Cardiac disease in pregnancy

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PERIPARTUM CARDIOMYOPATHY

Definition (ESC, 2010):

Idiopathic cardiomyopathy presenting as heart failure due to LV systolic dysfunction.

Occurs towards the end of pregnancy or up to 6 months postpartum.

Exclusion diagnosis: no other identifiable cause of heart failure.

LV may not be dilated, but **EF <45%** is typical.

EPIDEMIOLOGY:

Western countries: 1 in 1,000–4,000 pregnancies.

Highest incidence:

Nigeria: 1 in 100 live births

Haiti: 1 in 300 live births

Ethnicity: Afro-Caribbean women more commonly affected, worse prognosis.

Risk Factors

1.Race: The disease more commonly manifests in women of Afro Caribbean lineage and carries a poor prognosis.
2.Obstetric: multiparity, multiple pregnancy, advanced maternal age (>30 years)
3.Medical:obesity, chronic hypertension,pre-eclampsia
Pre-eclampsia association:
22% of PPCM cases have pre-eclampsia
~4x higher than global general population prevalence (5%)
Suggests shared pathophysiology

Pathogenesis

Despite decades of research, the exact cause of PPCM remains unclear, but current evidence supports a multifactorial model rather than a single theory.

Historical Theories (Myocarditis)

Old hypothesis: Viral myocarditis was believed to be a cause.

Evidence against:

Biopsies at autopsy showed no difference in viral markers between PPCM and controls.

Cardiac MRI failed to show myocarditis-like features.

Hemodynamic Stress Theory

it has been speculated that the associated haemodynamic stress placed upon the heart may contribute to PPCM. The compensatory changes occur mainly in the second trimester, while the signs and symptoms of PPCM arise late in the third trimester,as well as intothe postpartum period. Therefore, the disease timeline does not correlate

Pathogenesis

Current Consensus – Multifactorial 'Two-Hit' Hypothesis

A two-hit model has gained most support:

Hit 1: Genetic predisposition

Hit 2: Vascular–hormonal insult during late pregnancy/postpartum

A) Genetic Factors

Strong regional & familial clustering suggests inheritance.

Mutations in genes such as TTN, TTNCI, BAG3, PTHLH, PGC-1α.

These regulate cardiac myocyte function→ mutations increase vulnerability to PPCM.

Pathogenesis

(B) Vascular–Hormonal Factors

STAT3 Pathway Dysfunction

Normally, STAT3 (transcription factor) is highly active in pregnancy & postpartum.

If STAT3 is reduced →

↑ Reactive Oxygen Species (ROS)

↑ Cathepsin D activity

Cathepsin D cleaves prolactin → 16-kDa prolactin fragment

🔴16-kDa prolactin fragment effects:

Endothelial dysfunction

Microvascular damage

↑ microRNA-146a, which disrupts signalling & promotes cardiomyocyte apoptosis

Seen in biopsy & transplant samples from PPCM women.

💊 Bromocriptine (prolactin antagonist) improved cardiac function in STAT3-deficient mice basis for clinical use.

Pathogenesis

Placental Factors (sFlt-1)

Placenta secretes sFlt-1 (soluble fms-like tyrosine kinase receptor 1) late in pregnancy.
Function: Anti-angiogenic → reduces bleeding risk during labour.
In normal women: sFlt-1 ↓ after delivery (placenta removed).
In PPCM women: sFlt-1 stays high.
Effects:
Inhibits VEGF & PlGF → impaired angiogenesis.
Causes endothelial dysfunction & microvascular damage.
Towards the end of pregnancy sFlt-1 increase as part of the adaptive process to minimise the likelihood of haemorrhage in labor and drastically reduce following childbirth (following removal of the placental source of sFlt-1), but persist in women with PPCM.
There is a well-known association between sFlt-1 and preeclampsia. The resultant endothelial dysfunction is thought to be a common factor between hypertension and PPCM co-existence

Pathogenesis

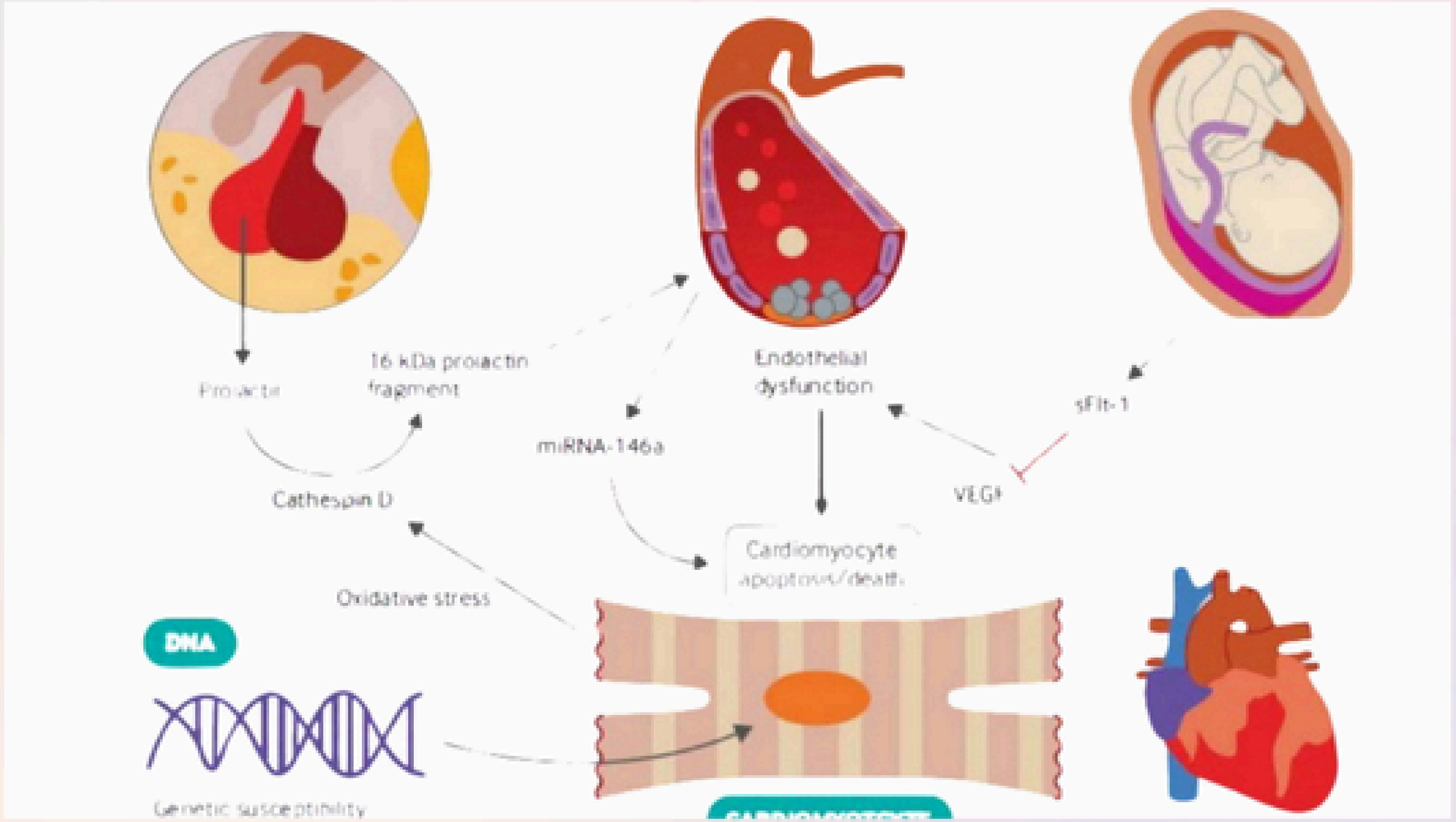


Figure 1. Two-hit hypothesis explanation for pathogenesis of peripartum cardiomyopathy.9 Secretion of prolactin by the anterior pituitary, enhanced production of endothelial microRNA-146a (miRNA-146a) and placental secretion of soluble fms-like tyrosine kinase receptor 1 (sFlt-1) on a background of genetic susceptibility ultimately leads to endothelial dysfunction and cardiomyocyte8 apoptosis. Abbreviations: VEGF = vascular endothelial growth factor.

CLINICAL PRESENTATION OF PPCM

1. Onset

Timing of symptoms:

78%: within 4 months postpartum

9%: in the last month of pregnancy

13%: earlier in pregnancy (rare)

2. Symptoms (mimic systolic heart failure)

Cardiorespiratory:

Dyspnoea (shortness of breath)

Orthopnoea (breathlessness when lying flat)

Paroxysmal nocturnal dyspnoea (PND)

Unexplained cough (esp. when lying down)

Palpitations, dizziness

Systemic / congestion signs:

Leg swelling (peripheral oedema)

Abdominal discomfort (hepatic congestion)

Precordial chest pain

CLINICAL PRESENTATION OF PPCM

3. History:

Known congenital or acquired heart disease/family history of ischemic or non-ischemic HF.

4.Clinical Examination

Typical findings:

Sinus tachycardia

Raised JVP

Pulmonary crepitations

Additional signs:

Third heart sound (S3)

Displaced apex beat

Red flags (MBRRACE 2019):

Tachypnoea

Chest pain

Persistent tachycardia

Orthopnoea

⚠ These must always be investigated for cardiac disease (not dismissed as “normal pregnancy changes”).

Complications

1-VENTRICULAR ARRYTHMIAS

occur in 20% There have been cases of arrhythmia leading to sudden cardiac death during both the initial and later stages of the disease.

2-INTRACARDIAC THROMBUS

particularly when the left ventricular ejection fraction (LVEF) is less than 35%. The resultant thrombus can dislodge into the bloodstream and embolise to other organs resulting in MI, PE

3-ACUTE CARDIOGENIC SHOCK

requiring inotropic or mechanical circulatory support.

Differential Diagnosis :

Other cardiac conditions in pregnancy:
Benign dyspnea in pregnancy
Arrythmias
Asthma
Pre-existing cardiomyopathy
Hypertensive heart disease
Valvular disease (mitral stenosis, aortic stenosis)
Myocardial infarction (3–4x higher postpartum vs non-pregnant women)
Pulmonary embolism (5–10x higher risk in pregnancy/postpartum)
Amniotic fluid embolism (rare; shock + respiratory failure during/after delivery)

INVESTIGATIONS

ELECTROCARDIOGRAM

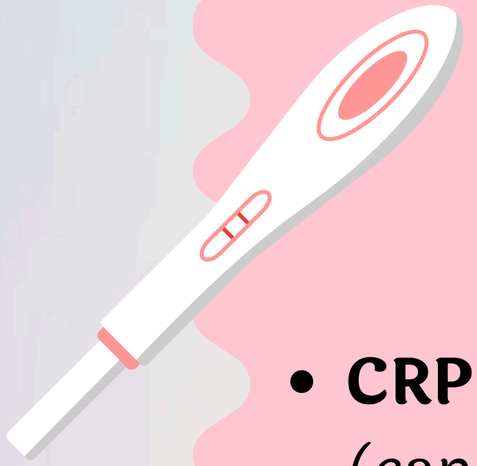
No ECG finding is specific to PPCM.

Common findings:

- Sinus tachycardia
- Nonspecific ST-segment and T-wave abnormalities

These can help discriminate from other conditions with similar presentations, but do not confirm PPCM.

INVESTIGATIONS



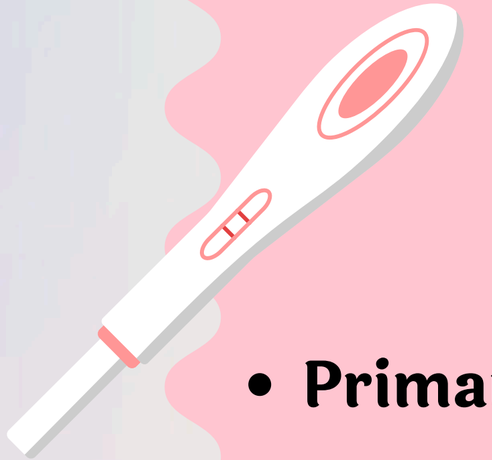
LABORATORY TESTS

- **CRP and leucocyte count** may be **elevated** but **non-specific** (can reflect inflammation/heart failure).
- **BNP** (brain natriuretic peptide)/ **NT-proBNP** (N-terminal pro-brain natriuretic peptide) **may be elevated**
- **Troponin T** (cardiac enzymes) **is often normal** in PPCM

Chest X-ray

Non-specific for PPCM.

May show features of cardiac strain:
Cardiomegaly, Pulmonary oedema, Pleural effusion



INVESTIGATIONS

ECHOCARDIOGRAPHY

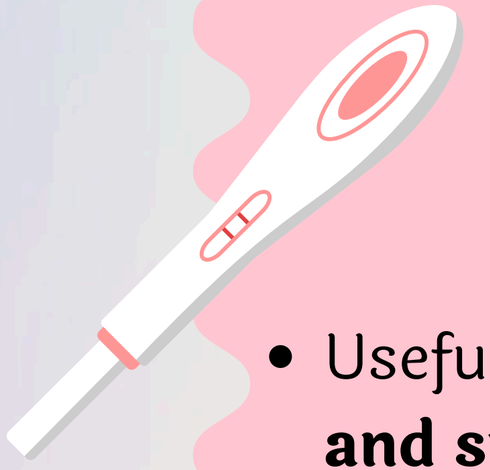
- **Primary investigation for diagnosis and risk stratification.**
- Advantages: no radiation; widely available.
- Diagnostic criterion: **LVEF < 45% is essential for diagnosis.**
- Prognostic value: Predicts likely recovery/outcomes

Right ventricular dysfunction (rare) → worse prognosis

Recovery predictors: LV size and LVEF at diagnosis

LVEDD > 6 cm or LVEF < 30% → poor outcome

(need for mechanical support/transplant/death)



INVESTIGATIONS

CARDIAC MRI

- Useful for detailed assessment: **Accurate chamber volumes and systolic function**
- **More sensitive than an echo** for detecting intracardiac thrombus
- Detects tissue injury patterns (intra/interstitial damage, hyperaemia, capillary leakage, necrosis, fibrosis)
- **Not first-line in pregnancy due to gadolinium concerns**
- **Breastfeeding is generally safe after gadolinium**

Usually not routine; mainly used when the diagnosis is uncertain or to rule out other causes of LV dysfunction.

INVESTIGATIONS

ENDOMYOCARDIAL BIOPSY

- Lower chance of spontaneous recovery
- Higher risk of poor outcomes

Management of PPCM in Pregnancy & Labour

- Most cases present postpartum, but antenatal PPCM tends to appear late in pregnancy.
- Manage in a high-risk obstetric unit with multidisciplinary care (at minimum obstetrics + cardiology; consider ICU/anesthesia early).

- Core decision: timing of delivery. Balance maternal haemodynamic status vs fetal prematurity.
- Delivery is not “automatic”—it’s based on maternal and fetal indications.

1. Timing & mode of delivery

A. Urgent delivery (regardless of gestational age)

Consider urgent delivery if there is:

- Advanced heart failure
- Haemodynamic instability
- Significant deterioration that cannot be stabilized safely

C. Induction of labour

- Consider induction for obstetric reasons and/or worsening cardiac function

B. Stable maternal status

- **Spontaneous vaginal delivery is acceptable** if there is no overt cardiac failure.
- **Caesarean section:**
  - Not routine for PPCM
  - Only for obstetric indications or severe maternal compromise.

D. Preterm delivery (24–35+6 weeks)

- Give antenatal corticosteroids for fetal lung maturity

2. Intrapartum care

A. Fetal monitoring: Continuous CTG.

B. Maternal monitoring

- Hourly:
  - Fluid balance
  - Vitals: BP, HR, RR, SpO_2
- Continuous:
  - ECG
  - Pulse oximetry
- Use **non-invasive cardiac output monitoring** if available.

If unstable / deteriorating

- Escalate early to the **ICU**
- Convert to **invasive monitoring**:
  - **Arterial line**
  - **Central venous pressure**
  - **Advanced CO monitoring**

3.Analgesia / Anaesthesia

Low-dose regional analgesia (epidural/spinal) is generally preferred:

- Helps reduce pain-related sympathetic surges
- Can **reduce the risk of cardiac instability**

Ensure:

- Titration carefully
- Close haemodynamic monitoring

4. Third stage of labour (post-delivery haemostasis)

- Use **slow oxytocin infusion** to reduce sudden haemodynamic changes.

Second-line uterotonics

- Misoprostol
- Carboprost ($PGF_2\alpha$)

Avoid (important in PPCM)

- Long-acting oxytocin analogues (risk of haemodynamic effects)
- Ergometrine

(increases risk of hypertension and can exacerbate heart failure)

PHARMACOLOGICAL TREATMENT

(A) Pregnant & haemodynamically stable

1) Non–heart-failure specific

- **Salt restriction**
- **Diuretics (loop)**: furosemide / bumetanide

2) Afterload / neurohormonal blockade

- **Beta-blocker** (prefer selective β1): metoprolol
- **Hydralazine + nitrates (afterload reduction)**

PHARMACOLOGICAL TREATMENT

(A) Pregnant & haemodynamically stable

3) Anticoagulation

- **LMWH**
- Warfarin & DOACs are contraindicated in pregnancy

Contraindicated in pregnancy:

- **ACE inhibitors (ACEi)**
- **ARBs**
- **MRAs** (spironolactone/eplerenone)

PHARMACOLOGICAL TREATMENT

(B) Postpartum while breastfeeding & haemodynamically stable

ACEi/ARB/MRA

- ACEi, ARB, and MRA are compatible with breastfeeding (if clinically needed)

HF regimen compatibility

- Beta-blockers, hydralazine, nitrates: safe during breastfeeding

PHARMACOLOGICAL TREATMENT

(B) Postpartum while breastfeeding & haemodynamically stable

Anticoagulation

- **LMWH or warfarin: both acceptable**
- **Hydralazine/nitrates can be withdrawn if ACEi/ARB is tolerated/started.**

PHARMACOLOGICAL TREATMENT

C) Postpartum and NOT breastfeeding

- Use HFrEF guideline-directed medical therapy, including:
  - **ARNI: sacubitril/valsartan**
  - **SGLT2 inhibitors**
  - Standard HFrEF options otherwise
- **DOACs if anticoagulation is indicated**

PHARMACOLOGICAL TREATMENT

(D) Haemodynamically unstable

- **Admit to ICU**
- Maternal stabilization first (this often determines the timing of delivery)

Core principles

- Optimize preload IV diuretics (treat congestion)
- Avoid overdiuresis

PHARMACOLOGICAL TREATMENT

(D) Haemodynamically unstable

Core principles:

- **Oxygenation/ventilation**: Keep SpO_2 > 95%, Upright position, CPAP if needed, Intubate if oxygenation remains refractory
- **Circulatory support**: Use **inotropes** and/or **vasopressors** based on shock phenotype

PHARMACOLOGICAL TREATMENT

(D) Haemodynamically unstable

- **Catecholamines** (e.g., dobutamine, adrenaline) can **increase myocardial O_2 demand**. Therefore, they're not ideal compared with alternatives when possible
- **Levosimendan is preferred** (weak evidence)

Delivery decision (if instability occurs)

- **Urgent delivery** if decompensation occurs during pregnancy {maternal safety first}

PHARMACOLOGICAL TREATMENT

(D) Haemodynamically unstable

Bromocriptine (important PPCM-specific therapy)

- Bromocriptine = prolactin blocker
- Recommended (ESC Class IIb, 2018)
    - **Must combine with anticoagulation**, because bromocriptine increases thrombosis risk (so anticoagulation is mandatory)

Mechanical Circulatory Support

Indication: when inotropes fail

- Short-term options
  - **Intra-aortic balloon pump** (IABP) ↓ afterload ↑ coronary/perfusion
  - **Intraventricular pump** (Impella) provides mechanical circulatory support by **unloading the LV / increasing forward flow**
  - **ECMO**: Consider when there is severe pulmonary dysfunction

Mechanical Circulatory Support

- Long-term options
  - LVAD / BiVAD Used as a bridge to:
    - recovery
    - cardiac transplant

Frequency:

- **~5%** of PPCM patients **require mechanical support.**

Prognosis:

- **Outcomes after transplant** in PPCM are **worse** than in other causes of heart failure.

Breastfeeding in PPCM

Prolactin is implicated in PPCM pathogenesis → theoretical concern that breastfeeding could **worsen/trigger disease.**

Who can **breastfeed**?

- Acceptable **if stable and LVEF ⩾ 45%**
- **Discourage** if:
  - **Symptomatic** (ongoing heart failure symptoms)
  - **Haemodynamically unstable**
  - **LVEF < 45%**

Breastfeeding in PPCM

Medication considerations:

- Generally **considered safe: enalapril, captopril**
- **Contraindicated**/avoid: **spironolactone, carvedilol**

Key principle

- **Prioritize maternal recovery** (if the mother's cardiac status is not stable enough, breastfeeding should be discouraged regardless of theoretical concerns).

Subsequent pregnancy in PPCM

1) Risk of relapse and outcomes

- **Persistent LV dysfunction** → about 50% chance of clinical deterioration
- Mortality: up to ~20%
- **Complete recovery of LV function → better overall prognosis**, but still ~20% relapse risk

Poor prognostic markers (high-risk for recurrence):

- Initial LVEF < 25%
- **Failure of LVEF to normalize with treatment**

Subsequent pregnancy in PPCM

1) Risk of relapse and outcomes

Counselling point:

- Women with **poor recovery / persistent LV dysfunction** should be advised **not to get pregnant again.**
- ESC/AHA guidance: Subsequent pregnancy is contraindicated if LVEF has not normalized.

Subsequent pregnancy in PPCM

2) If pregnancy is considered despite risks

- **Pre-conception MDT counselling** (cardiology + obstetrics; discuss risk/alternatives)
- **Close antenatal monitoring**
- **Serial echocardiograms throughout pregnancy** (monitor LV function trend)

3) Anticoagulation option

- **LMWH** may be considered **during pregnancy if LV dysfunction persists.**

Contraception in women with prior/ongoing PPCM

- **Reliable contraception is essential to prevent unplanned pregnancy** (which carries high maternal risk in PPCM).
- **Barrier methods are not reliable** enough on their own.
- **Preferred options: LARC** (Long-Acting Reversible Contraception), especially:
  - **IUD**: copper or hormonal
  - **Progestin-based LARC**
- **Avoid estrogen-containing contraceptives.** Estrogen increases thromboembolic risk.
- **Sterilisation (permanent contraception) is possible,** but requires careful counselling because it is irreversible and involves procedure-related risks.

Prognosis in PPCM

- about 50–80% **recover LV function within 6 months.**
- Mortality: improved from ~30–50% (1970s) to ~10% in current practice.
- **Afro-Caribbean women** have **poorer recovery** on average.
- Comorbidity effect: the **presence of hypertension is associated with better recovery.**

Predictors of poor outcome:

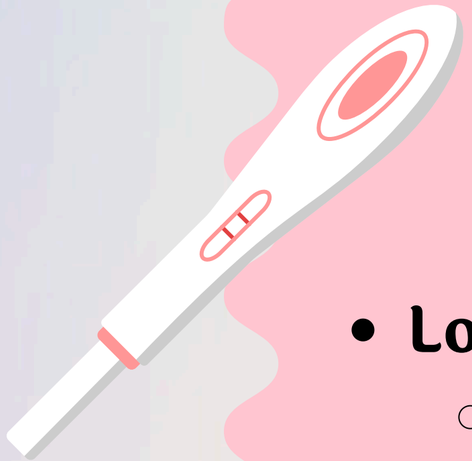
- **LVEF < 30%**
- **LVEDD > 6 cm**

Conclusion (PPCM)

- Nature of disease: **Rare but serious condition** affecting otherwise healthy **women of childbearing age.**
- Current understanding: Has improved, but many questions remain unanswered.

Treatment overview

- **Bromocriptine** is the **only therapy specific to PPCM.**
- Other treatments are **standard heart failure therapies** (adapted for pregnancy/postpartum and breastfeeding status).



Conclusion (PPCM)

Research gaps

- **Long-term prognosis** in women with:
  - complete recovery, and
  - incomplete recovery
- Optimal duration of medical therapy
- Identification of PPCM-specific diagnostic biomarkers
- Development of PPCM-specific treatments

Future directions

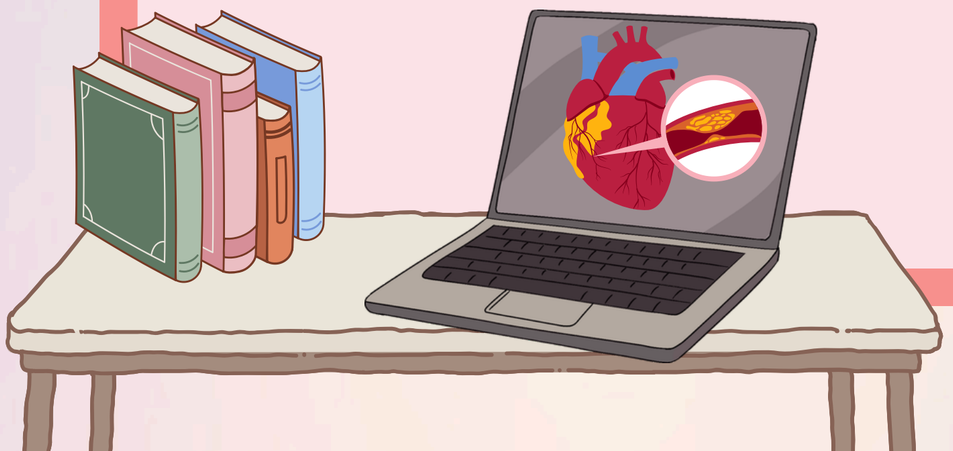
- Establish national, multicentre, and international registries
- Continued research to improve understanding and optimize management

Table 2. Safety profile of drugs for heart failure.[18]

Drug	Safety during pregnancy	Safety during lactation	Absence of full recovery of LV function	Complete recovery of LV function
Beta blocker	Safe, metoprolol is the recommended beta blocker.	Safe	Essential for all patients. Titrate to maximally tolerable dose.	Continue for at least 12 months.
ACE inhibitor	Avoid. Teratogenic due to risk of fetal kidney injury.	Low transfer of enalapril and captopril, hence relatively safe.	Essential for all patients. Titrate to maximally tolerable dose.	Continue for at least 12 months.
Angiotensin receptor blocker	Avoid, teratogenic.	Limited data, so best to avoid.	Recommended for women who cannot tolerate ACE inhibitor. Titrate to maximally tolerable dose.	Continue for at least 12 months.
Mineralocorticoid receptor antagonist	No data, so best avoided.	Limited data, so best avoided.	Recommended for all patients with LVEF <40%. Eplerenone may be considered because it is associated with fewer hormonal side effects.	Continue for at least 6 months. After this, discontinue if there is sustained recovery of the structure and function of left ventricular structure and function.
Diuretics	Use sparingly as they can cause decreased placental blood flow. Thiazides and furosemide are most commonly used.	Thiazides are the best-studied drug during lactation and are well tolerated.	Continue only when symptom control is needed (for oedema and congestion). Early tapering of doses when there is good symptom control even before complete recovery of LV function.	Discontinue as soon as symptom control for oedema and congestion is achieved.
Vasodilators	Includes nitrates and hydralazine. Use with caution, may precipitate uterine hypoperfusion.	Safe	Continue only when symptom relief is needed.	Discontinue when asymptomatic.

Abbreviations: ACE inhibitor = angiotensin-converting enzyme inhibitor; LV = left ventricle; LVEF = left ventricular ejection fraction.

Management of palpitations and cardiac arrhythmias in pregnancy



Introduction and Epidemiology

Palpitations are common in pregnancy due to a hyperdynamic circulation (↑ cardiac output, ↑ heart rate, ↑ stroke volume → increased cardiac awareness)

Most are physiological (sinus tachycardia, ectopics from ↑ myocardial excitability), but some reflect pathological arrhythmias, which are the most frequent cardiac complication in pregnancy.

Epidemiology

Palpitations are usually benign:

around 50% of pregnant women who are investigated for palpitations are found to have ectopic beats or non-sustained arrhythmias.

High-risk groups:

Congenital/structural heart disease

Previous arrhythmia (recurrence ~43%)

PHYSIOLOGICAL CHANGES

Cardiac output ↑ ~50% (↑ stroke volume from plasma expansion + ↓ SVR + ↑ HR)
Heart rate ↑ 10–20 bpm mainly in third trimester
Intrapartum: further ↑ CO
Postpartum: less well defined reaching up to 110 beats per minute within the first 48 hours.

ECG CHANGES

Sinus tachycardia
Left axis deviation(cardiac displacement by gravid uterus)
T-wave inversion (III, V1–V3)
Q waves (II, III, aVF)
Frequent atrial and ventricular ectopics

Arrhythmogenesis

Hemodynamic: chamber stretch activates stretch-sensitive ion channels → premature depolarization

Hormonal: estrogen ↑ adrenergic receptor sensitivity

Autonomic: ↑ sympathetic tone lowers depolarization threshold

PALPITATIONS DEFINITION AND MECHANISM

Palpitations are defined as an unpleasant awareness of the heartbeat, which may be perceived as fast, irregular, or forceful due to increased cardiac output and contractility.

Under normal conditions, the heartbeat is not perceived; however, increased stroke volume and heart rate in pregnancy amplify the mechanical force of contraction, and women may experience the sensation of "punding heartbeat".

Ectopic beats are perceived as a “skipped beat”

Table 1. Differential diagnosis of palpitations in pregnancy

Aetiology	Diagnosis
Physiological/ benign	Relative sinus tachycardia of pregnancy Exercise or stress-induced sinus tachycardia Occasional ectopic beats
Arrhythmias	Supraventricular tachycardia/extrasystoles Atrial fibrillation/flutter Ventricular tachycardia/extrasystoles Bradyarrhythmias: sinus bradycardia, atrioventricular heart block
Systemic causes	Hyperthyroidism Anaemia Sepsis Hypovolaemia Pulmonary embolus Hypoglycaemia Phaeochromocytoma Postural orthostatic tachycardia syndrome (POTS)
Psychosomatic	Anxiety Panic disorder
Drugs	Caffeine Nicotine Alcohol Cocaine, heroin, amphetamines Sympathomimetic drugs, e.g. salbutamol inhalers Vasodilators, anticholinergics Recent withdrawal of beta blockers

A detailed history is essential to differentiate physiological from pathological palpitations by identifying the nature, frequency, and triggers of symptoms.

History	Comments
Frequency and duration Onset and offset	Sudden onset and offset may raise the suspicion of arrhythmia
Circumstances	At rest or on exertion
Provoking and relieving factors	Vagal manoeuvres, rest
Associated symptoms?	Pre-syncope, syncope
What is their chronology?	Shortness of breath; ask about orthopnoea and paroxysmal nocturnal dyspnoea Chest pain Dizziness
Symptoms suggestive of systemic disorders	Sepsis Thyrotoxicosis
Did the symptoms pre-date pregnancy?	Pre-existing symptoms may raise suspicion of underlying arrhythmia
Prescription or illicit drug use	Including caffeine intake
Personal history of heart disease	–
Family history of heart disease or sudden cardiac death	Raises suspicion of underlying heart disease, inherited cardiomyopathy or channelopathies

Examination

Vital signs including:
pulse
blood pressure
respiratory rate
oxygen saturation
temperature
help assess hemodynamic stability and identify systemic causes.

An irregular pulse may indicate atrial fibrillation due to chaotic atrial depolarization, while a very rapid regular pulse suggests SVT.

Cardiac auscultation may reveal murmurs indicating structural abnormalities

Hypotension indicates reduced cardiac output and possible hemodynamic compromise due to ineffective ventricular filling or contraction.

Respiratory examination may detect pulmonary edema caused by left ventricular dysfunction or fluid overload.

Investigations

Who is at risk of arrhythmias?

Table 3. Features raising suspicion of an arrhythmia

Reassuring features	Features requiring further attention
Awareness of a fast, regular heartbeat, particularly when lying down	Fast and irregular heart beat
Occasional 'thumping sensation' suggestive of occasional ectopic beats	Palpitations waking from sleep or at work
Pre-vasovagal symptoms preceding the palpitations	Dizziness following the onset of palpitations
	Shortness of breath, chest pain, syncope
	Associated headache, sweating or abdominal pain and/or hypertension (consider phaeochromocytoma)
	Personal history of pre-existing cardiac disease
	Family history of cardiac disease, e.g. long QT syndrome, cardiomyopathy, sudden death

Full blood count to exclude anaemia
Thyroid function tests to exclude thyrotoxicosis
12-lead ECG (WPW 'delta wave'/LQTS)

Investigations

Ambulatory ECG Monitoring –When to Use

•If history or examination shows concerning symptoms or is inconsistent with physiological palpitations

• Helps capture true episodes of palpitations, especially if they do not occur during a standard ECG

****Limitation: Symptoms can be intermittent and unpredictable, so test sensitivity varies**

Box 1. Interpreting the results of a Holter monitor

- If symptoms correspond with a sinus tachycardia and all other investigations are normal (in the absence of suspicion of systemic pathology, e.g. pulmonary embolus or sepsis), reassure that this is physiological and do not perform any further investigations.
- Infrequent atrial or ventricular ectopic beats, couplets and triplets without other suspicion of an underlying structural heart disease on clinical examination or an inherited arrhythmia do not require further investigation.[17]
- If there is significant arrhythmia and the patient is symptomatic, seek urgent advice from Cardiology.

Holter Monitor (Most Used in Pregnant Women)

• Continuous external device connected by chest electrodes

• Recording duration: 24–72 hours

• Patientdiary: Record symptomsandactivities→helpsinterpretation

• Diagnostic yield: Detects clinically significant arrhythmias in 3–24% of women during 24 hours

Other Ambulatory Monitoring Devices

Event Recorder:

• Portable handheld device placed on the chest during palpitations

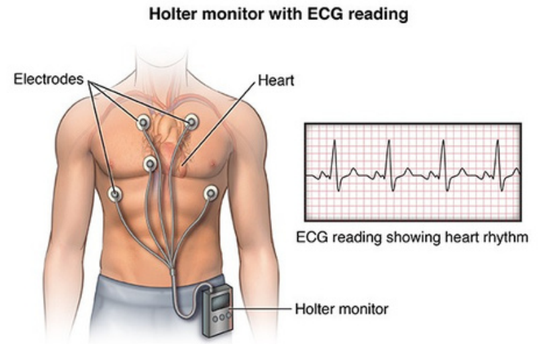
• Records one-lead ECG

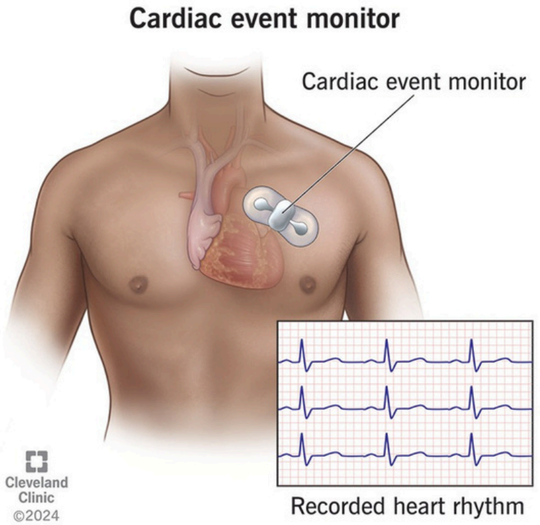
• Useful for infrequent symptoms

More invasive devices:

• Implantable loop recorder: Internal device for long-term arrhythmia monitoring

• Pacemaker or Internal Cardiac Defibrillator: Automatically detects serious arrhythmic events





Investigations

Echocardiography

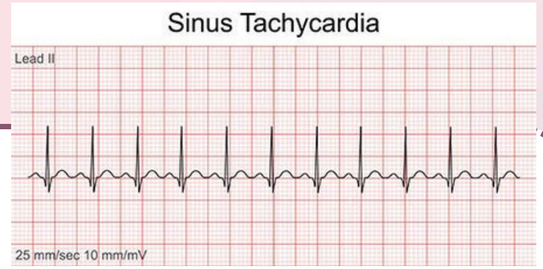
evaluates structural heart disease such as valvular abnormalities or cardiomyopathy, which alter cardiac geometry and predispose to arrhythmias.

Table 4. Who needs an echocardiogram?

Features suggesting need for echocardiogram	Echocardiogram may not be indicated
Diagnosed arrhythmia	Symptoms consistent with physiological changes in pregnancy
Audible heart murmur	Isolated sinus tachycardia at time of symptoms on ambulatory ECG
Concerning features on history, e.g. shortness of breath	Infrequent ectopic beats in the absence of other signs of structural heart disease/ inherited arrhythmia
Known structural heart disease	
Previous chemotherapy with cardiotoxic agents	
Family history of inherited arrhythmia, e.g. long QT syndrome, sudden cardiac death	

ECG = electrocardiogram.

Tachyarrhythmias



Sinus tachycardia

increase in heart rate due to activation of the sinus node.

- Usually occurs as a physiological response to pregnancy because of increased blood volume andthe metabolic demands of the fetus.
- Small studies (up to 55 women) show average heart rate in the third trimester: 80–87 bpm.
- This type of tachycardia is usually normal and asymptomatic.
- Sometimes it is difficult to distinguish physiological vs pathological tachycardia.
- If tachycardia is unexplained or persistent, consider:
- Inappropriate sinus tachycardia: excessive heart rate not proportional to activity or rest
- POTS(Postural OrthostaticTachycardiaSyndrome): increased heart rate on standing, withsymptoms such as dizziness, syncope, or palpitations

Patient experience:

- The patient may feel her heart beating fast, sometimes especially at rest or when lying down.
- Usually not associated with chest pain or syncope if physiological.

Atrial and ventricular premature beats

Atrial and ventricular premature beats or ectopic beats arevery common in pregnancy.
One Holter monitor study detected atrial or ventricular beats in more than 50% of pregnant women.
Women should be reassured that ectopic beats are benign and treatment is not usually required.

Tachyarrhythmias

Supraventricular Tachycardia (SVT)

• SVT is the most common non-benign arrhythmia in pregnancy, occurring in about 24 per 100,000 women
It may present for the first time during pregnancy in 3.8%–34% of affected women.
Common Types and Causes
1.AVNRT (Atrioventricular nodal reentrant tachycardia) – most common
where a re-entry electrical circuit forms within the AV node and surrounding atrial tissue
2.AVRT or Accessory pathway mediated tachycardia
• An extra conduction pathway allows impulses to bypass the AV node, leading to tachycardia.
• Example: Wolff–Parkinson–White (WPW) syndrome,
characterized on ECG by a short PR interval and delta wave.
Clinical Presentation
• Sudden onset and sudden termination of palpitations.
• May be associated with:
• Dizziness or syncope
• Chest pain
• Hemodynamic compromise, especially with underlying structural heart disease.

Management
1) First-line: • Vagal manoeuvres(e.g., Valsalva manoeuvre)
If unsuccessful:
2)• Intravenous adenosine
• Terminates ~90% of SVT episodes in pregnancy.
• Safe when used in a monitored clinical setting.

Management
Alternative treatments:
• Verapamil or Metoprolol.
•Direct current cardioversion if there is hemodynamic instability.
Prophylaxis:
•Beta-blockers in women with known recurrent PSVT
Catheter ablation of an accessory pathway is not recommended in pregnancy

Tachyarrhythmias

Atrial fibrillation and Atrial flutter

Uncommon in pregnancy

Usually associated with: Mitral stenosis, metabolic abnormalities, electrolyte imbalance

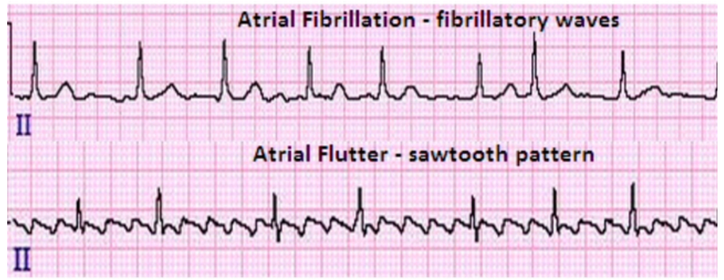
New onset during pregnancy --> Investigate underlying cause

Atrial fibrillation (AF)

Irregular, independent atrial muscle contraction

Absence of P waves on ECG

Irregularly irregular ventricular rhythm



Atrial flutter

Atrial rate≈300bpm

Commonly 2:1 conduction-->ventricular rate ≈150bpm

Characteristic "saw tooth" ECG pattern

Clinical significance in pregnancy:

Hemodynamic compromise (especially in Mitral stenosis)

Increased risk of systemic embolism in an already pro-thrombotic state of pregnancy

Persistent AF requires anticoagulation preferred agent in pregnancy: **LMWH**

Acute management

If hemodynamically unstable

First-line : Direct current cardioversion

If stable:

Pharmacological cardioversion (IV flecainide or ibutilidein in structurally normal heart)

Rate control: Beta-blockers first line

Tachyarrhythmias

Ventricular Tachycardia (VT)

Arrhythmia originating from the ventricular muscle

• High-frequency depolarization → wide and abnormal QRS on ECG

Causes: Structural heart disease/Primary electrical disease

• Idiopathic VT in healthy women often originates from Right Ventricular Outflow Tract (RVOT)

Usually presents as short, non- sustained VT

ECG shows Left Bundle Branch Block (LBBB) pattern

Treatment:

Beta blockers or Verapamil

–Clinical Significance in Women with Heart Disease

• High risk of sudden cardiac death (SCD) requires immediate treatment

Box 2. Cardiac disease associated with ventricular tachycardia[31]

Cardiac disease

- Hypertrophic cardiomyopathy
- Peripartum cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy
- Congenital heart disease, including previous cardiac surgery
- Valvular disease
- Myocardial infarction

Inherited channelopathies

- Long QT syndrome

Acute management

If hemodynamically unstable

First-line : Direct current cardioversion

If stable:

Pharmacological cardioversion (flecainide or sotalol) drug choice depends on underlying cardiac pathology

Prophylaxis in High-Risk Patients

• Beta blockers

• Amiodarone

• Implantable Cardioverter Defibrillator (ICD)

ICD indications:

• Primary prevention in high-risk patients

• Secondary prevention after a life-threatening VT episode

Tachyarrhythmias

Long QT syndrome

LQTS is characterised by a prolonged QT interval on the ECG, secondary to a disorder of ventricular myocardial repolarisation.

This can lead to ventricular arrhythmias, typically torsade de pointes and a risk of sudden death.

inherited LQTS, gene defects code for cardiac ion channels.

Numerous genes have been identified, with KCNQ1, KCNH2 and SCN5A mutations being most commonly associated.

Events appear to be less common during pregnancy, but there is a significant increase in the risk of cardiac events in the postpartum period, particularly in women with type 2 LQTS.

Management

Continue beta-blockers during pregnancy and postpartum

Avoid electrolyte imbalance ($\downarrow K^+$, $\downarrow Mg^{2+}$)

Avoid QT-prolonging drugs (e.g. ondansetron, erythromycin, trimethoprim, prochlorperazine)

Hyperemesis = high risk (vomiting → electrolyte disturbance + poor drug tolerance)

Associated with sudden infant death syndrome (SIDS)

Bradyarrhythmias

Bradyarrhythmias= slow heart rhythm (<60 bpm)

• Rare in pregnancy

• Usually well tolerated

-AV Block in structurally normal hearts

• First-degree AV block: usually benign

• Second-degree AV block (Wenckebach / Mobitz I): usually asymptomatic

•Complete(Third-degree)AV block:

• Can be congenital

• Can be acquired (e.g., after surgery for congenital heart disease)

•Some cases first diagnosed during pregnancy→often previously undetected Pregnancy Tolerance

•Asymptomatic women:pregnancy and delivery usually well tolerated

• Symptomatic women:

• Pacemaker recommended

• Can be:

• Temporary → to cover delivery

•Permanent→safe during pregnancy

Bradyarrhythmias

Bradyarrhythmias= slow heart rhythm (<60 bpm)

• Rare in pregnancy

• Usually well tolerated

-AV Block in structurally normal hearts

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• Symptomatic women:

• Pacemaker recommended

• Can be:

• Temporary → to cover delivery

•Permanent→safe during pregnancy

Anti arrhythmic drugs in pregnancy

Table 5. Antiarrhythmic drugs in pregnancy (BNF)[16,38]

Drug	Safety/complications	Breastfeeding
Adenosine	Benefits outweigh risk of fetal toxicity in large doses	Safe (short half-life)
Amiodarone	Suitable for short-term use in emergencies Prolonged use: fetal thyroid abnormalities, growth restriction and prematurity; risk may outweigh benefit	Avoid long-term use Risk neonatal hypothyroidism
Beta blockers	Commonly used, benefits generally outweigh risks Possible relationship with growth restriction in fetus but many confounding factors, e.g. hypoglycaemia, hyperbilirubinaemia	Safe
Digoxin	Safe unless toxic doses	Safe
Flecainide	Likely safe, insufficient data to suggest any fetal issue Also used to treat fetal supraventricular tachycardia	Present in breastmilk, not known to be harmful
Lidocaine	Safe unless toxic doses	Safe
Verapamil	Safe Rapid injection may cause maternal hypotension and associated fetal distress	Safe

Management of pregnancy in pre-existing arrhythmia

women with a pre-existing cardiac arrhythmia should be managed by a multidisciplinary team, which should include an obstetrician with an interest in maternal medicine, a cardiologist and an anaesthetist.

Box 3. General principles of the management of pre-existing arrhythmias

Preconception

- Condition-specific advice on risk in pregnancy
- Review of medication and changes advised if appropriate
- Optimisation of condition prior to conceiving; consider referral for accessory pathway ablation

Antenatal

- Review of medication
- Growth scans if on beta-blockers
- Anaesthetic review and planning
- Planning for birth

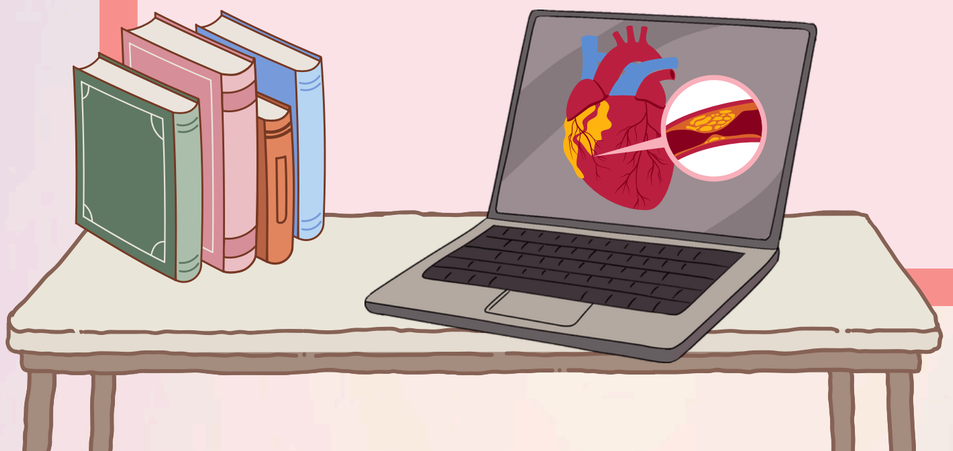
Intrapartum

- Vaginal birth usually recommended
- Consider place of birth if risk of arrhythmia high; co-located unit with appropriate facilities
- Consider continuous cardiac monitoring in those at high risk
- Care plan to include advice on management of acute arrhythmia and ensure drugs/facilities available in advance, and drugs to be avoided

Postnatal

- Period of inpatient monitoring
- Some conditions high risk of postnatal event, e.g. long QT syndrome
- Plans for medication and breastfeeding
- Ensure plans for continuing cardiological care/investigations arranged

Acute coronary syndromes in pregnancy



ACS in Pregnancy: A Critical Maternal Health Crisis

No progress in 20 years

Maternal mortality from cardiovascular disease has not improved in almost 20 years; this contrasts to significant sustained improvement in cardiovascular outcomes in the nonpregnant population, particularly regarding acute coronary syndrome (ACS).

Recognition gap

Many of the women who died in recent MBRRACE-UK reports presented with cardiac symptoms and signs, but this was not always recognised upon clinical review.

Urgent intervention

Early involvement of senior clinicians and a multidisciplinary approach is crucial for improving maternal outcomes.

Pregnancy & ACS Risk Factors

The incidence of ACS is increasing alongside advancing maternal age, with an accompanying increased prevalence of typical and pregnancy-specific cardiac risk factors.

Acute coronary syndromes (ACS)

Rare but important cause of maternal mortality.

Early recognition and an evidence-based multidisciplinary (MDT) approach are key in improving outcomes.

Epidemiology

- Rates of maternal ACS: 0.6–10 per 100,000 pregnancies worldwide (6.2 per 100,000 deliveries)
- Mortality rate: 5.1–11.0%.

Maternal mortality data
(MBRRACE-UK 2017–2019: Mothers and Babies report)

Cardiovascular disease:

Leading cause of maternal deaths.
Accounts for 17% of all maternal death

Ischemic heart disease (IHD):

- Responsible for around one-fifth of cardiovascular maternal deaths.
- Relative risk compared with nonpregnant women ،Pregnant women are 3–4 times more likely to suffer ACS than age-matched non-pregnant women.
- The incidence of ACS in pregnancy is rising.

Pregnancy-Related Pathophysiology Relevant to ACS:

1- Haemodynamic and cardiovascular changes across pregnancy

- Blood volume increase: increases by 50%.
- Cardiac output: increases by 50%.
- Heart rate: basal heart rate increases.
- Anaemia:
  - Women often become anaemic:
  - 1)Due to haemodilution.
  - 2) iron deficiency.
- Blood Pressure and Peripheral Vascular Resistance:
  Fluctuate during pregnancy.
- Around delivery:
  These haemodynamic parameters can shift rapidly.

Pregnancy-Related Pathophysiology Relevant to ACS:

2- Labour and delivery

- Oxygen consumption in labour increases.
- There is a potential for massive blood volume loss.

3- Thrombotic tendency

- Pregnancy is a prothrombotic state.
- The risk of thromboembolism rises throughout pregnancy and into the postpartum period.

4- Connective tissue / vascular changes

- The woman's connective tissue weakens and stretches in preparation for birth.
- This includes the vasculature.

5- Impact on coronary circulation

- The demands on the maternal cardiovascular system are such that:
- Even a small reduction in coronary flow can precipitate significant myocardial ischaemia.

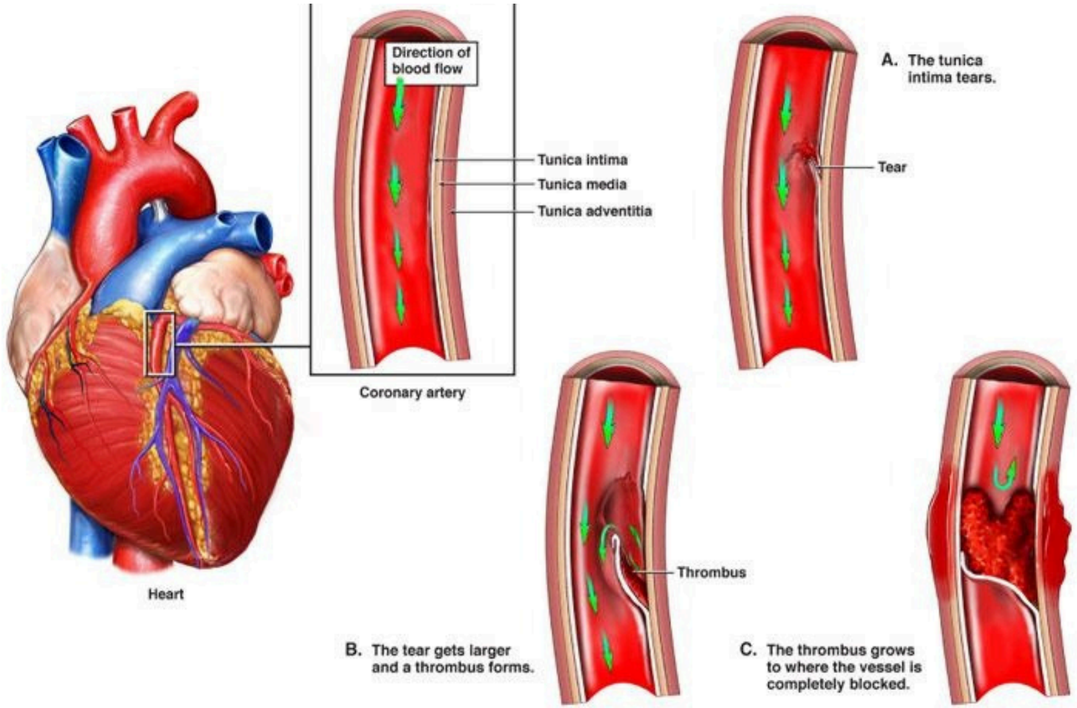
Etiologies of ACS in Pregnancy

Several different aetiologies can underlie ACS in pregnancy.
Prevalence rates differ between studies.

1 Pregnancy-Associated Spontaneous Coronary Artery Dissection (PASCAD)

Historically, PASCAD has been the predominant aetiological subgroup.
One study: PASCAD was the underlying cause in 27% of cases.
Older studies: Reported rates up to 43%.

PASCAD is a subgroup of the whole population SCAD(spontaneous coronary artery dissection).



Pregnancy-Associated Spontaneous Coronary Artery Dissection (PASCAD)

Pathology / mechanism:

There is a **sudden** disruption of the coronary arterial wall

Separation of the intimal lining from the outer vessel

Tearing of the intima:
Allows formation of a false lumen.

↓

This false lumen:

- **Propagates.**
- **Compresses the true lumen.**
- **Creates flow-limiting stenosis or obstruction.**

↓

Blood flow into the false lumen:
- **Blood clots and expands, forming an intramural haematoma.**

Timing:

PASCAD usually occurs:

1)In the late third trimester.
2)in the early postpartum period.

Pathogenesis:

- The pathogenesis is not fully understood.

- The typical timing suggests association with:
1) Increases in haemodynamic stress.
2)Fragility of the vasculature across this period.

The haematoma:
- **Exerts pressure on the true lumen.**
- **Collapses the true lumen.**
- **Compromises coronary flow.**

Etiologies of ACS in Pregnancy

2 Atherosclerosis

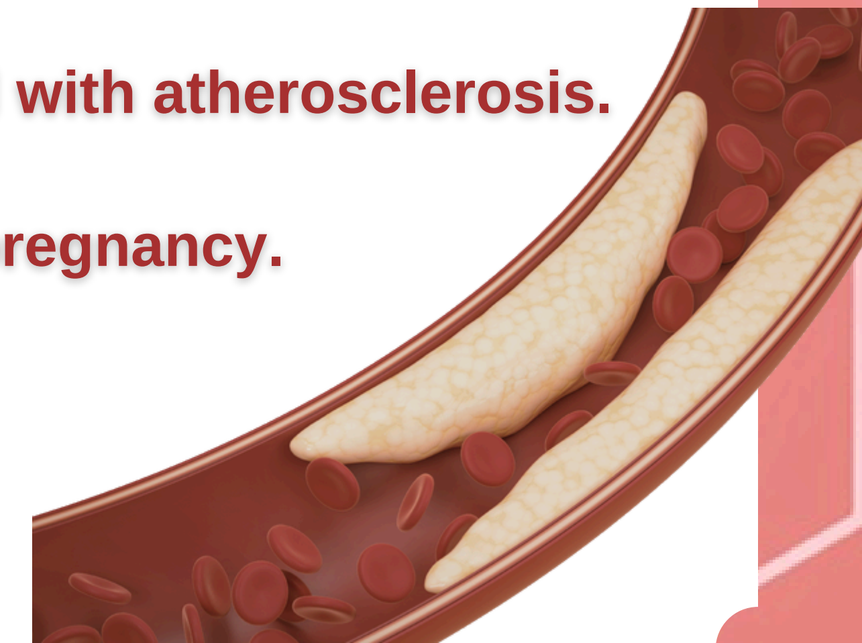
Current predominance:
- Atherosclerosis has now overtaken PASCAD as the most prevalent aetiology.

Probable reasons:
- Rising maternal age.
- Increased prevalence of relevant comorbid risk factors.

Epidemiological data

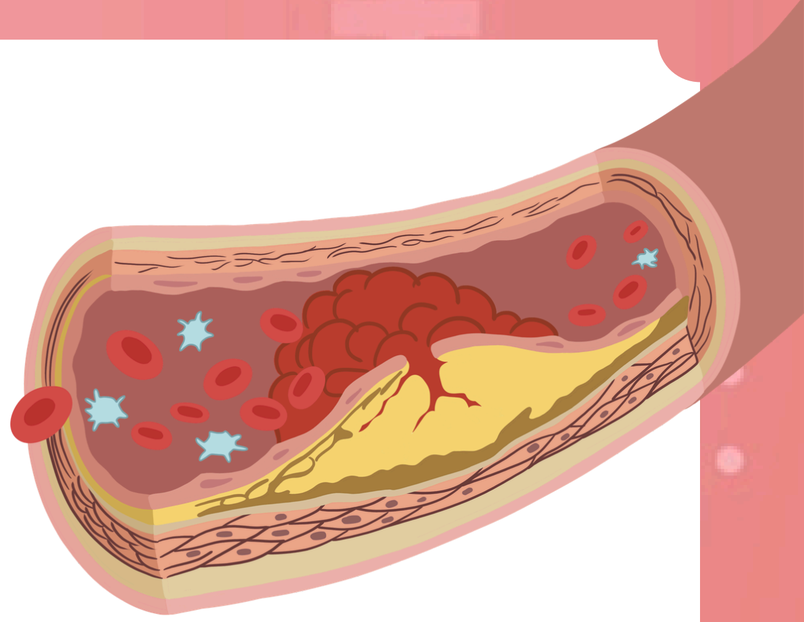
Baris et al.:
- Found that 39% of ACS in pregnancy were associated with atherosclerosis.
- Included:
□ Women with pre-existing IHD.
□ Women who presented with IHD for the first time in pregnancy.

Roth et al.:
- Found similar numbers (40%).



Plaque formation and progression:

Atherosclerotic plaques usually form at:
- Areas of haemodynamic shear stress.
- Regions where the endothelium is vulnerable to injury.



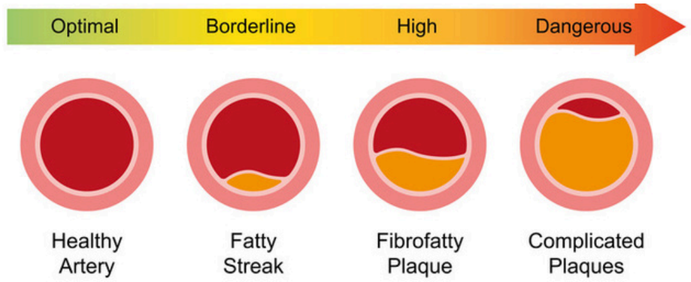
Following vascular trauma:

- **Localized accumulation of lipid within the intima.**
- **Recruitment and proliferation of:**
  - Immune cells.
  - Smooth muscle cells.
  - Fibrous connective tissue deposition occurs.

- **The soft lipid core beneath the fibrous cap:**
  - Enlarges.
  - Weakens the arterial wall.
  - Precipitates plaque rupture.

Upon plaque rupture:
- Blood is exposed to the lesion.
- Leads to rapid platelet recruitment and thrombosis.

Risk factors
- The condition exhibits the same traditional risk factors as in the non-pregnant population



Etiologies of ACS in Pregnancy

3 Thrombosis and Thromboembolism

- Occur within otherwise normal coronary vessels.
- Account for 10–20% of ACS cases in pregnancy.

4 Coronary Artery Spasm:

Frequency

-Presents rarely.
-Accounts for approximately 2% of ACS cases in pregnan

Pathophysiology

Described as a poorly understood phenomenon

Management

Can usually be managed conservatively with medication

Risk Factors for ACS in Pregnancy

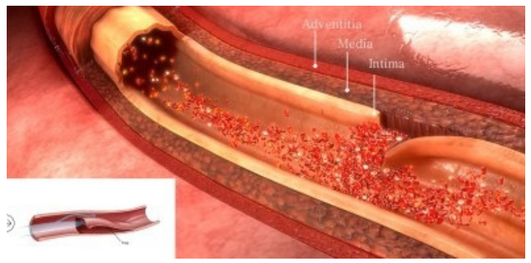
Risk factors can be categorized according to **underlying pathophysiology**:

Risk Factors for PASCAD

1) Connective tissue disorders
(e.g. Marfan syndrome).

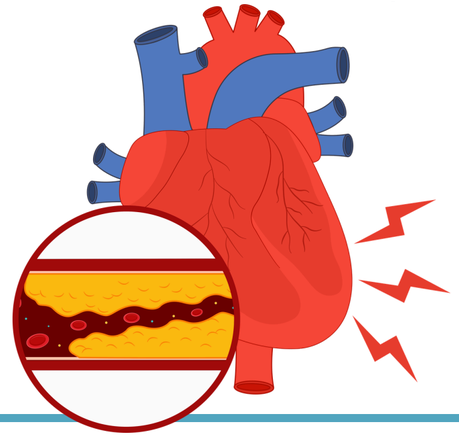
2) Hypertension.

3) Family history of spontaneous coronary artery dissection (SCAD).



Risk Factors for Atheromatous Disease

1)Increased maternal age.
2)Obesity.
3)Smoking.
4)Diabetes mellitus.
5)Hypertension.
6)Dyslipidemia.
8)Family or personal history of ischemic heart disease (IHD).



Factors Increasing Thrombotic Risk (Other than pregnancy itself)

1)Thrombophilia.
2)Hypertensive disorders.
3) Blood transfusion.
4)Infection.
6)Multiparity.
7)Sickle cell disease.
8)Blood transfusion:
-Represents increased thrombosis risk associated with anaemia.
- Is probably a surrogate marker for ergometrine use.
- Ergometrine may be more associated with coronary artery spasm than with either of the other ACS aetiologies (PASCAD or atheromatous disease).

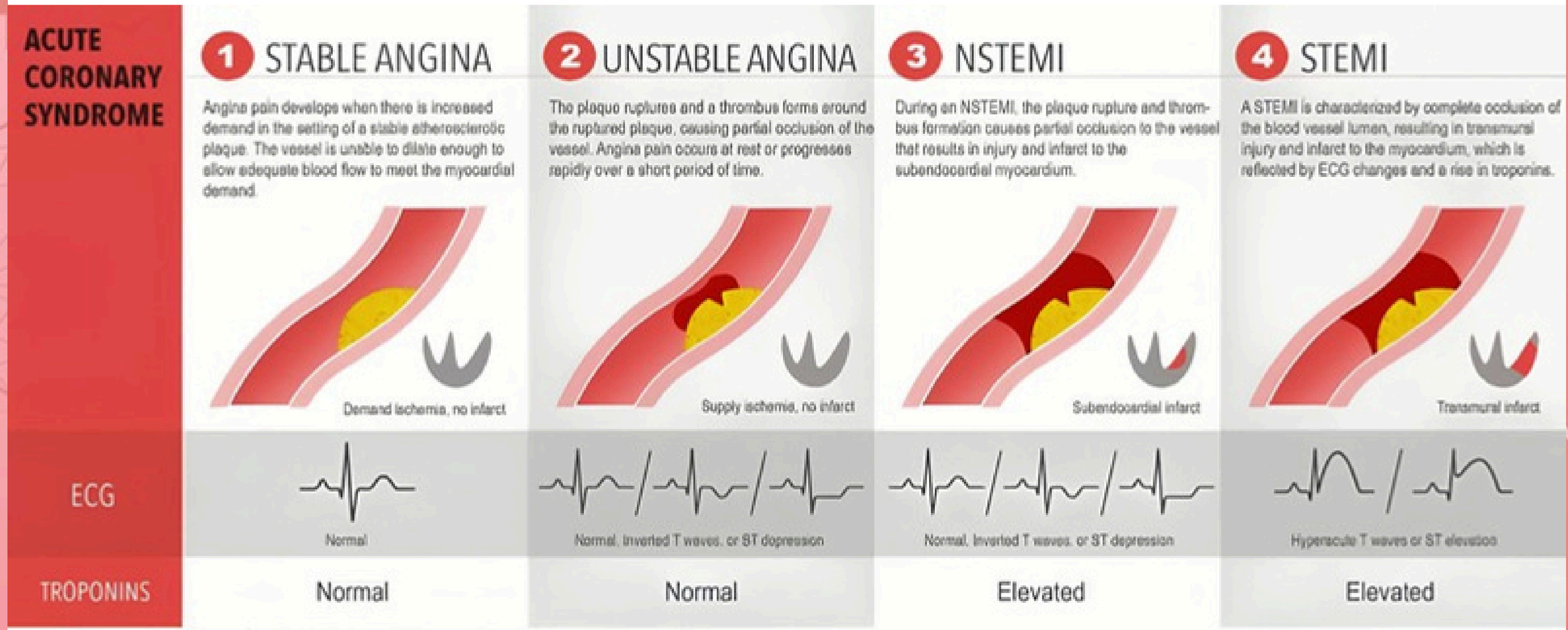
Timing of ACS in Relation to Pregnancy

ACS can occur <u>at any gestation</u>.

- Distribution in one study:
  - **73%** of cases occurred **during pregnancy**.
  - **27%** occurred in the **postpartum period**.
- **Trimester association:**

-Antenatal ACS is significantly <u>more common</u> in the **third trimester** than at earlier gestations.

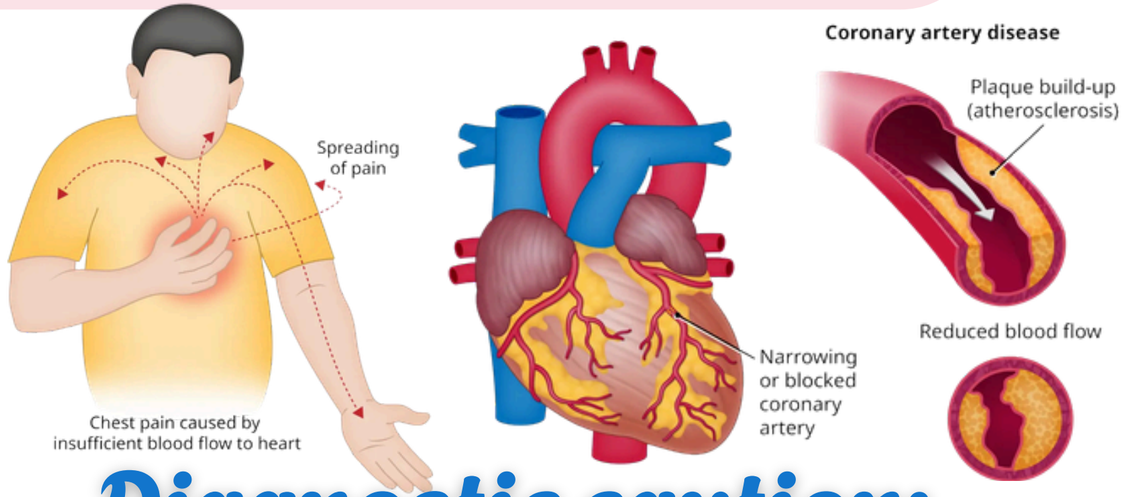
Spectrum of Ischemic Heart Diseases:



This infographic was created by Paula Sheath and Leah Zhao for the Sirens to Scrubs series of CanadiEM.org.

Spectrum of Ischemic Heart Diseases:

1 Stable Angina



Typical features:

1)Chest pain or shortness of breath (Relieved by rest)

2)No biomarker rise.

3)In most cases, the ECG is normal.

Diagnostic caution:

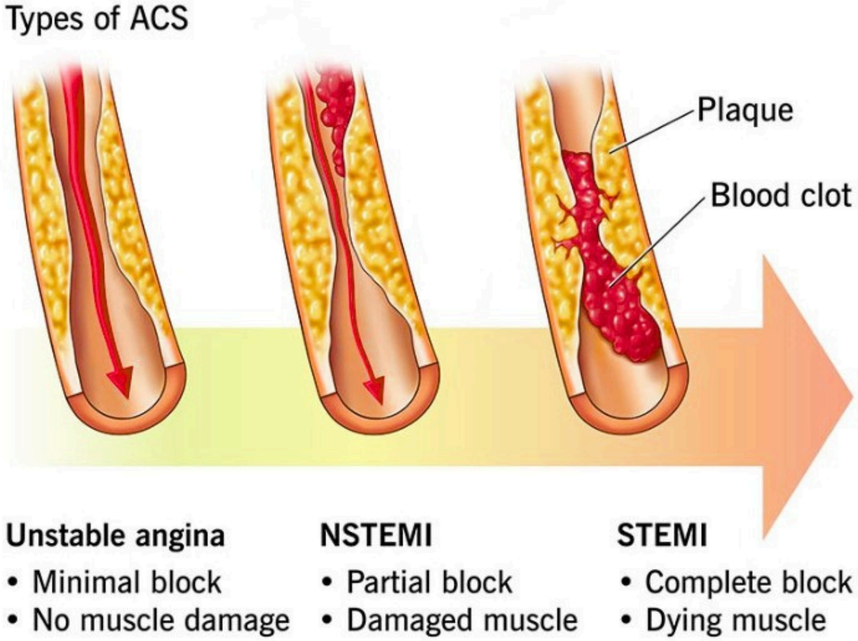
If a provisional diagnosis of ACS has been made but:
- There is a normal ECG, and
- A normal biomarker profile, in the presence of continuing chest pain:
The clinician should review the diagnosis.

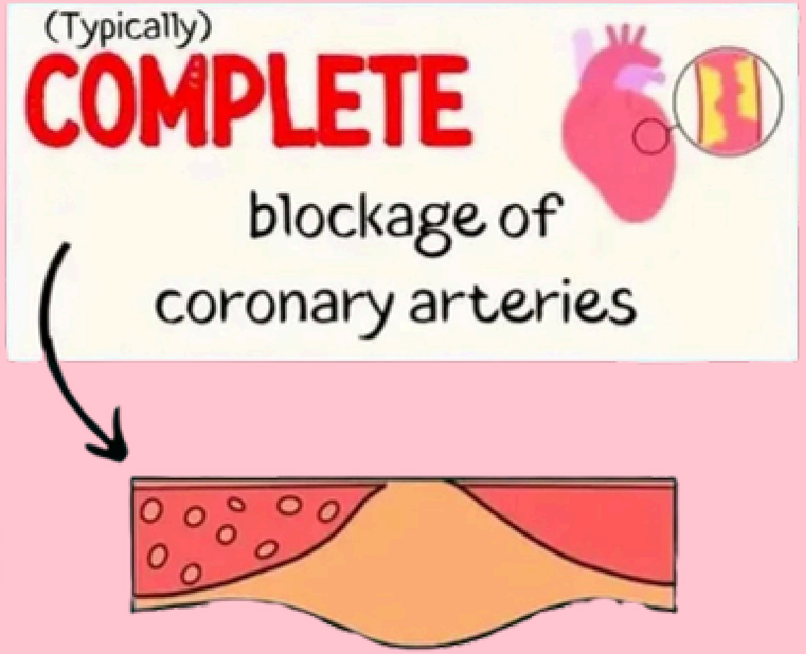
Spectrum of Ischemic Heart Diseases:

2 ACS & Related Conditions

ACS in pregnancy can be divided into three subgroups:

1. ST-elevation myocardial infarction (STEMI).
2. Non-ST-elevation myocardial infarction (NSTEMI).
3. Unstable angina (UA).





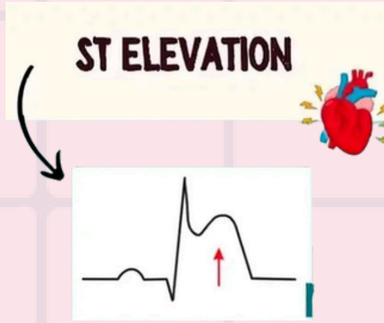
ST-Elevation Myocardial Infarction (STEMI)

STEMI accounts for roughly <u>75%</u> of maternal ACS emergency presentations.

01 Definition

Presence of:
ST elevation, or New left bundle branch block (LBBB)
On an electrocardiogram (ECG).

Within a clinical context consistent with ACS, typically:
-Chest pain.
- Shortness of breath (SOB).
-Ventricular arrhythmia.



02 Clinical severity

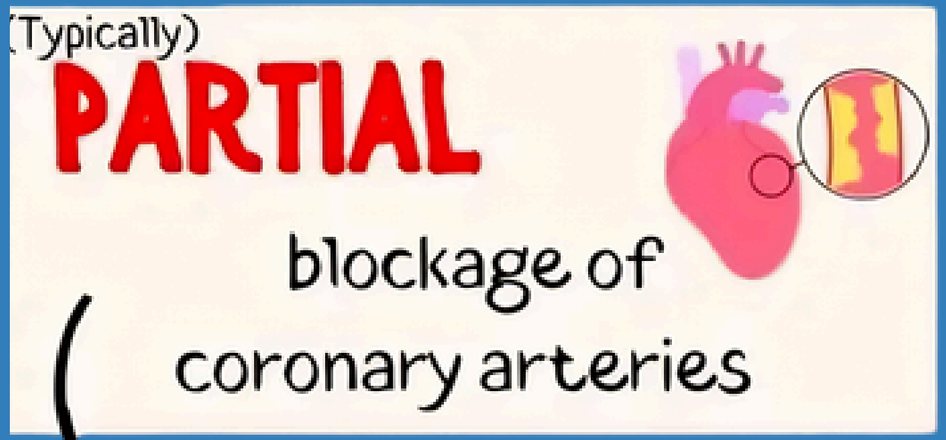
Haemodynamic instability is often present.

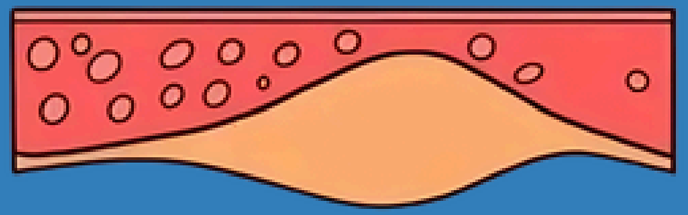
03 Importance of time to treatment

- Time to definitive treatment is the strongest predictor of outcome.
- National targets often focus on:
•A 1-hour window between ECG diagnosis and restoration of coronary flow.

04 Biomarkers

Cardiac biomarkers would inevitably be raised.
- However, ECG and clinical history are sufficient for diagnosis in this context.





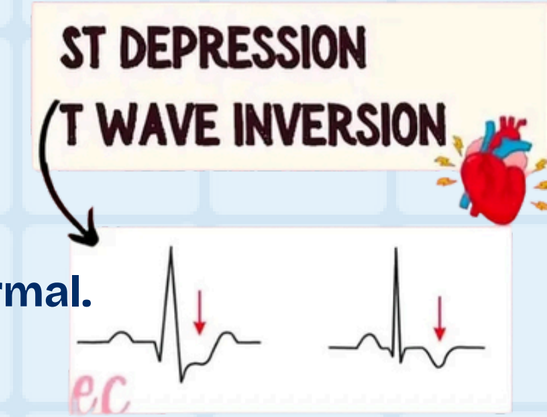
Non-ST-Elevation Myocardial Infarction (NSTEMI)

01 Clinical severity

-Typically presents with a more stable clinical situation than STEMI.
-However, haemodynamic instability is not uncommon.

02 ECG Features

ECG changes may include:
• ST depression.
• T-wave inversion.
- However, the ECG can also be normal.

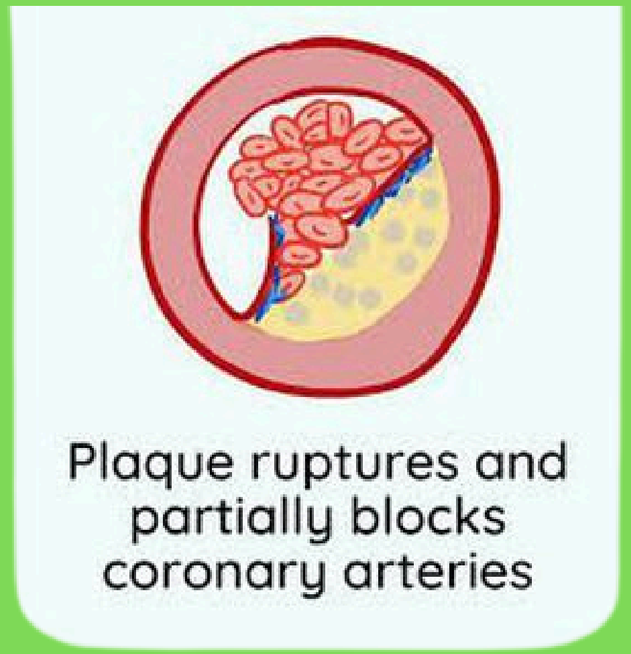


03 Management principle

Risk assessment on an individualised basis is crucial:
• To stratify patients who would benefit from:
- An emergency procedure versus
- A procedure delay or conservative management.

04 Biomarkers

Cardiac biomarkers are **raised**.



Unstable Angina (UA)

01 **Definition**

chest pain or shortness of breath (SOB) at rest.

02 **ECG Features**

The same changes as NSTEMI
(e.g. ST depression, Twave inversion).

03 **Biomarkers**

No rise in cardiac biomarkers.

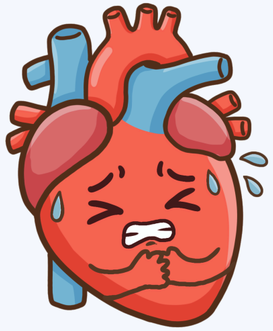
Clinical Presentation of ACS in Pregnancy

General Presentation

Pregnant women with ACS usually present similarly to age-matched women in the non-pregnant population. However, atypical symptoms are more common in both groups (pregnant and non-pregnant women).

Typical Symptoms

- Chest pain (often central).
- Epigastric pain.
- Pain may radiate to:
  - The neck.
  - The arm.



Atypical Symptoms:

(More commonly described by women than men)

- Nonspecific pain.
- Nausea.
- Vomiting.
- Back pain.
- Dyspnoea(shortness of breath).
- Hyperhidrosis (excessive sweating).
- Agitation.

EXAMINATION FINDINGS

Examination may be **unremarkable**.

Vital signs:

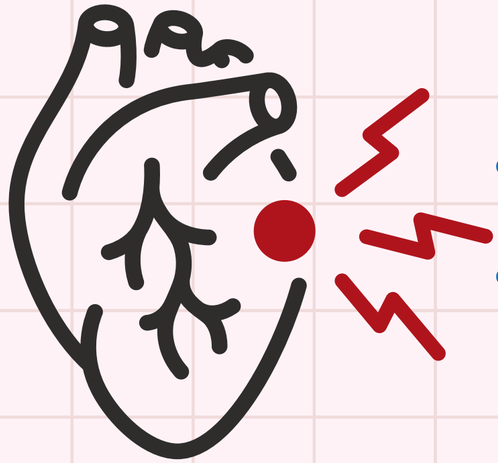
- Can indicate haemodynamic instability.
- Important to be aware that:
  - In pregnant women, symptoms are often downplayed or dismissed.
  - This can lead to **delayed** or **missed** diagnosis.

Relationship Between A etiology and Presentation

PASCAD, thrombosis,and coronary artery spasm:

Usually present as:

- STEMI
- NSTEMI.



Atheromatous disease:

May present as:

- Stable angina
- Unstable angina.

Sever presentations

Cardiac arrest

Rarely, women may present in cardiac arrest.

Management:

Should follow maternal cardiac arrest guidelines.

Cardiac causes should always be considered.

DDX of chest pain in pregnancy

- Pulmonary embolism (PE).
- Aortic dissection.
- Pneumonia.
- Gastro-oesophageal reflux disease (GORD).
- Musculoskeletal (MSK) pain
- Anxiety.

Investigations

Electrocardiogram (ECG)

The ECG is the most familiar of cardiac investigations.
- In a normal pregnancy untroubled by cardiac disease, ECG changes can occur.

- **Physiological ECG changes in normal pregnancy**
  - **Reduction in PR interval.**
  - **Sinus tachycardia.**
  - **Left axis deviation.**
  - **Q-waves and T-wave inversion in: Leads II, III, aVF.**
  - **T-wave abnormalities in: Leads V1–V3.**

- **Pathological ECG changes suggesting acute ACS:**

1)ST elevation:
• Typically within a coronary territory.
• With reciprocal ST depression.
2)ST depression alone.
3)T-wave inversion.
4)New bundle branch block (BBB).

Suggested mechanisms for these physiological ECG changes

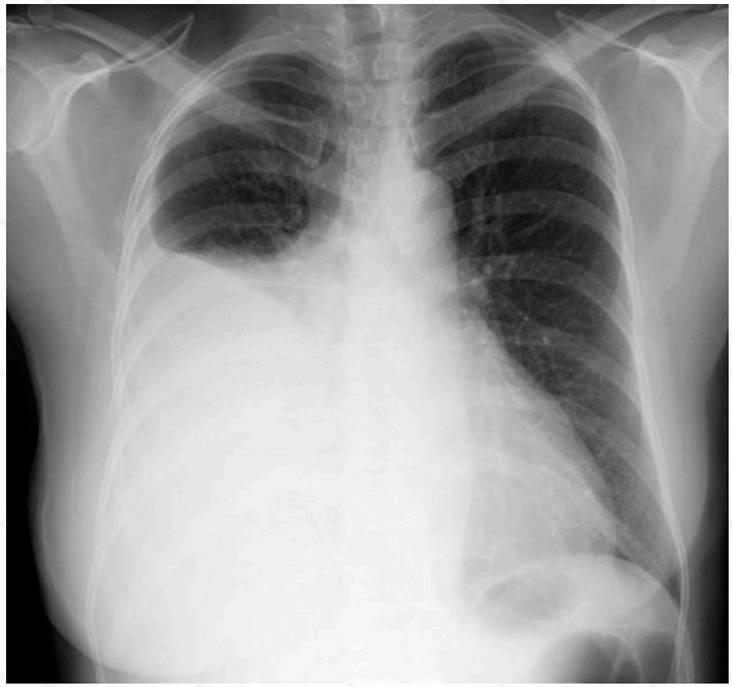
1)Suggested Maternal organ displacement.

2) Changing myocardial conductive properties due to:
• Higher levels of sympathetic hormonal modulation.

3) Left ventricular hypertrophy (LVH):
• To support higher cardiac output.mechanisms for these physiological ECG changes

Investigations

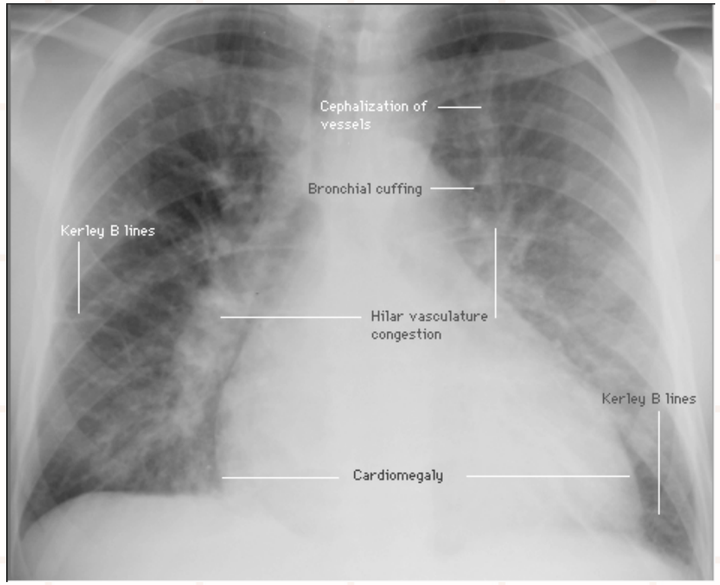
Chest X-Ray (CXR)



- Value in ACS
  - There are **no specific signs** of ACS on CXR.
  - However, it may show evidence of related left ventricular failure.

- Possible CXR findings

  - Pulmonary oedema.
  - Venous congestion.
  - Pleural effusions.
  - Septal lines.



- **Radiation exposure and fetal risk**
  CXR delivers an estimated fetal dose of **less than 0.0001 mGy.**
  Doses associated with fetal malformation are reported to be: Greater than 50 mGy.
  • Therefore, fetal radiation exposure from a single CXR is far below teratogenic leve

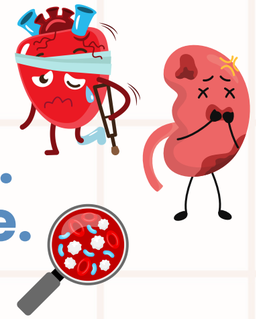
Investigations

Biomarkers → Troponin

There are no benign reasons why cardiac troponin would rise within the maternal population.(Always pathological)

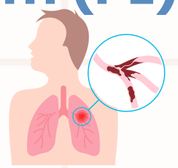
A positive troponin profile must therefore be definitively investigated.
- ACS should be the primary diagnosis to exclude.

Highly sensitive, but nonspecific marker of any form of myocardial injury.

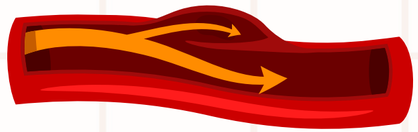


Non-ACS causes of raised troponin:

- Myocarditis.
- Renal failure.
- Sepsis.
- Pulmonary embolism (PE).



- Profound hypertension.
- Cardiomyopathy.
- Aortic dissection



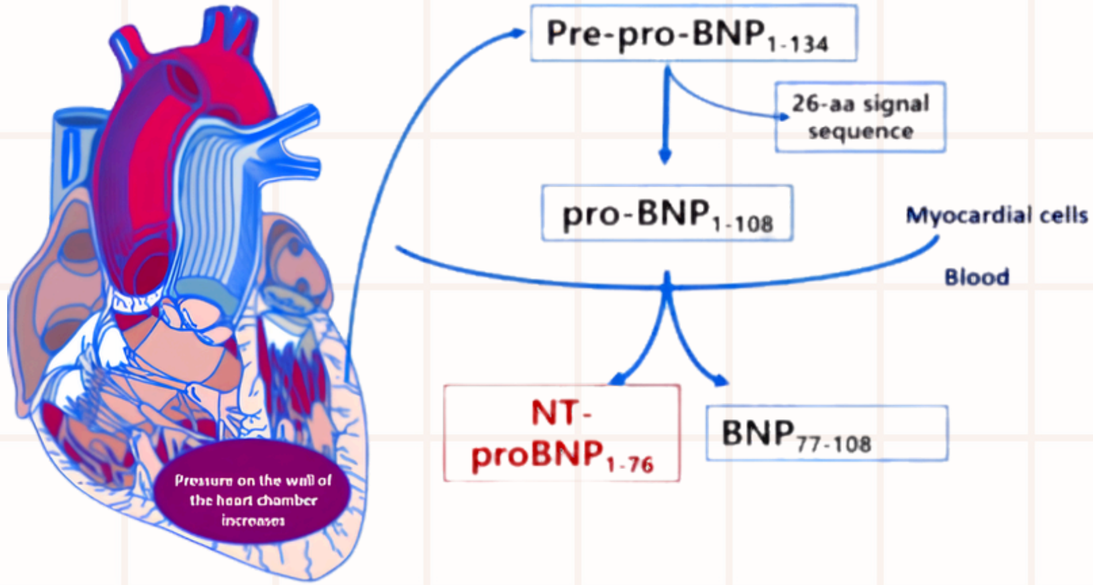
- Takotsubo cardiomyopathy.
- Pre-eclampsia.
- Cardiac decompensation.
- Autoimmune diseases with cardiac involvement.

Investigations

Biomarkers

Brain natriuretic peptide (BNP) and NT-proBNP

- Role in ACS
- Do not typically have a role in the diagnosis of ACS.
- Usefulness
- May be useful in excluding other differentials (Cardiac failure)



Investigations

Echocardiogram (Echo)

General role

Echo can provide a **rapid**, **noninvasive** assessment of:
- Cardiac structure.
- Cardiac function.

Physiological echo changes in normal pregnancy

Increases in:
- Left ventricular (LV) mass.
- End-diastolic left ventricular volume (LVEDV).
- Left atrial (LA) dimensions.
- These changes represent adaptations to increased volume.

- Flow velocities across cardiac valves increase during pregnancy:
- Due to increased cardiac output.
- Crucial point:

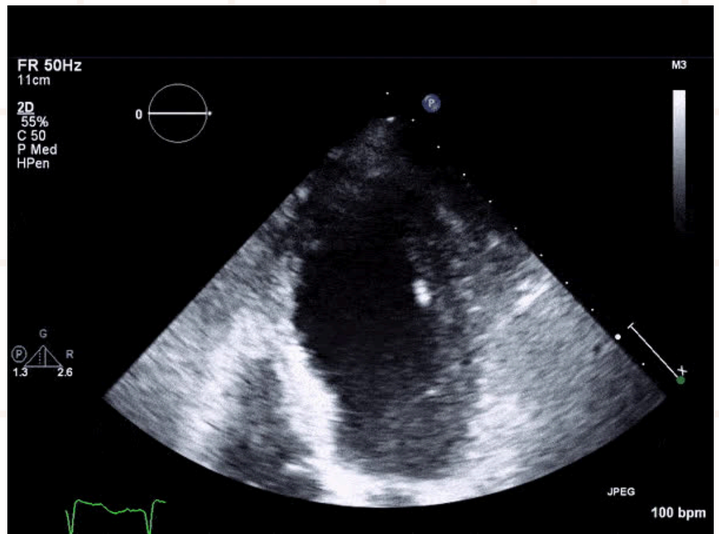
 Left ventricular ejection fraction (LVEF) should:
 Remain stable.
 Stay within normal limits throughout pregnancy in a healthy woman.

Echo findings in ACS

- May demonstrate regional wall motion abnormalities (RWMA):
- In a specific coronary territory.

–The affected region:

Appears less contractile than other unaffected regions



Investigations

Angiography

- **Gold Standard for Emergent Assessment**
- **Angiography is available 24/7 across the UK and allows arterial access to coronary imaging within approximately 5 minutes with immediate potential intervention.**
- **Fetal radiation exposure is low (about 0.075 mGy) with radial access preferred.**
- **Iodinated contrast crosses the placenta but is not considered teratogenic.**
- **Angiography permits definitive diagnosis and immediate therapeutic procedures when required.**

Management: Multidisciplinary Approach with PCI as Gold Standard

Management requires multidisciplinary team comprising obstetricians, anaesthetists, cardiologists, paediatricians, and the woman. Care provided in environment with cardiac and obstetric intensive care capabilities

Step 1: Diagnostic Angiography

Arterial access **(radial preferred)**, catheter manipulation into coronary artery, vessel engagement and imaging.

Step 2: Revascularisation

Wire inserted proximal-to-distal through occlusion; options include balloon angioplasty, thrombus aspiration, or localised lysis

Step 3: Stent Placement

Drug-eluting stents now preferred (same DAPT requirement as bare metal) to maintain coronary flow and prevent restenosis.

Important considerations:

Increased risk of coronary dissection as PCI complication,PASCAD may propagate with intervention, Larger role for conservative management based on haemodynamic stability.

However: Pregnancy is NOT a contraindication for PCI

Timing: Delay until after 4th month if possible (post-organogenesis), but immediate intervention if haemodynamically unstable

Thrombolysis relatively contraindicated due to bleeding risk.

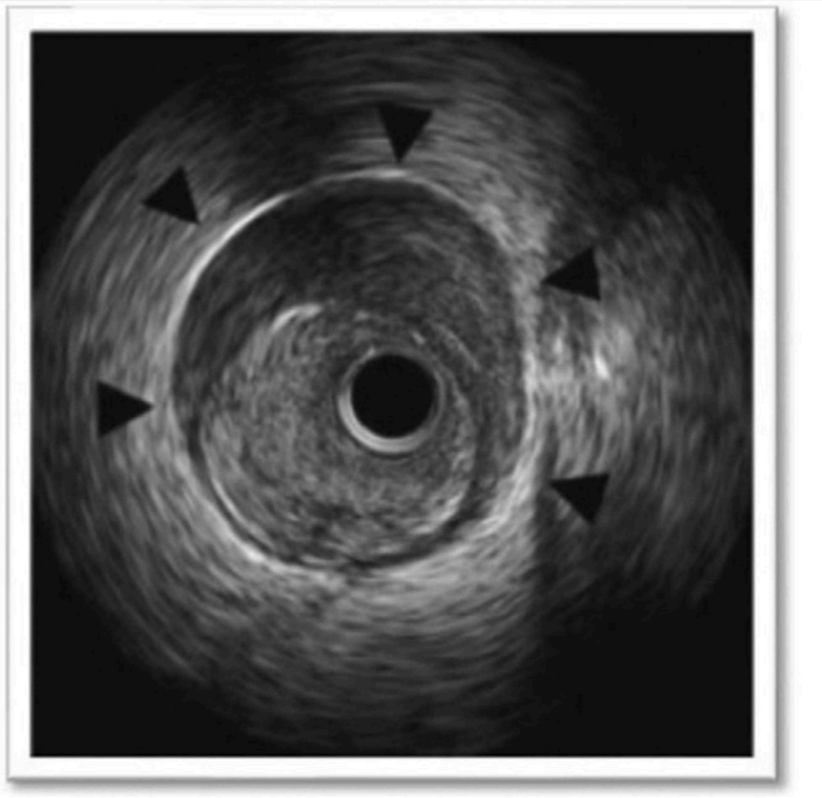


Figure 2. Intravascular ultrasound (IVUS) of the left anterior descending coronary (LAD) artery. Black arrows demonstrate the false lumen secondary to pregnancy-associated spontaneous coronary artery sissection (PASCAD).

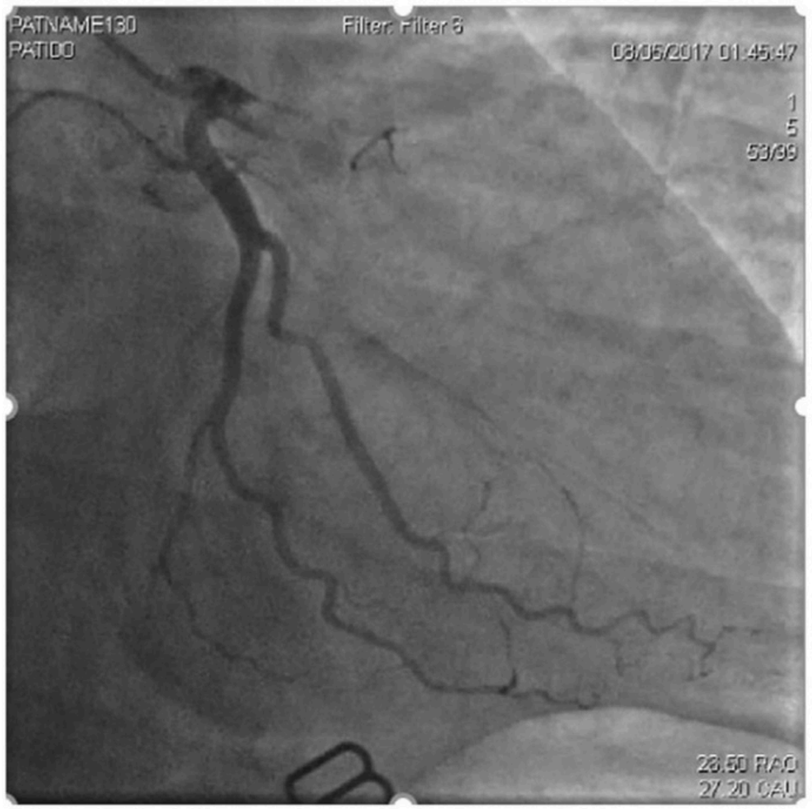


Figure 3. Right anterior oblique caudal (RAO caudal) of the left coronary system demonstrating an occluded LAD artery.

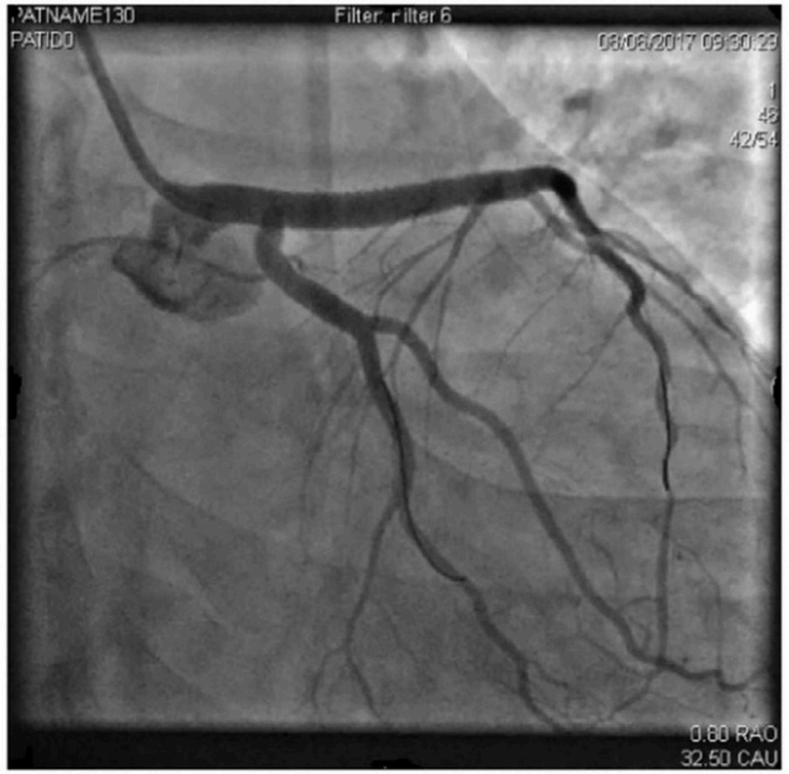


Figure 4. RAO caudal of the left coronary system demonstrating a revascularised LAD artery following percutaneous intervention (PCI). In this case the LAD was stented to reopen the vessel and seal the dissection flap closed.

Pharmacotherapy: Balancing Maternal Benefit and Fetal Safety

01 Acute Phase Medications

- **Aspirin**: 300mg loading, then 75mg daily. Safe in pregnancy and breastfeeding.
- **Heparin**: Therapeutic subcutaneous dosing. LMWH and UFH safe but require careful bridging around delivery.
- **Clopidogrel**: 600mg loading, then 75mg daily. Emerging safety evidence, recent review shows acceptable risk. Long half-life, stop 5-7 days before regional anaesthesia.
- **DAPT**: Required for specific periods post-stent to prevent catastrophic restenosis.
- **Nitrates**: For chest pain but may precipitate uterine hypoperfusion.
- **Support**: Inotropes or mechanical augmentation for refractory instability.

02 Secondary Prevention

- **Beta Blockers:** Labetolol (most hypertension data), Bisoprolol (preferred for IHD, more cardioselective). Associated with fetal hypoglycaemia and growth restriction.
- **ACE inhibitors/ARBS/ARNIS:** Contraindicated in pregnancy (teratogenic). ACE inhibitors can be used in breastfeeding
- **Statins**: Should be avoided, not proven safe
- **Diuretics**: Furosemide safe but use cautiously. Thiazides and spironolactone avoid in pregnancy.
- **Anticoagulants**: Direct oral anticoagulants insufficient safety data. Warfarin teratogenic but safe in breastfeeding.

Delivery and Future Pregnancy: Individualized Planning Essential

➢ Delivery Timing and mode

Outside extreme circumstances, delivery is **rarely** recommended acutely. Preferably delay delivery several weeks from the acute event to allow maternal recovery. Delivery does not need to precede PCL. Aim for delivery by **40 weeks gestation** when possible. Mode of delivery should be determined by obstetric factors, **induction of labour is considered safe**.
Prostaglandin E and oxytocin can be used.

Avoid prostaglandin Fand **ergometrine** due to vasoconstriction risk.

For the second stage, consider allowing two hours of passive descent before active pushing; use instrumental delivery for routine obstetric indications

Delivery and Future Pregnancy: Individualized Planning Essential

➢ Future pregnancy risk assessment

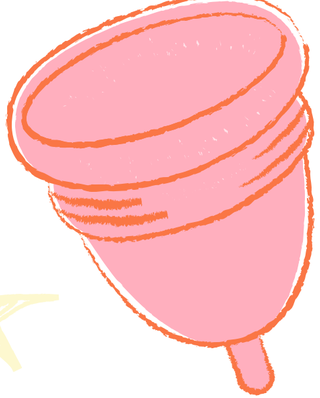
Recurrence risk is approximately **9%** in women with a history of ischemic heart disease

Pregnancy should be delayed for **at least 12 months** after an acute coronary event

Use risk stratification with the modified WHO classification (mWHO), Women in mWHO class III or IV should receive careful counselling and may be advised against pregnancy, If already pregnant and assessed as high risk, discussion of termination of pregnancy is appropriate; surgical termination may be preferred over medical termination because of a lower failure rate

➢ Preconception Optimization

Preconception care requires specialist multidisciplinary team management in a tertiary unit, including an obstetrician with obstetric medicine expertise, an experienced cardiologist, and an obstetric anaesthetist.
Review medications and switch teratogenic drugs to safer alternatives where possible. Address modifiable risk factors such as obesity, smoking, alcohol use, glycaemic control, and blood pressure. Effective contraception is essential for those not planning pregnancy or still optimizing health. Ensure regular cardiac investigations and close monitoring throughout any subsequent pregnancy



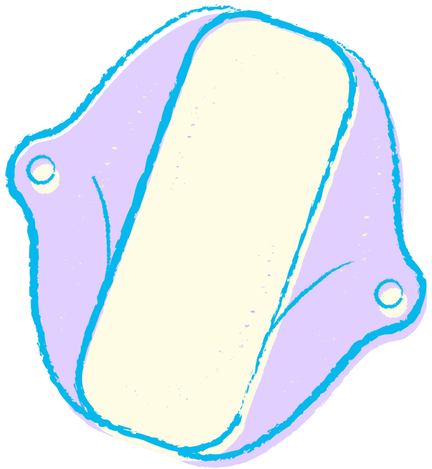
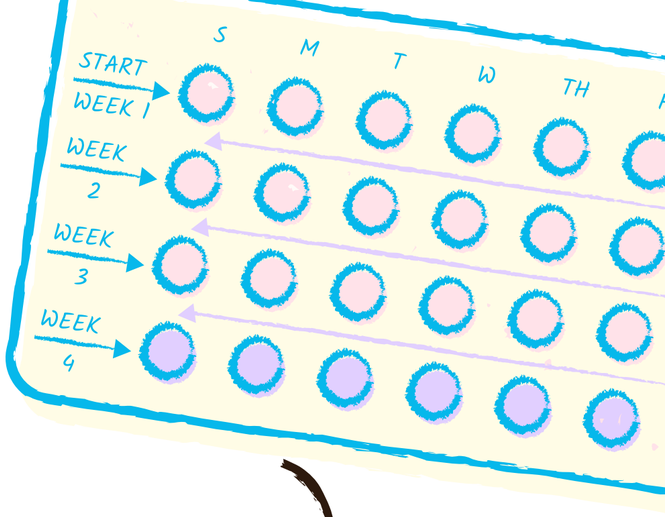
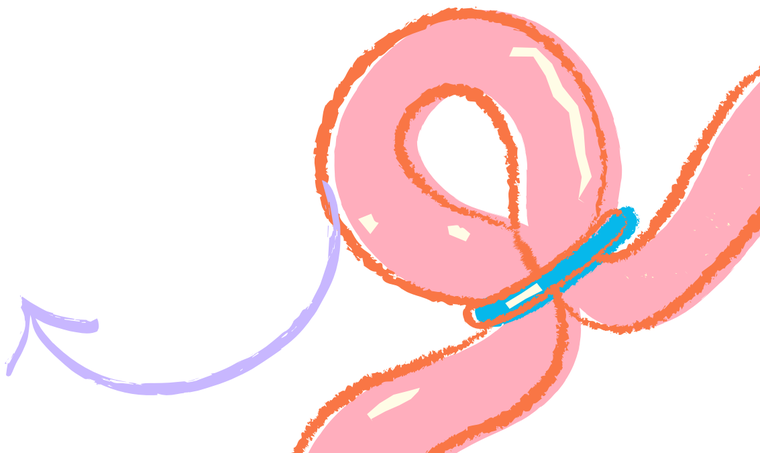


Table 1. Contraceptive options in women with a history of ischaemic heart disease

Method of contraception	Contraindication(s)
Barrier methods	Not recommended alone owing to high failure rates
Combined hormonal contraceptive	Avoid (UKMEC 4) in IHD, chronic heart failure and stage 2 hypertension owing to increased risks of VTE, MI and worsening hypertension
Systemic progesterone-only contraceptives	No contraindications
Intrauterine methods	Insert in hospital setting because of the risk of vasovagal collapse secondary to cervical stimulation at time of insertion in women with residual left ventricle dysfunction
Sterilisation	Laparoscopy may not be possible in women with severely impaired cardiac function
Emergency contraception	Levonorgestrel, ulipristal acetate and Cu-IUD are safe

Cu-IUD = copper intrauterine device; IHD = ischaemic heart disease; MI = myocardial infarction; UKMEC = UK Medical Exclusion Criteria; VTE = venous thromboembolism





Thank You