A brief introduction to cardiomyopathy Focus on postpartum cardiomyopathy

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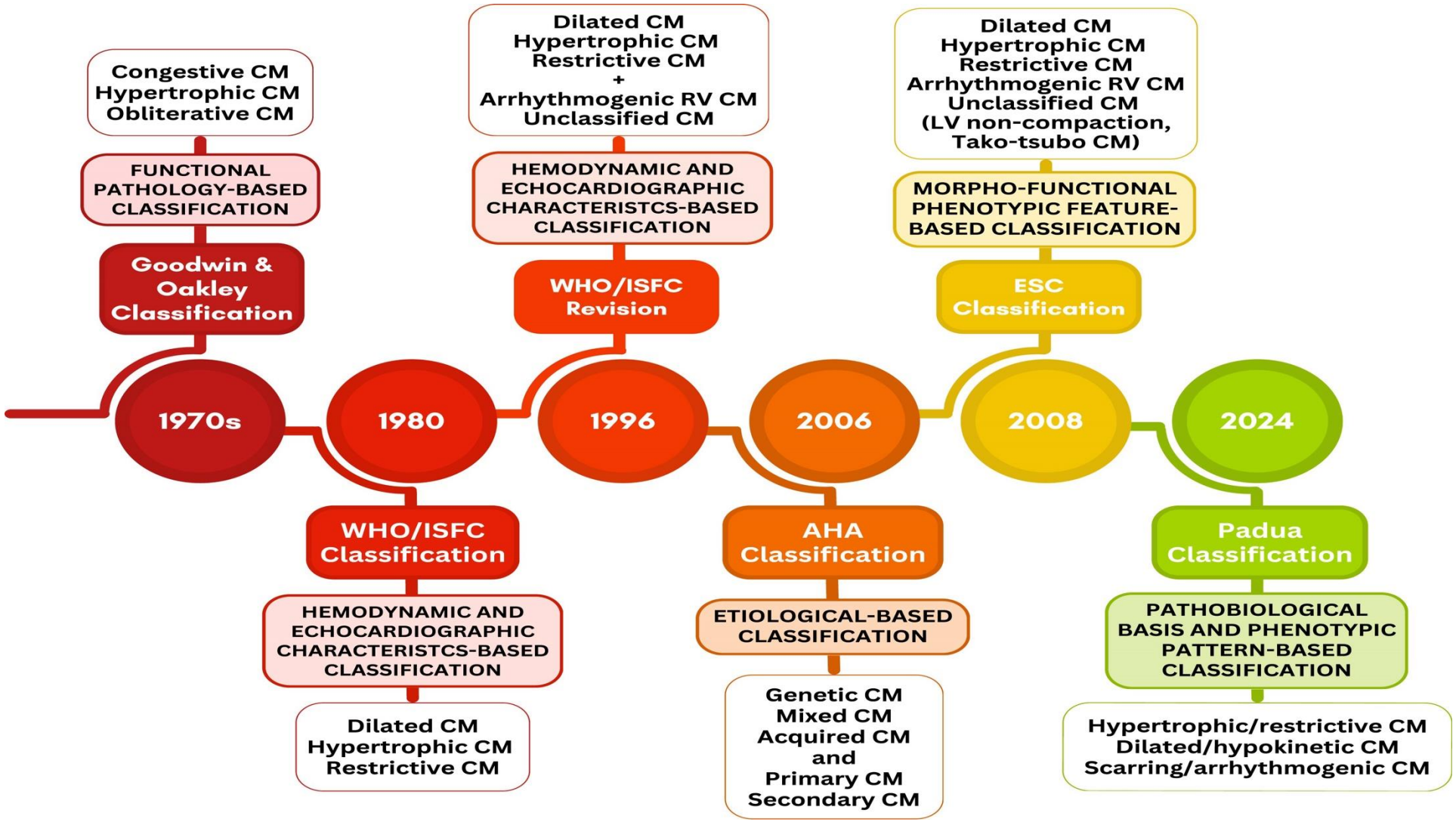
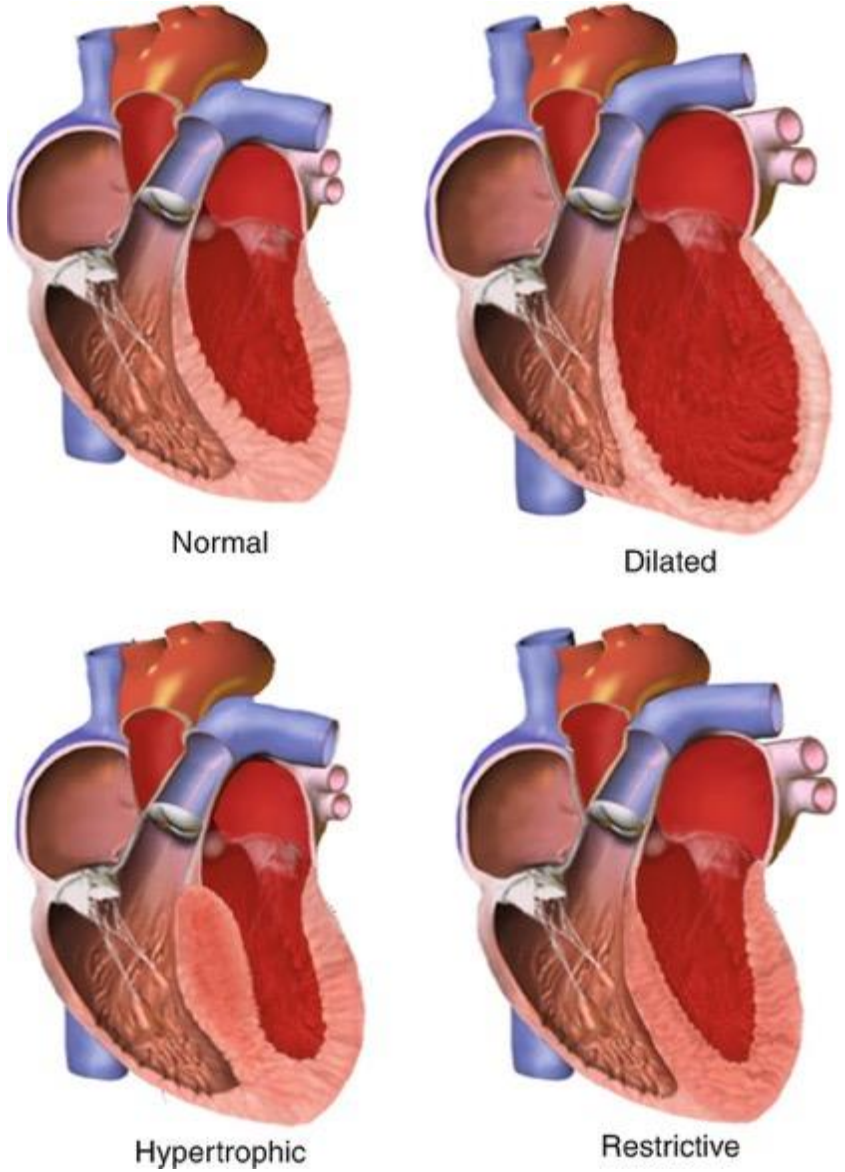
Topics

- Definition of cardiomyopathy
- A case presentation of peripartum cardiomyopathy
- Discussion on management & implication in primary care set up


1970s
Congestive CM
Hypertrophic CM
Obliterative CM
FUNCTIONAL PATHOLOGY-BASED CLASSIFICATION
Goodwin & Oakley Classification
1980
WHO/ISFC Classification
HEMODYNAMIC AND ECHOCARDIOGRAPHIC CHARACTERISTCS-BASED CLASSIFICATION
Dilated CM
Hypertrophic CM
Restrictive CM
1996
Dilated CM
Hypertrophic CM
Restrictive CM
+
Arrhythmogenic RV CM
Unclassified CM
HEMODYNAMIC AND ECHOCARDIOGRAPHIC CHARACTERISTCS-BASED CLASSIFICATION
WHO/ISFC Revision
2006
AHA Classification
ETIOLOGICAL-BASED CLASSIFICATION
Genetic CM
Mixed CM
Acquired CM
and
Primary CM
Secondary CM
2008
Dilated CM
Hypertrophic CM
Restrictive CM
Arrhythmogenic RV CM
Unclassified CM
(LV non-compaction, Tako-tsubo CM)
MORPHO-FUNCTIONAL PHENOTYPIC FEATURE-BASED CLASSIFICATION
ESC Classification
2024
Padua Classification
PATHOBIOLOGICAL BASIS AND PHENOTYPIC PATTERN-BASED CLASSIFICATION
Hypertrophic/restrictive CM
Dilated/hypokinetic CM
Scarring/arrhythmogenic CM


Normal
Dilated
Hypertrophic
Restrictive

What is cardiomyopathy – Padua Classification2024

Diseases primarily affecting the myocardium which are characterized by *permanent* structural and functional heart abnormality.

Those conditions are excluded in which the myocardial disorder is not a primary event but occurs secondarily to coronary artery disease, hypertensive heart disease, valvular disease, or congenital heart disease.

Although the original (prototype) cardiomyopathy phenotype is genetically determined, a number of non-genetic diseases (‘phenocopies’) may exhibit phenotypic features, which closely resemble those of the inherited aetiologic variant.

In a significant number of cases, the cause of the disease may be not-identifiable (‘idiopathic’). Accordingly, each cardiomyopathy phenotype is sub-classified into genetic and non-genetic variants

Non-genetic cardiomyopathies are further sub-divided into variants with an identifiable cause and idiopathic forms.

The heart muscle involvement may occur in isolation or part of a generalized multi-organ disease.

3 distinctive categories of cardiomyopathies

What has changed?

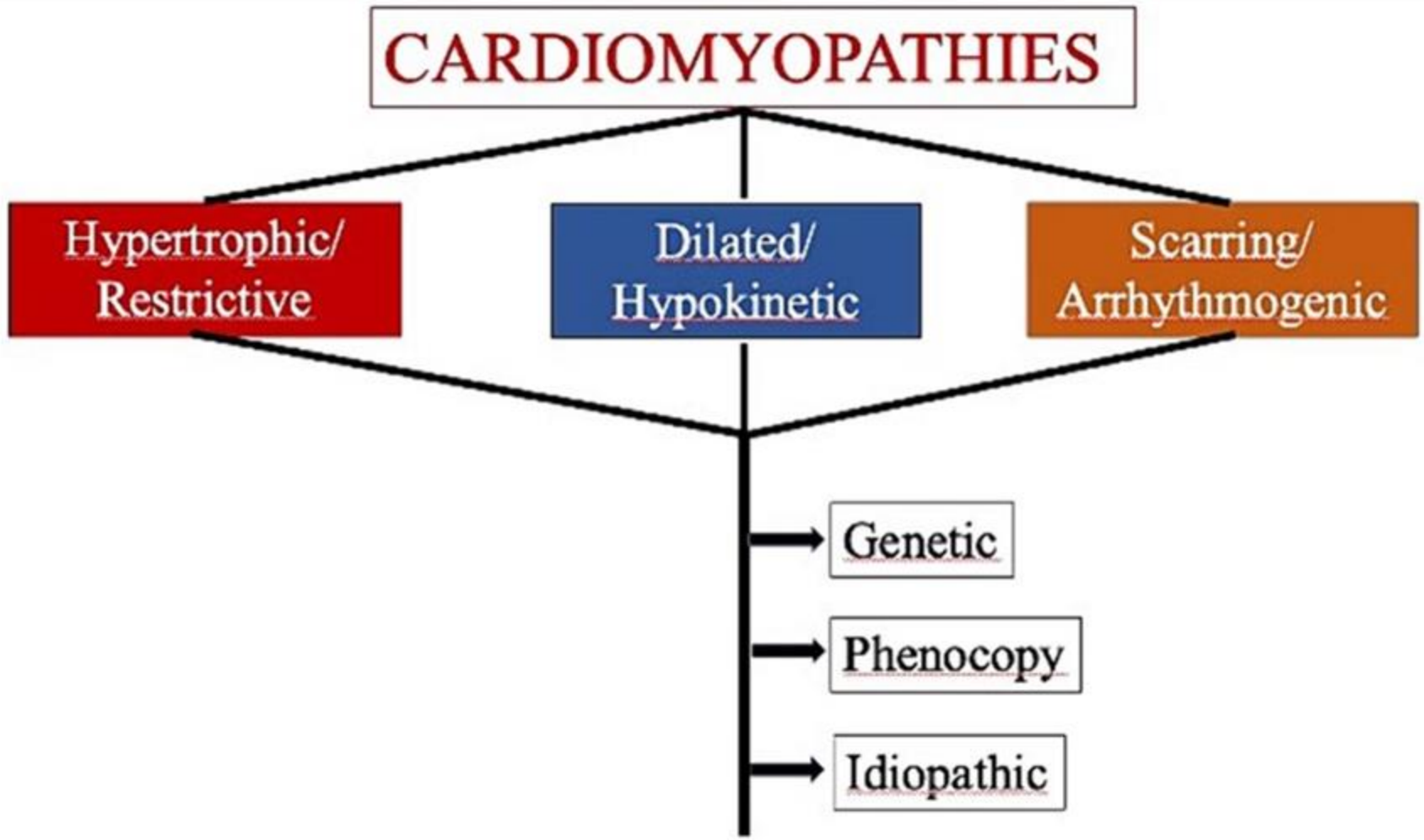
- Scientific advances in molecular biology and genetics
- use of contrast-enhanced cardiac magnetic resonance (CMR) for morpho-functional imaging and structural myocardial tissue characterization.
- cardiomyopathies that rely on the combination of the distinctive pathobiological basis (genetics, molecular biology, and pathology) and the phenotypic features (morpho-functional and structural ventricular remodeling)

3 different disease categories, each with a combined designation based on both ‘anatomic’ and ‘functional’ phenotypic features

Hypertrophic/restrictive cardiomyopathy (H/RC).

Dilated/hypokinetic cardiomyopathy (D/HC).

Scarring/arrhythmogenic (S/AC).


CARDIOMYOPATHIES
Hypertrophic/
Restrictive
Dilated/
Hypokinetic
Scarring/
Arrhythmogenic
Genetic
Phenocopy
Idiopathic

Hypertrophic/restrictive (H/RC) cardiomyopathies

Include genetic heart muscle disorders from genetically defective sarcomeric and non-sarcomeric proteins (storage and infiltrative diseases)

And

Non-genetic phenocopies, manifesting with the phenotypic pattern of LV diastolic dysfunction, with no or variable degree of ventricular hypertrophy.

Dilated/hypokinetic (D/HC) cardiomyopathies

Include genetic heart muscle disorders from a variety of defective genes encoding for proteins of cytoskeleton, sarcomere, nuclear envelope and intercalated discs.

And

Non-genetic phenocopies, manifesting with the phenotypic pattern of LV dilatation and/or systolic dysfunction

Scarring/arrhythmogenic (S/AC) cardiomyopathies

Include genetic heart muscle disorders from genetically defective intercalated discs/desmosomal proteins.

And

Non-genetic phenocopies, with the phenotypic pattern of non-ischaemic myocardial scarring of the LV, RV, or both, resulting scar-related ventricular arrhythmias and may lead to impairment of systolic function caused by myocyte death.

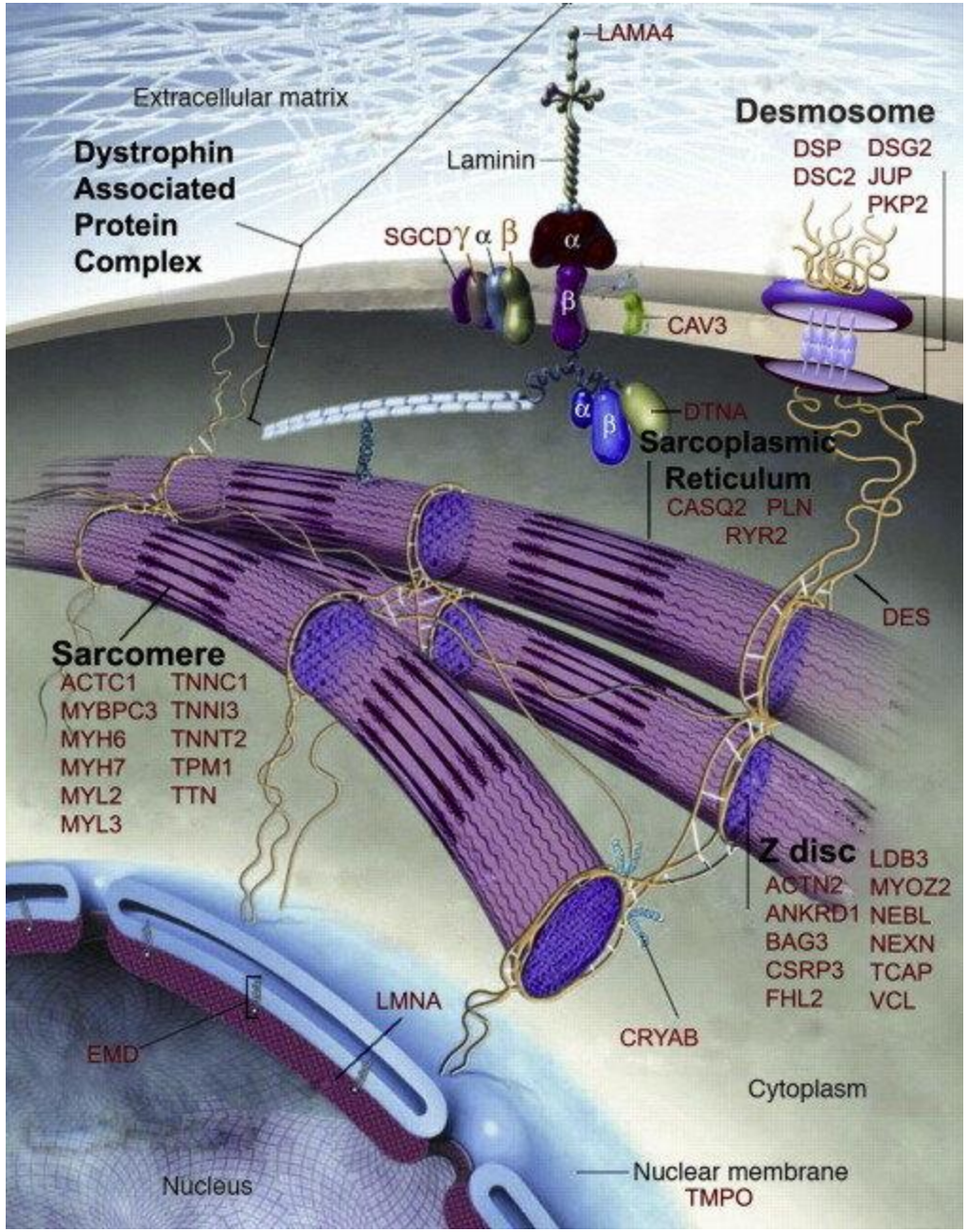
Not Included....

Acute myocarditis

Takotsubo syndrome, and left ventricular non-compaction.

Ventricular remodelling caused by electrical dyssynchrony and tachycardiomyopathies.

Primarily electrical abnormalities such as cardiac ion channel diseases are excluded from this definition.


LAMA4
Extracellular matrix
Desmosome
DSP DSG2
DSC2 JUP
PKP2
Dystrophin
Associated
Protein
Complex
Laminin
SGCD γ α β
α
β
CAV3
α
β
DTNA
Sarcoplasmic
Reticulum
CASQ2 PLN
RYR2
DES
Sarcomere
ACTC1 TNNC1
MYBPC3 TNNI3
MYH6 TNNT2
MYH7 TPM1
MYL2 TTN
MYL3
Z disc
ACTN2
ANKRD1
BAG3
CSRP3
FHL2
LDB3
MYOZ2
NEBL
NEXN
TCAP
VCL
LMNA
EMD
CRYAB
Cytoplasm
Nucleus
Nuclear membrane
TMPO

Types of ventricular remodelling

Diseases with **secondary** myocardial involvement: ischemic heart disease, valve disease, congenital heart diseases

Diseases with **primary** myocardial involvement: Cardiomyopathies

Ventricular myocardial scar w/or w/o regional/global dilatation

Post-MI ventricular scar (ischemic heart disease)

Scarring/arrhythmogenic cardiomyopathies

Regional ventricular contractile dysfunction and scar-related arrhythmias

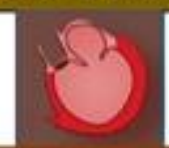
Ventricular hypertrophy

Pressure overload (Hypertension, Aortic valve stenosis)

Hypertrophic/restrictive cardiomyopathies

Impaired ventricular diastolic function and diastolic heart failure

Ventricular dilatation



Volume overload (Mitral/aortic valve regurgitation, congenital heart diseases)

Dilated/hypokinetic cardiomyopathies

Ventricular dilatation/dysfunction and systolic heart failure

Post-partum Cardiomyopathy

- Ms SL

Ms SL

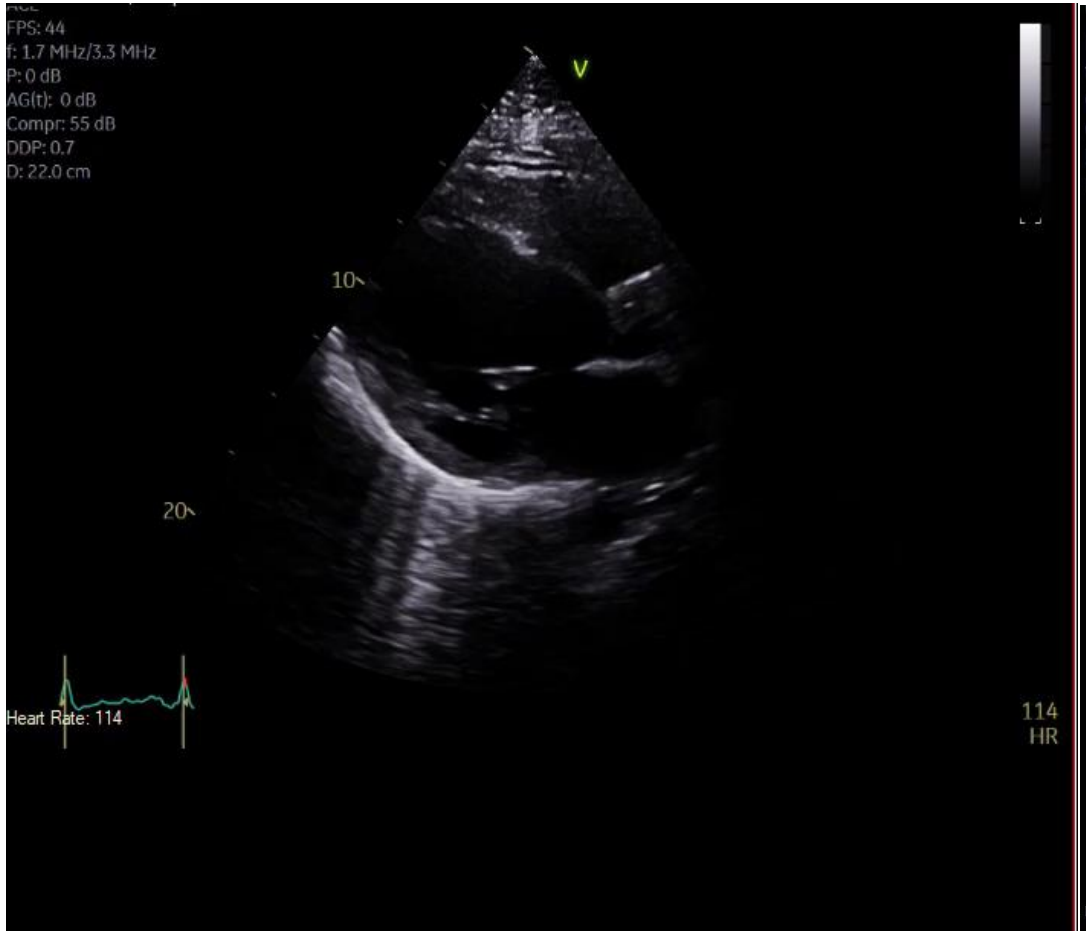
- 35 yo F
- Delivered 5th child on 28/10/24 at IGH
- Uncomplicated Spontaneous Vaginal delivery, however represented 2/11/24 requiring hysteroscopy & removal of retained placental products
- BMI 44.5, T2DM (poorly controlled – was on Metformin + insulin throughout pregnancy).
- Anxiety, depression, GORD, mild asthma, smoked 10-15/day, minimal ETOH, poor diet with high caffeine intake
- Tachycardic, with mitral murmur throughout both admissions
- Significant social stressors: partner not working
- Family history: Mother DCM – deceased age 60, Brother cardiac condition – unsure of nature
- Represented to IGH ED 22/1/25: SOB + chest pain > ?CAP

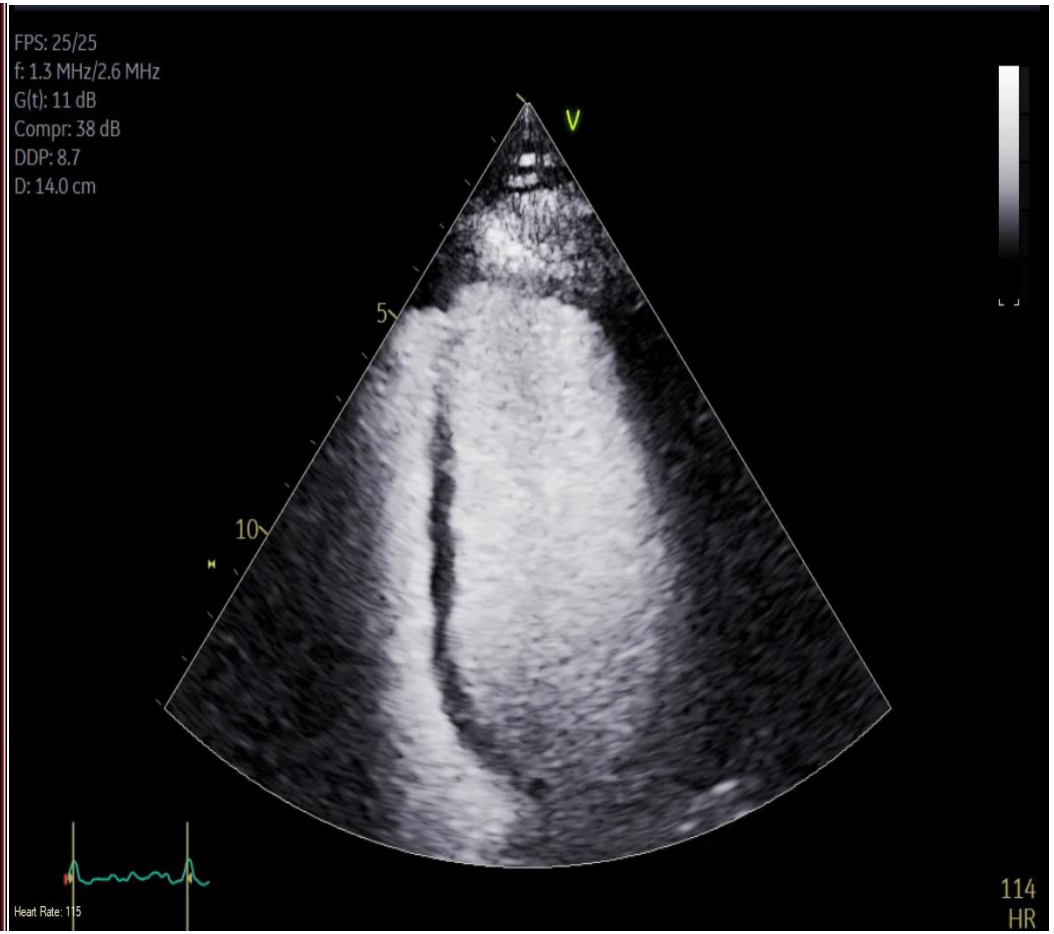
Investigations

3/2/25: TTE

- Dilated cardiomyopathy with severe left ventricular impairment (LVEF 15-20%)
- Severe functional mitral & tricuspid regurgitation
- Severe peri-partum cardiomyopathy
- 25/2/25 : CTPA – No PE
- Bloods – Lactate elevated at 470(N < 250), elevated transaminases, normal renal functions.

Echocardiogram





PPCM - Definition

NHLBI in the 1990s defined PPCM as heart failure that develops in the last month of pregnancy or up to five months postpartum with left ventricular systolic dysfunction (left ventricular ejection fraction (LVEF) <45% or fractional shortening <30%, or both).

However, a large proportion of patients who otherwise meet the criteria for PPCM present before 36 weeks' gestation, raising concerns that the NHLBI definition may be overly restrictive and lead to the underdiagnosis of PPCM.

In 2010 the European Society of Cardiology (ESC) defined peripartum cardiomyopathy as heart failure that occurs "towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found.

Risk Factors and Epidemiology of PPCM

- **Global Incidence:** PPCM is estimated to affect around 1 in 2,000 deliveries worldwide. However, its incidence varies significantly by region. It is most common in Nigeria, with an incidence of 1 in 100 births, while in Japan, it is much rarer at 1 in 20,000.
- **Western Countries and Ancestry**: In Western nations, the reported incidence ranges from 1 in 1,000 to 1 in 5,000 deliveries. It is significantly more prevalent in women of African ancestry.
- **Pregnancy-Related Conditions**: Certain pregnancy-related complications are strong risk factors:

  1. Hypertensive Disorders: Gestational hypertension and pre-eclampsia are associated with a three-fold

  increased risk of PPCM.

  2. A meta-analysis found that pre-eclampsia was present in 22% of PPCM cases, and other hypertensive

  disorders were present in 37%.
- **Multiple Gestations**: Carrying multiple babies (twins, triplets, etc.) is another strong risk factor, reported in 7% to 14.5% of PPCM cases.
- **Other Risk Factors**:

  Advanced Maternal Age: The risk of PPCM increases with maternal age, particularly from 30 years and older.

  Cocaine Use: Maternal use of cocaine is identified as a risk factor for the condition.

Wide Geographical Variation - EORP Registry data

- Worse outcomes in Africa & middle east.
- 67% Diagnosed postpartum

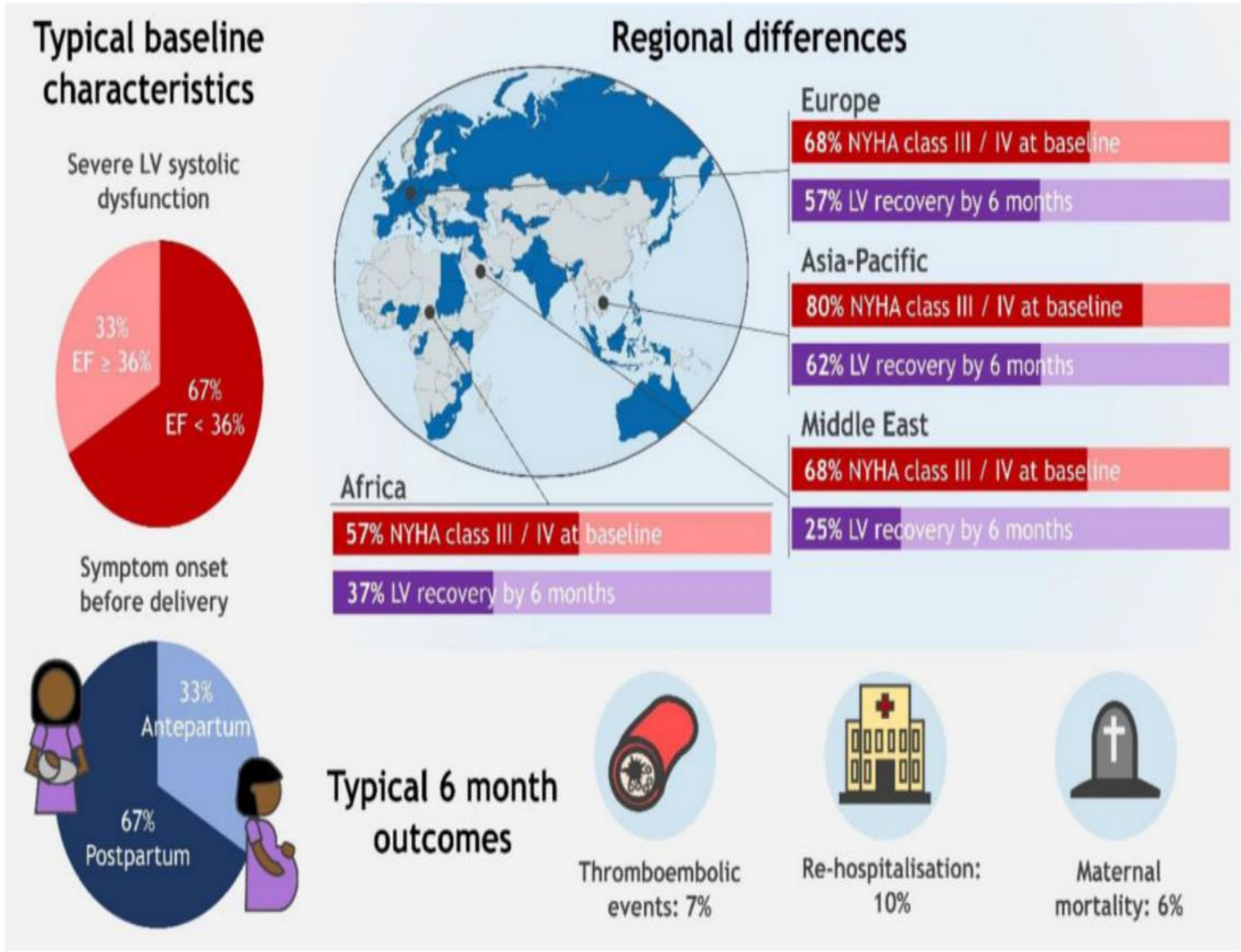


Figure 1 Baseline characteristics, 6-month outcomes, and regional differences observed in the peripartum cardiomyopathy (PPCM) EURObservational Research Programme (EORP). Analysis of 739 PPCM patients from 49 countries. In the map, countries in blue are those included

Pathophysiology

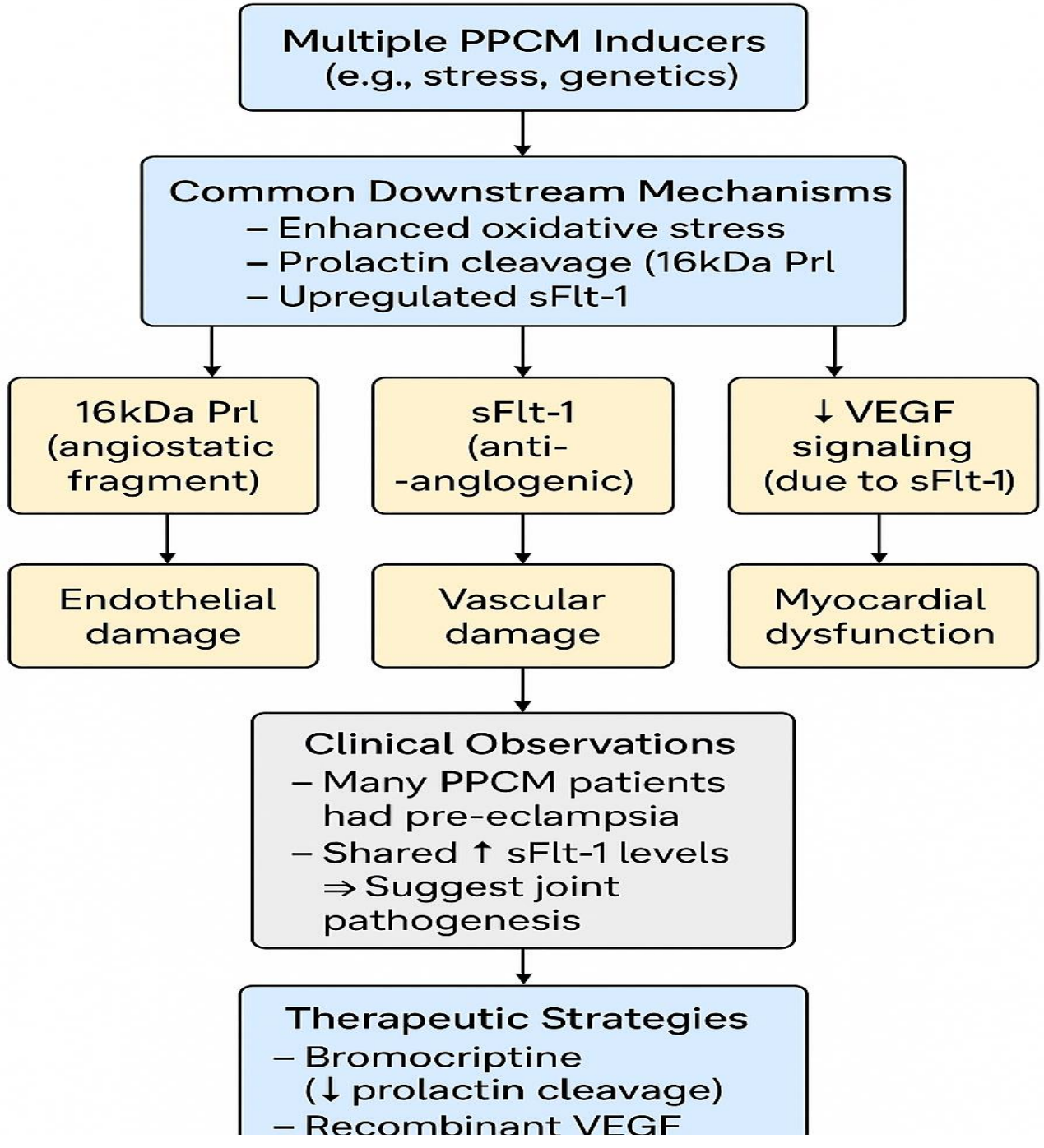
Oxidative stress & possible prolactin(16 KDa fragment) induced vascular dysfunction.

Increased levels of the soluble fms-like tyrosine kinase 1, an antagonist of the vascular endothelial growth factor, secreted by the placenta during the last months of pregnancy, it induced PPCM in mice.

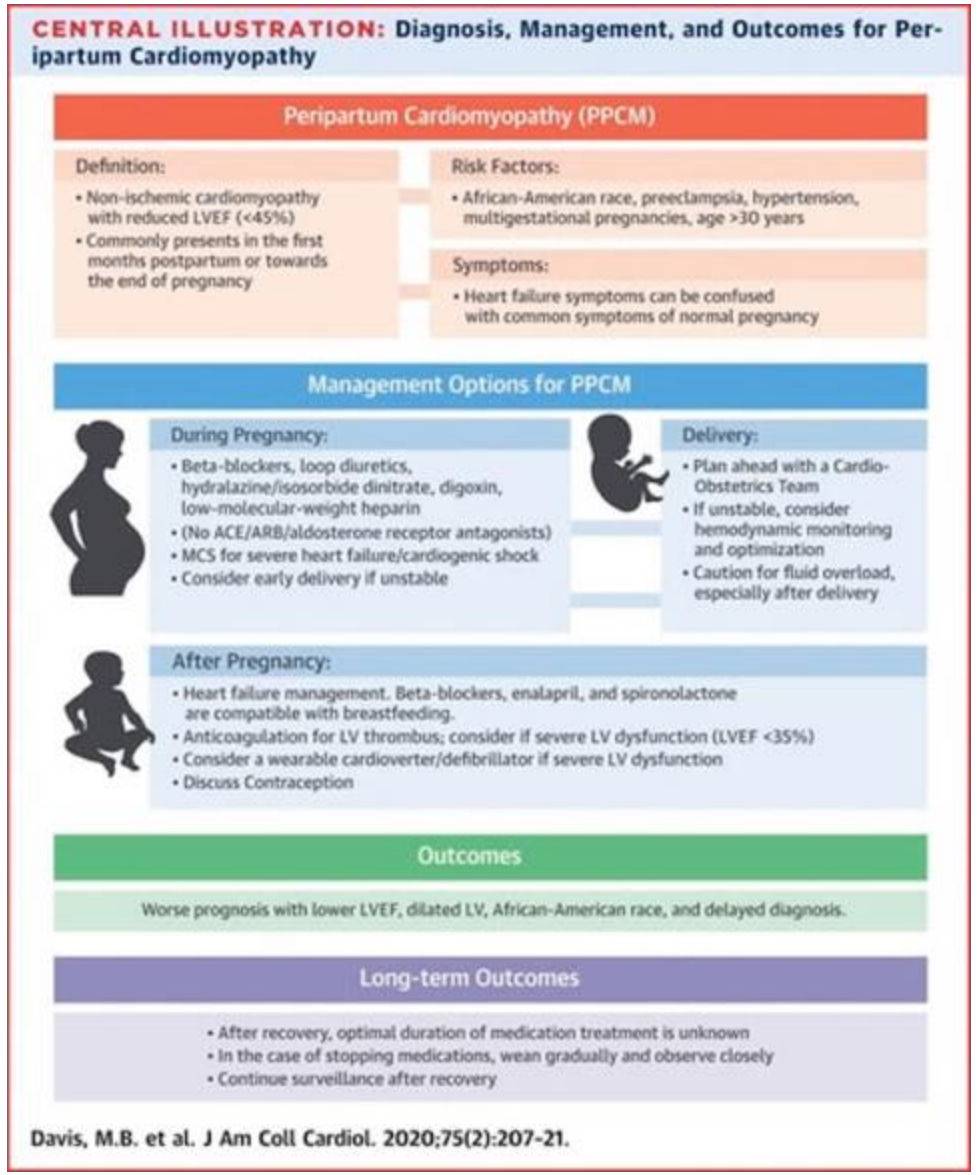
This molecule is also elevated in preeclampsia and multiple gestations, thus partially explaining the association between these conditions and PPCM.

Conversely, secretion of relaxin-2, a pregnancy-associated vasculoprotective hormone, is suppressed in PPCM.

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. Heterozygous loss-of-function genetic variants(Titin Gene mutation) in one of several genes associated with nonischaemic dilated cardiomyopathy have been encountered in almost 15 % of PPCM case.


Multiple PPCM Inducers
(e.g., stress, genetics)
Common Downstream Mechanisms
– Enhanced oxidative stress
– Prolactin cleavage (16kDa Prl
– Upregulated sFlt-1
16kDa Prl
(angiostatic
fragment)
sFlt-1
(anti-
-anglogenic)
↓ VEGF
signaling
(due to sFlt-1)
Endothelial
damage
Vascular
damage
Myocardial
dysfunction
Clinical Observations
– Many PPCM patients
had pre-eclampsia
– Shared ↑ sFlt-1 levels
⇒ Suggest joint
pathogenesis
Therapeutic Strategies
– Bromocriptine
(↓ prolactin cleavage)
– Recombinant VEGF

CENTRAL ILLUSTRATION: Diagnosis, Management, and Outcomes for Peripartum Cardiomyopathy



Davis, M.B. et al. J Am Coll Cardiol. 2020;75(2):207-21.

Genetics Testing

- Significant genetic Overlap with Dilated Cardiomyopathy (DCM)
- **Role of TTN Mutations**: Truncating mutations in the titin (TTN) gene, a well-known cause of DCM, are found in 10–15% of PPCM patients
- **Other Shared Genes**: Other DCM-associated genes, including DSP, FLNC, and BAG3, are also present in PPCM patients
- "**Multiple Hit" Model**: PPCM is likely triggered by a "multiple hit" model. A genetic predisposition, which may not cause disease on its own (low penetrance), is combined with environmental or physiological stressors, such as the hemodynamic and hormonal changes of pregnancy
- **Low Penetrance**: The penetrance of TTN truncating mutations is very low (95% of carriers show no cardiac disease), suggesting that cardiac function is preserved in the absence of additional stressors.

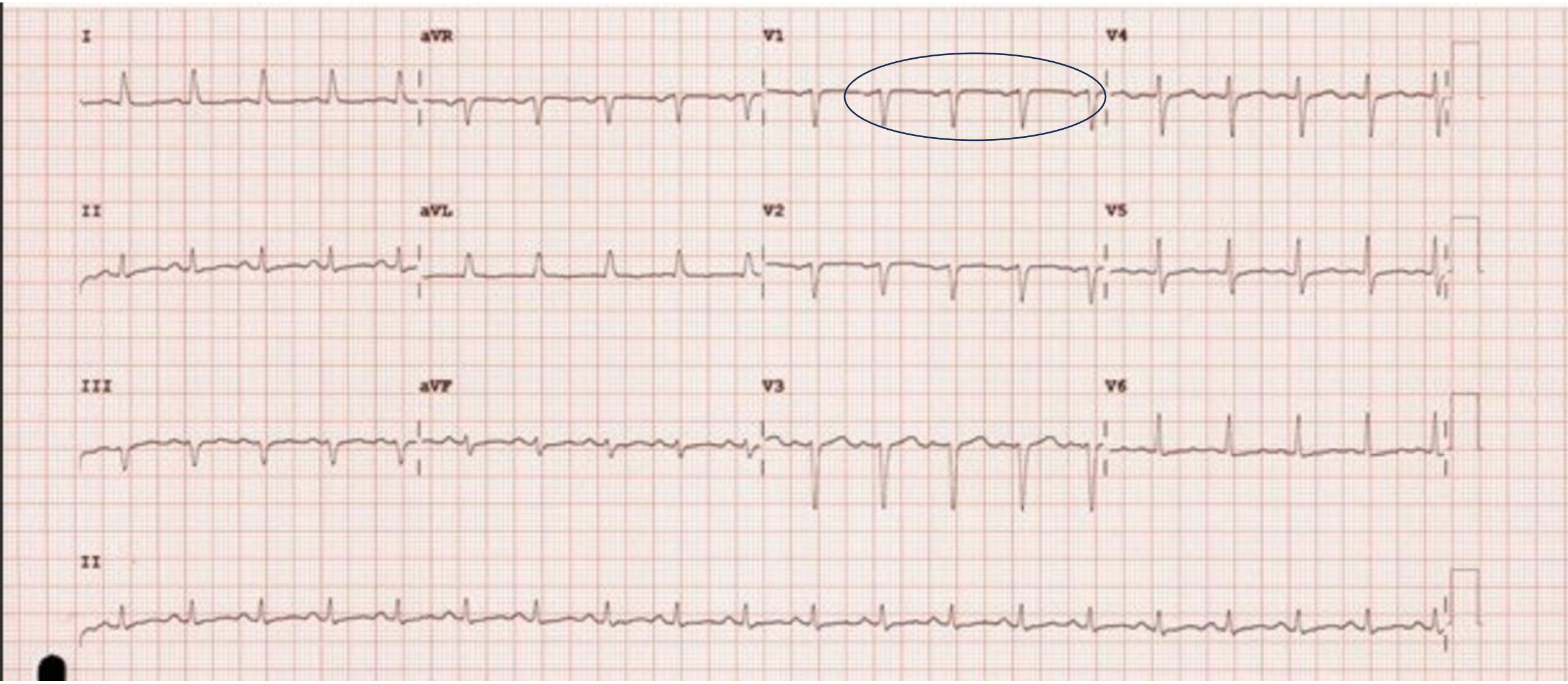
Clinical Implications:

- Genetic findings may impact prognosis and management (e.g., TTN mutations may suggest a worse prognosis; FLNC and DSP mutations may require aggressive anti-arrhythmic care).
- Identifying a pathogenic variant in a patient allows for genetic testing and monitoring of family members.
- Despite these findings, there are currently no formal recommendations for genetic testing in PPCM patients.
- Yet, genetic testing may be considered in cases of PPCM, especially in cases of familial aggregation of cardiomyopathies, for prognosis and to facilitate family screening.

Diagnostic Evaluation

- **Initial Action**: Urgent assessment by a specialist is required for any peripartum woman showing signs or symptoms of heart failure.
- **Electrocardiogram (ECG)**: Abnormalities are found in almost all PPCM patients. T wave inversion, BBB, varying AV block, ST depression, sinus tachy/ bradycardia. **LAA**(negative deflection of P wave at its terminal > 40 msecs is associated with poor recovery of LV function.
- **Chest X-ray:** Can help rule out other causes of breathlessness (e.g.infection).Commonly shows an enlarged heart/ congestion/ normal.
- **Echocardiography:**
- Confirms cardiac dysfunction and quantifies its severity.
- Excludes alternative causes of heart failure (e.g., congenital heart disease, valve disease).
- Typically shows enlarged left ventricular volumes and a mean left ventricular ejection fraction (LVEF) around 30%.
- A comprehensive assessment of the right heart is crucial, as reduced right ventricular function is a predictor of poor outcomes.
- Useful for assessing the presence of LV thrombus

- **Cardiac Magnetic Resonance Imaging (MRI):**
- Useful for excluding other conditions like myocarditis, left ventricular non-compaction, or infiltrative diseases.
- **Routine Lab Tests:**
- Routine blood work can identify reversible factors like anemia and assess for end-organ damage.
- Measuring natriuretic peptide levels is recommended.
- **Genetic Testing:**
- Not yet routine clinical practice but may be considered, especially if there is a family history of cardiomyopathies.
- The identification of a disease-causing mutation has clinical relevance for both the patient and her family members, including offspring.
- When a pathogenic mutation is found, cascade screening of relatives is recommended to identify other carriers who may need follow-up and routine cardiac screening.
- Patients must receive counseling before and after genetic testing to understand the implications.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Diagnosis of Exclusions

Differential Diagnosis	Considerations
Takotsubo cardiomyopathy	Echocardiogram may show classic apical ballooning
Familial cardiomyopathy	Family history, genetic testing
Pre-existing cardiomyopathy	History of HF prior to pregnancy; prior echo studies with low LVEF before pregnancy
Recurrent peripartum cardiomyopathy	Ask about symptoms of HF that occurred after a prior pregnancy
Pre-eclampsia	Preserved systolic function on echocardiogram
Hypertrophic cardiomyopathy	Left ventricular hypertrophy, LVOT obstruction, preserved systolic function, genetic testing
Myocarditis	Consider if viral prodrome, histological diagnosis, fulminant presentation
Arrhythmogenic right ventricular cardiomyopathy	Consider with family history, genetic testing, echocardiographic findings
Left ventricular noncompaction	Echocardiographic and CMR findings
Chemotherapy-related cardiomyopathy	History of chemotherapy, particularly doxorubicin
Valvular heart disease	Echocardiographic findings; congenital aortic stenosis; mitral stenosis from rheumatic heart disease in endemic country. Patients with PPCM may also have valve disease, i.e., mitral regurgitation
Congenital heart disease	May be diagnosed for the first time during pregnancy by echocardiography
Tachycardia-arrhythmia mediated cardiomyopathy	Consider if specific underlying rhythm abnormality. Note that sinus tachycardia may be secondary to heart failure during pregnancy
Hypertensive heart disease	Left ventricular hypertrophy; less common in young people unless very longstanding history of hypertension
Ischemic heart disease	Cardiovascular risk factors; angina; prior CAD; consider SCAD and MINOCA diagnoses
Cardiomyopathy related to other systemic medical diseases	Consider in the appropriate context, i.e., systemic lupus erythematosus, antiphospholipid syndrome, hemochromatosis
Cardiomyopathy related to other acute conditions	May consider if patient has other conditions such as sepsis, treatment in intensive care unit, post-respiratory arrest
Pulmonary embolism	Dyspnea, tachycardia with preserved LVEF

IGH admission Feb

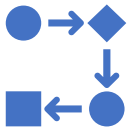
- Targeted therapy commenced – limited by hypotension
- FR 1.5L, daily weights
- Diuresis
- TPCH discussion 7/2/25 re: heart transplant/mechanical assist device: deemed not eligible due to BMI + smoking status
- NSVT

Limitations to therapy and complications for Ms SL

Represented to IGH 25/2/25 – hypotension - Adrenaline infusion

Withheld Bisoprolol, Frusemide, Spironolactone > re-referred to PICS

Restarted Bisoprolol 3/3/25, increased 17/3/25



Restarted MRA on alternate days 12/3/25, with plan to increase to daily in 3 weeks

Slow titration

Lifestyle, social + medication management

Social work: financial hardship

- Inability to afford medications resulting in anxiety

- At risk of significant health deterioration if non-compliant

- Community funding: $1298.51 (pharmacy, gas, electricity)
- Obtained approval to waive IGH pharmacy invoice

- Weight loss: would benefit from GLP-1 however unable to afford privately. Has T2DM however not on other therapies, therefore does not meet PBS criteria. HbA1C 5.6%

Contraception – rx for Levonorgestrel IUD

Smoking cessation

Medication compliance – dosette box

Readmission June 2025

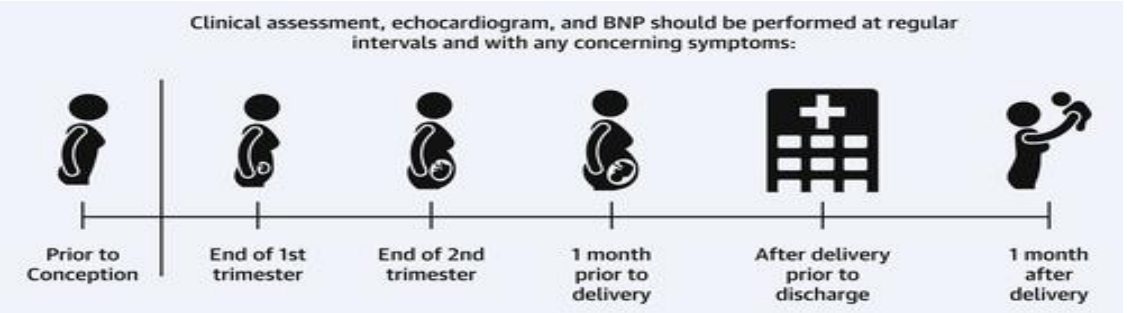
- Eminent cardiogenic shock
- ICU admission – commenced on inotropes.
- Rediscussed with The Prince Charles & accepted for further assessment regarding transplant/advance heart failure therapy.
- Deemed unsuitable & returned to IGH early July 2025
- Profoundly hypotensive, inotrope dependent.
- Genetic testing organised
- Palliative care input - currently at a hospice

Subsequent pregnancy

Subsequent Pregnancy	Recovered (LVEF ≥50%)	Nonrecovered (LVEF <50%)
Preconception or First Visit	Preconception risk counseling and follow-up planning. Clinical and LVEF reassessment off renin-angiotensin blocking agents for 3 months. Baseline echocardiogram and BNP/NT-proBNP level.	Preconception risk counseling including discussion of alternative ways to build a family. If pregnant and not considering termination: Close follow-up planning, stop renin-angiotensin blocking agents and switch to hydralazine/isosorbide dinitrate. Baseline echocardiogram and BNP/NT-proBNP level.
Maternal Risks	~20% have a relapse Severe deterioration is rare Mortality unlikely Rate of subsequent recovery is high	Higher risk of relapse ~50% show further deterioration in LV dysfunction Increased morbidity and mortality Premature delivery and abortion more common
Medications	Continue beta blocker therapy (metoprolol tartrate preferred). Yield of starting prophylactic beta blocker therapy unclear. Diuretics and hydralazine/isosorbide dinitrate in case of clinical or LV functional deterioration.	Continue beta blocker therapy (metoprolol tartrate preferred). Hydralazine/isosorbide dinitrate for hemodynamic and symptomatic improvement. Consider digoxin. Consider anticoagulation if severe LV dysfunction (LVEF <35%).
Follow-up	Close monitoring of symptoms during pregnancy and the postpartum period with repeat echocardiographic assessment of LV function and BNP/NT-proBNP level at the end of the 1st and 2nd trimesters, 1 month prior to delivery, after delivery prior to hospital discharge, 1 month postpartum, and at any time if symptoms develop.	
Labor and Delivery	Multidisciplinary team for planning; patient involved. Spontaneous vaginal delivery preferred unless fetal or maternal instability. Monitor for volume overload in the first 48 hours after delivery in cases of recurrent LV dysfunction.	Multidisciplinary team for planning; patient involved. Spontaneous vaginal delivery preferred unless fetal or maternal instability. Early delivery if further decrease in LV function and hemodynamic deterioration. Consider hemodynamic monitoring for optimization prior to delivery and monitoring during and after delivery. Monitor for volume overload in the first 48 hours after delivery.

Melinda B. Davis et al. *JACC* 2020; 75:207-221.

Proposed Echo follow up



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JACC JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY

Prognosis

- Recovery of LV function >50% after 6 months in 44%(13-25% middle east, 37% Africa, 57% Europe). At 12 months ~ 58%.
- Mortality 1.4 -3.4% within 30 days. 8% at 6 months(11.5% Asia-pacific, 10.9% Africa, 0.7% Europe). At 12 months 9.8%(15.2% Africa), At 2 years 10%.

Adverse Prognostic Markers & Specific therapy

Worse NYHA class

LVEF<25%, LVEDD > 60 mm

QT Prolongation

Being from African ancestry

Known PPCMP with persistent LV dysfunction.

Multiparity

Age> 30-35 years or < 20 years

Diagnosis during pregnancy

RV involvement

Low systolic BP

Bromocriptine is showing some promise(ESC recommendation Class 2B,level of evidence B) - 2.5 mg BD for 8 weeks + GDMT. Ongoing trial REBIRTH – results awaited ~2029. Associated with increased risk in PPCM scenario.

Discussion – key points, Treatment & management

- **Multidisciplinary Approach:** Treatment for PPCM requires a team including cardiologists, obstetricians, and other specialists to manage all aspects of a patient's care.
- **Initial Treatment:** PPCM is treated similarly to other forms of systolic heart failure, with an emphasis on pregnancy-safe therapies such as dietary sodium restriction, diuretics, and beta-blockers.
- **Medication During Pregnancy:**
  - **Diuretics (furosemide and hydrochlorothiazide)** can be used at low doses with careful monitoring to avoid harm to the fetus.
  - **Beta-blockers (beta-1 selective)** can be used cautiously. Carvedilol is effective as it also helps decrease afterload.
  - **Hydralazine** is a safe vasodilator.
  - **Digoxin** is safe for pregnancy and can be used for its inotropic and chronotropic effects.
  - **Contraindicated Medications:** ACE inhibitors, MRAs, ARBs, and nitroprusside are unsafe during pregnancy. Beta-blockers are contraindicated during breastfeeding.
- **Anticoagulation:** Anticoagulation is generally not recommended unless the left ventricular ejection fraction is below 30-35% or atrial fibrillation is present due to the high risk of blood clots.
- **Delivery and Postpartum Care:**
  - The timing and method of delivery are decided based on the mother's clinical stability.
  - Breastfeeding is often recommended as many PPCM medications are safe for lactation.
  - **Family Planning:** Women must be counseled on the high risk of recurrence in future pregnancies. Contraceptive options like IUDs and subcutaneous implants are recommended, while combination oral contraceptives should be avoided in women with severe left ventricular dysfunction.
- **Genetic Counseling:** Genetic counseling and testing are recommended to identify genetic variants that may increase arrhythmia risk (e.g., in genes like *FLNC* and *DSP*) and to guide family screening.
- **Advanced Therapies:** In severe cases, treatments may include inotropic drugs, mechanical support like a left ventricular assist device (LVAD), or an implantable cardioverter-defibrillator (ICD).
- **Investigational Treatments:** Experimental therapies, such as bromocriptine (a prolactin inhibitor), are being studied based on the theory that PPCM is triggered by a harmful form of the prolactin hormone.

Prognosis and Outcomes

- **Recovery:** While over half of patients recover cardiac function, PPCM carries significant morbidity and mortality. Recovery typically happens within 3 to 6 months postpartum but can take up to 48 months.
- **Morbidity and Mortality:** PPCM is a leading cause of maternal mortality. Mortality rates can be as high as 20% globally, with higher rates among Black women in the United States and women in less developed regions.
- **Racial Disparities:** In the United States, **Black women** are **twice as likely as White women** to experience prolonged cardiac dysfunction. When recovery does occur, it takes them twice as long.
- **Prognostic Factors:**
  - **Favorable:** A good prognosis is associated with a smaller left ventricular size (diastolic dimension <5.5 cm), an ejection fraction above 30-35% at diagnosis, and non-African American ethnicity.
  - **Poor:** Poor outcomes are linked to a larger left ventricular size (diastolic dimension ≥ 6 cm), an ejection fraction below 35%, a delayed diagnosis, and African descent.
- **Future Pregnancies:** The recurrence rate of PPCM in subsequent pregnancies is high, ranging from 10% to 50%. Women are advised against future pregnancies if their ejection fraction remains low. If a subsequent pregnancy is desired, patients should wait at least five years after their heart function has normalized. It is advisable to go off all medications 3 months before conception = have an echo & proceed.
- **Differential Diagnosis**
- PPCM is a diagnosis of exclusion. This means other conditions with similar symptoms must be ruled out.
- Other possible diagnoses include pre-existing cardiac conditions that have worsened during pregnancy, Takotsubo cardiomyopathy, severe preeclampsia or eclampsia, pulmonary embolism, and amniotic fluid embolism syndrome.

Thank You