# *Cardiomyopathy in pregnancy*

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# *Peripartum cardiomyopathy*

**(LVEF < 45 %)**

Heart failure in the last month of pregnancy or within 5 months postpartum.

The absence of another identifiable cause of HF

The absence of recognizable heart disease prior to the last month of pregnancy

- ***DCM criteria in*** echocardiography;
  - -LV EF<45%
  - -LDEDD>117% predicted
  - -Familial HTX positive

# predisposing factors PPM

- multiparty
- African ethnicity,
- smoking
- diabetes
- pre- eclampsia
- malnutrition
- advanced age
- teenage pregnancy
- Maternal cocaine use
- Oral tocolytic therapy

# pregnancy-associated cardiomyopathy

***Acquired or inherited*** diseases:

- PPCM
- Toxic cardiomyopathies
- HCM
- Dilated cardiomyopathy (DCM)
- Takotsubo cardiomyopathy
- Storage diseases
- Hypertensive cardiomyopathy

# *Potential causes of PPCM*

- viral myocarditis
- nutritional deficiencies
- Autoimmunity
- Micro chimerism
- hemodynamic stresses
- vascular dysfunction
- hormonal insults
- underlying genetics.

# *Genetic Association of PPCM*

- DCM in other male relatives may represent a form of familial DCM .
- found PPM in all DCM families.
- Genetic analyses revealed a mutation (*TNNC1*)
- segregating with disease in a DCM family with a member having PPCM.

# *presentation*

Majority of PPCM cases present postpartum mostly in the week after delivery:

**acute HF**

- Orthopnea
- tachycardia
- elevated JVP,
- pulmonary rales
- PE.
- third heart sound
- displaced apical impulse

**ventricular arrhythmias**

**cardiac arrest**

# *Biomarkers*

- BNP
- troponin
- microRNAs, specifically **miR146a**

  (can differentiate PPCM from other forms)

# *DDX of PPCM*

- Pulmonary embolism (resulting from the hyper coagulable peripartum period)
- Pneumonia hypersinsivity
- acute pulmonary edema from prolonged tocolysis
- preeclampsia
- cardiac(myocardial infarction or Takotsubo cardiomyopathy)

# *Treatment of heart failure in pregnancy*

- require joint cardiac and obstetric care, serial echocardiograms, serum B-type natriuretic peptide, and fetal ultrasound.

# *Acute Heart Filure*

- Meating for task force memberes in< 15 min
- Reffer to a tetiarry center
- Induction of laber in> 23 week

- O2 :  oxygen saturation remains above 95 %.
- furosemide (20–40 )  mg IV for patients with hypervolemia as starting dose
- diuretics should be avoided in the absence of pulmonary congestion, due to the potential reduction in placental blood flow
- IV nitroglycerin (starting at 10–20 up to 200 mg/min) in patients with (SBP) > 110 mmHg
- used with caution in SBP between 90 and 110 mmHg.

# *Cardiogenic shock*

- low cardiac output and hypo perfusion or hypotension
- cool/clammy skin
- low urine output (<0.5 ml/kg/h)
- mental status changes
- hepatic dysfunction

# *Cardiogenic shock*

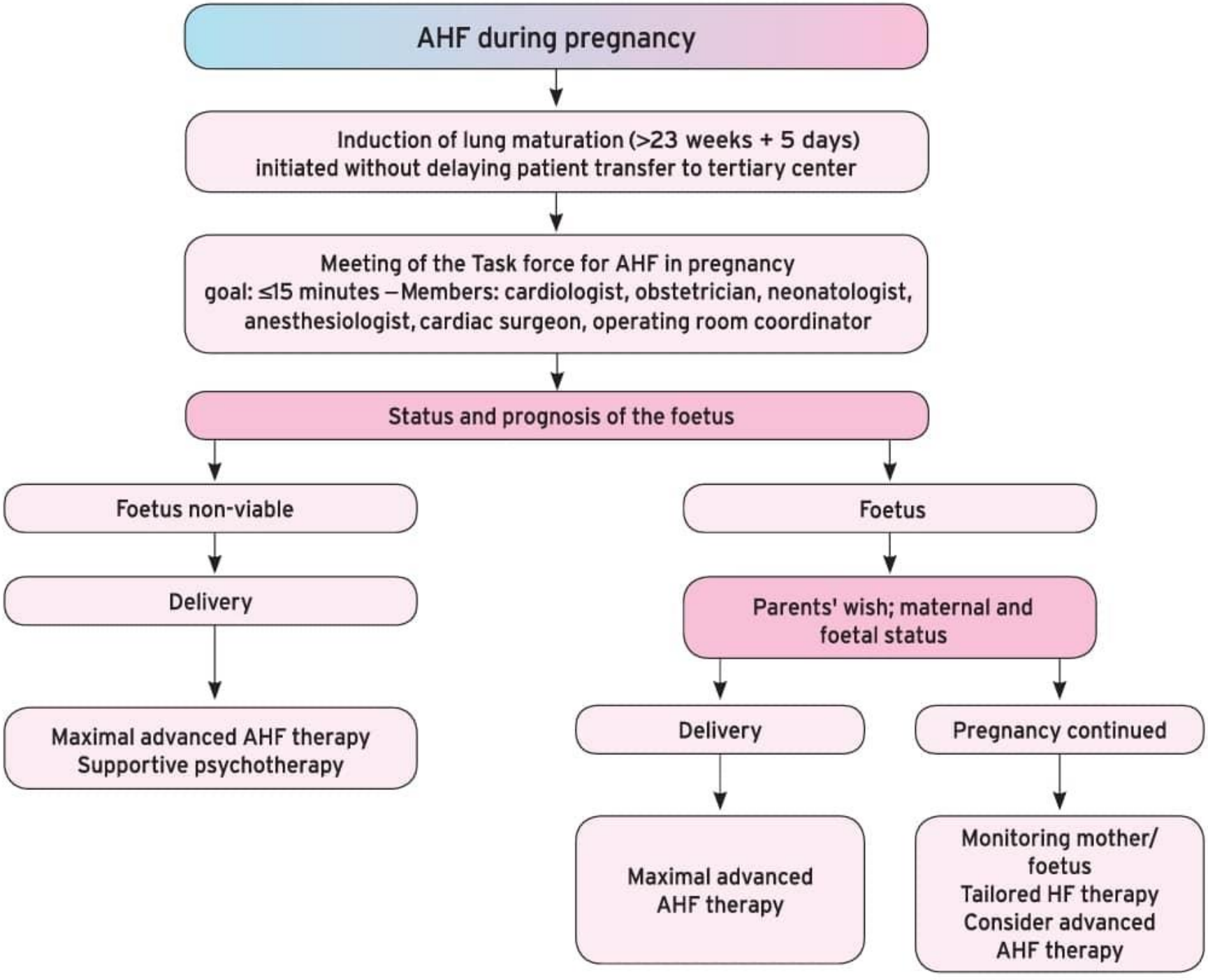
Urgent delivery by C/S (irrespective of gestation) with mechanical circulatory support immediately available.

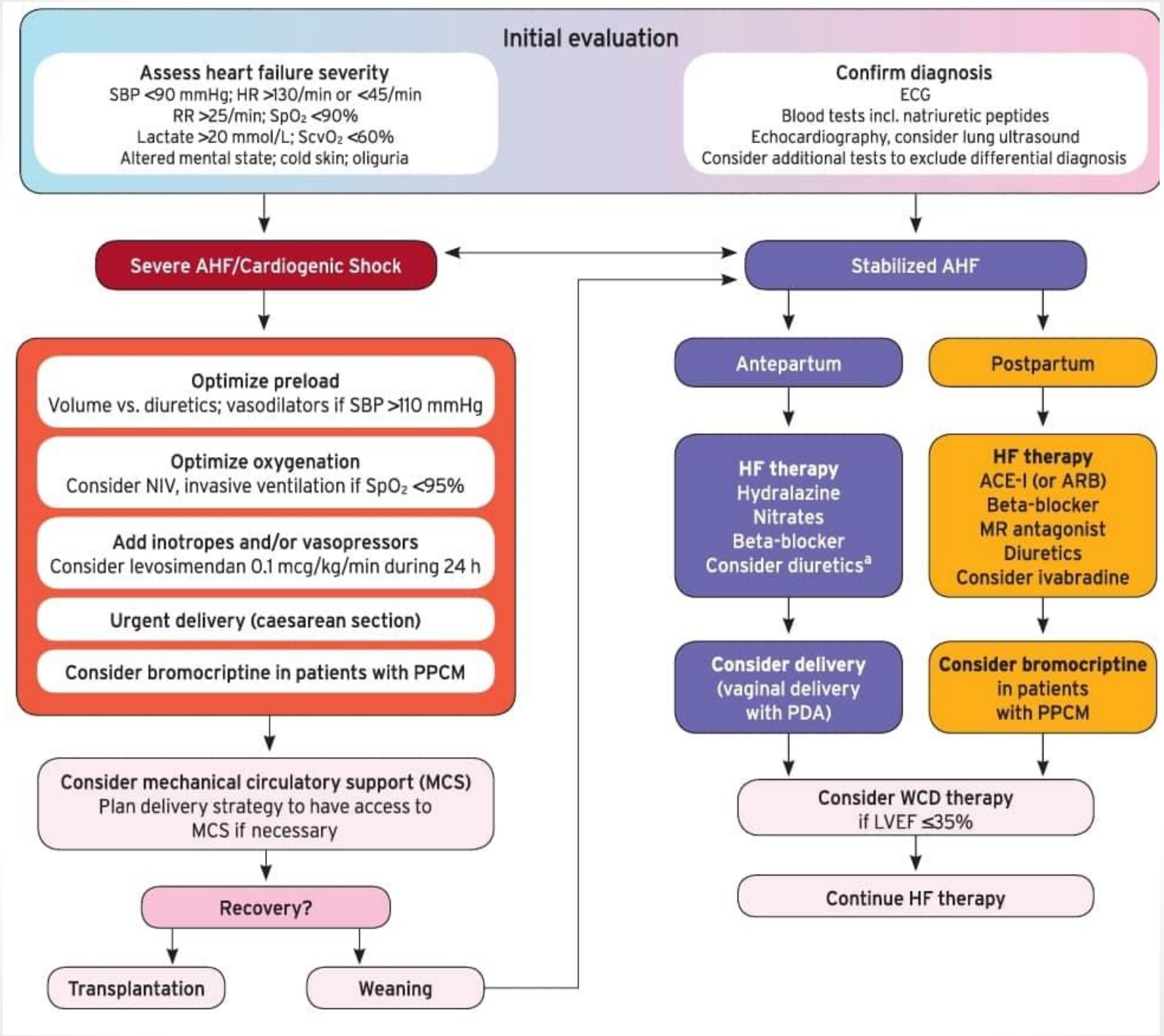
transferred early to a facility where mechanical circulatory support teams are available.

- PPCM patients are sensitive to the toxic effects of beta-adrenergic agonists, which should be avoided whenever possible

- norepinephrine should be avoided because their vasoconstrictor properties impair fetus pefusion

- Levosimendan may be the preferred inotrope


AHF during pregnancy
Induction of lung maturation (>23 weeks + 5 days) initiated without delaying patient transfer to tertiary center
Meeting of the Task force for AHF in pregnancy goal: ≤15 minutes – Members: cardiologist, obstetrician, neonatologist, anesthesiologist, cardiac surgeon, operating room coordinator
Status and prognosis of the foetus
Foetus non-viable
Delivery
Maximal advanced AHF therapy Supportive psychotherapy
Foetus
Parents' wish; maternal and foetal status
Delivery
Maximal advanced AHF therapy
Pregnancy continued
Monitoring mother/ foetus Tailored HF therapy Consider advanced AHF therapy


Initial evaluation
Assess heart failure severity
SBP <90 mmHg; HR >130/min or <45/min
RR >25/min; SpO2 <90%
Lactate >20 mmol/L; ScvO2 <60%
Altered mental state; cold skin; oliguria
Confirm diagnosis
ECG
Blood tests incl. natriuretic peptides
Echocardiography, consider lung ultrasound
Consider additional tests to exclude differential diagnosis
Severe AHF/Cardiogenic Shock
Stabilized AHF
Optimize preload
Volume vs. diuretics; vasodilators if SBP >110 mmHg
Optimize oxygenation
Consider NIV, invasive ventilation if SpO2 <95%
Add inotropes and/or vasopressors
Consider levosimendan 0.1 mcg/kg/min during 24 h
Urgent delivery (caesarean section)
Consider bromocriptine in patients with PPCM
Consider mechanical circulatory support (MCS)
Plan delivery strategy to have access to MCS if necessary
Recovery?
Transplantation
Weaning
Antepartum
Postpartum
HF therapy
Hydralazine
Nitrates
Beta-blocker
Consider diuretics[a]
HF therapy
ACE-I (or ARB)
Beta-blocker
MR antagonist
Diuretics
Consider ivabradine
Consider delivery (vaginal delivery with PDA)
Consider bromocriptine in patients with PPCM
Consider WCD therapy if LVEF ≤35%
Continue HF therapy

# *Chronic management of HF*

Both ***ARB*** and ***ACE inhibitors*** are contraindicated (risk category D) :

pose the risks of renal or tubular dysplasia, oligo hydramnion, growth retardation, ossification disorders of the skull, lung hypoplasia, contractures, large joints, anemia, and intrauterine fetal death.

- ***Spironolactone:***

(risk category D) can have anti androgenic effects and cause oral clefts during the first trimester and is therefore contraindicated

# *Chronic management of HF*

- combinations of hydralazine (risk category C) and isosorbide dinitrate (risk category B) may be used to treat cardiomyopathy)

- Beta blockers (risk category B) have not been shown to have teratogenic effects but may produce hypoglycemia and bradycardia

- treatment with ivabradine may be useful if the patient is not pregnant or breastfeeding.

# *Prognosis*

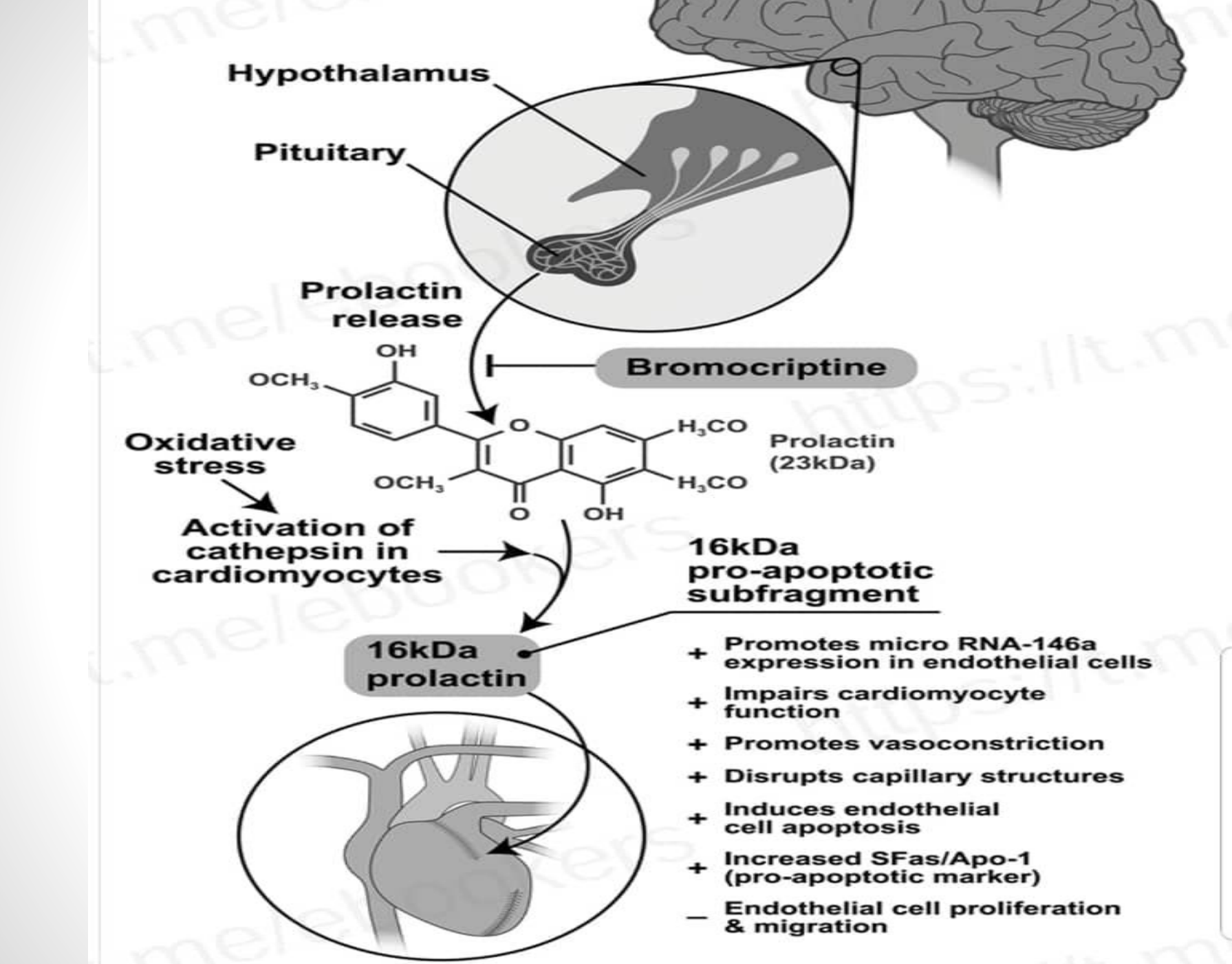
- LV EF< 30%
- LV end diastolic dimention >6 cm
- AF
- RV Dilation
- Fibrosis formation in CMR
- High level Pro BNP

# *Prognosis*

- a mortality ranging from 2.0% to 12.6%.
- 50%- 80% with PPCM LV EF returned to normal.
- When the EF has not recovered to >50–55%, subsequent pregnancy should be discouraged.

- Relapse of PPCM has been observed after rapid tapering of HF therapies.

- treatment should continue for at least 6 months after full recovery of LV function followed by gradual tapering


Hypothalamus
Pituitary
Prolactin release
Bromocriptine
Oxidative stress
Activation of cathepsin in cardiomyocytes
Prolactin (23kDa)
16kDa pro-apoptotic subfragment
16kDa prolactin
+ Promotes micro RNA-146a expression in endothelial cells
+ Impairs cardiomyocyte function
+ Promotes vasoconstriction
+ Disrupts capillary structures
+ Induces endothelial cell apoptosis
+ Increased SFas/Apo-1 (pro-apoptotic marker)
– Endothelial cell proliferation & migration

# Bromocriptine

- bromocriptine to standard HF therapy may improve LV recovery and clinical outcome in women with acute severe PPCM.

- Bromocriptine (2.5 mg once daily) for at least 1 week may be considered in uncomplicated cases.

- whereas prolonged treatment (2.5 mg twice daily for 2 weeks, then 2.5 mg once daily for 6 weeks) may be considered in patients with severe disease

- Urgent delivery irrespective of gestation duration should be considered in women with advanced HF and haemodynamic instability

- C/S is recommended with central anaesthesia. To prevent abrupt pressure or volume changes.

- epidural anaesthesia might be the method of choice but should be carefully titrated, guided by an expert anaesthetic team.

- In stable congestive HF, vaginal delivery is preferred with spinal/epidural analgesia

- In HF with reduced EF (HFrEF), breastfeeding is discouraged in more severe cases (e.g. NYHA III/IV).

- Stopping lactation reduces the high metabolic demand and enables early optimal HF

# THANK YOU