechanisms that lead to PPCM remain poorly defined. Many etiological processes have been suggested: **viral myocarditis, abnormal immune response to pregnancy, maladaptive response to hemodynamic stresses of pregnancy, stress-activated cytokines, excessive prolactin excretion, and prolonged tocolysis.** (*Ntusi NB, et al.2009*).

The objective of peripartum cardiomyopathy treatment is to keep extra fluid from collecting in the lungs and to help the heart recover as fully as possible. Many women recover normal heart function or stabilize on medicines. Some progress to severe heart failure requiring mechanical support. Patients with systolic dysfunction during pregnancy are treated the same as patients who are not pregnant. (*AHA. 2015*).

AIM OF THE ESSAY

The aim of this essay is to discuss the pathophysiology, diagnosis and management of peripartum cardiomyopathy in intensive care unit.

Chapter 1

*Anatomy and
Physiology of the heart
and changes during
pregnancy*

Anatomy of the heart:

The heart is a muscular organ in humans and other animals, which pumps blood through the blood vessels of the circulatory system. Blood provides the body with oxygen and nutrients, and also assists in the removal of metabolic wastes. The heart is located in the middle compartment of the mediastinum in the chest. (*Taber. et al, 2009*)

Commonly the right atrium and ventricle are referred together as the right heart and their left counterparts as the left heart. In a healthy heart blood flows one way through the heart due to heart valves, which prevent backflow. The heart is enclosed in a protective sac, the pericardium, which also contains a small amount of fluid. The wall of the heart is made up of three layers: epicardium, myocardium, and endocardium. (*Cecie Starr. et al, 2012*)

The heart pumps blood through the body. Blood low in oxygen from the systemic circulation enters the right atrium from the superior and inferior venae cavae and passes to the right ventricle. From here it is pumped into the pulmonary circulation, through the lungs where it receives oxygen and gives off carbon dioxide. Oxygenated blood then returns to the left atrium, passes through the left ventricle and is pumped out