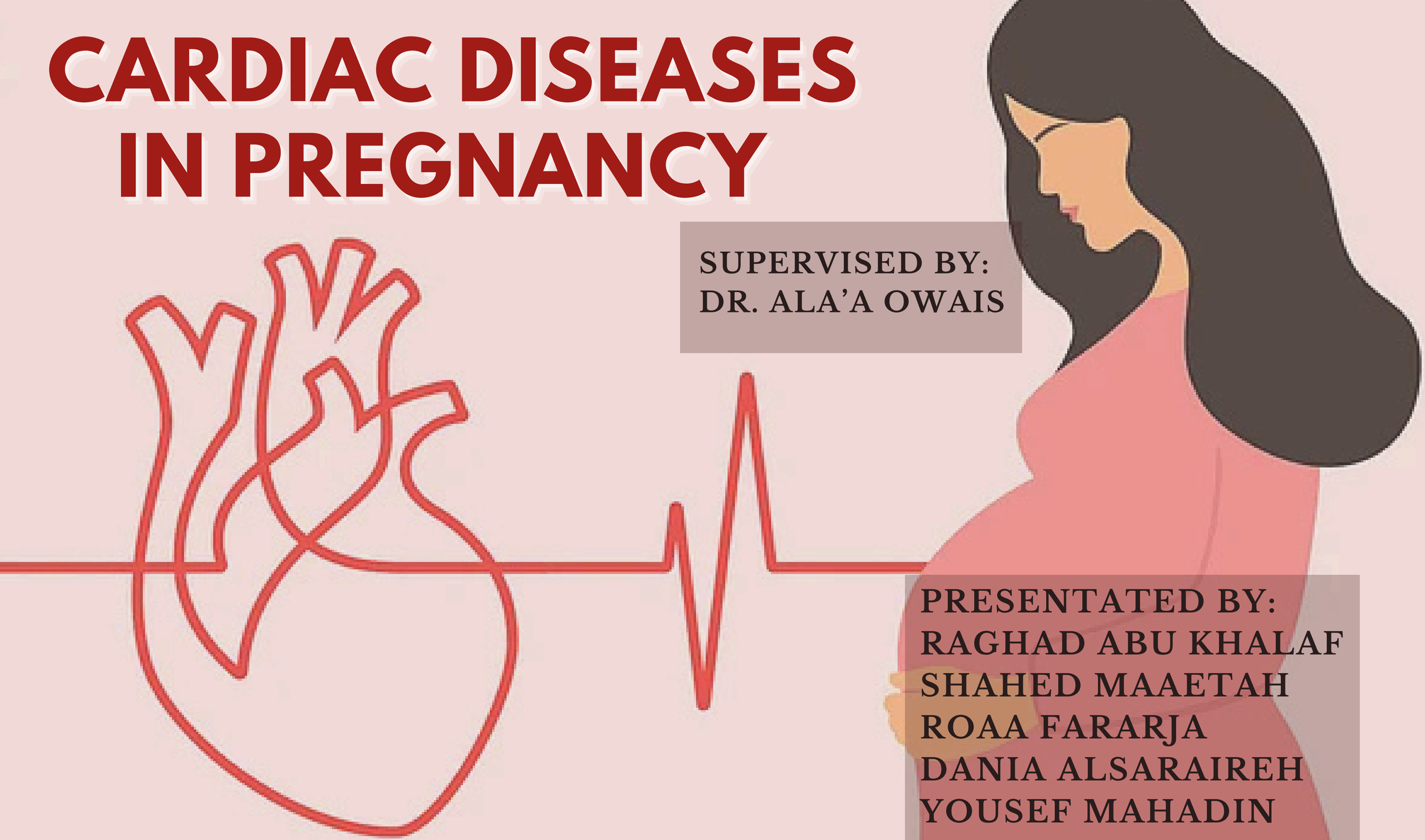
CARDIAC DISEASES IN PREGNANCY



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KEY CONCEPTS

PHYSIOLOCAL CHANGES

ECG CHANGES

ASSESSMENT

TREATMENT

DEFINITION

Palpitations are defined as an unpleasant awareness of the heartbeat. in which the sensations may be forceful, irregular or fast.

- **Palpitations are common complaint during pregnancy.**
- **Most are benign (due to normal physiology).**
- **Some may indicate significant underlying arrhythmias; (Most common cardiac complication in pregnancy)**
- **Multidisciplinary management is required in pre-existing significant arrhythmias or cardiac disease (congenital,structural)**
- **Palpitations associated with syncope or pre-syncope should always prompt further investigations**
- **Many anti-arrhythmic drugs are relatively safe in pregnancy, prompt and should be started with without delay**

• A cohort study of 1802 women with congenital heart disease, prevalence of arrythmias found to be 4.7% ,
•In another cohort study, women who had sustained tachyarrythmias prior pregnancy, 43% of them had recurrence during pregnancy or in the first month postpartum , Arrythmias may occur Denovo during pregnancy

KEY CONCEPTS

PHYSIOLOGICAL CHANGES

ECG CHANGES

ASSESSMENT

TREATMENT

PHYSIOLOGICAL CHANGES IN PREGNANCY

- ↑ Plasma volume (up to 50%) → ↑ cardiac output by approximately 50%
- ↑ Heart rate (10–20 bpm) mainly in 3rd trimester
- ↓ Systemic vascular resistance. so, largely increase stroke volume
- ↑ blood volume

During Intrapartum: COP rises further with SV and HR rising
Postpartum : changes in HR are less well defined, in which one study found that Heart rate is 110 bpm within the 1st 48 h postpartum

ECG CHANGES

DEMONSTRATE AN INCREASE IN HEART RYTHM. IT MAY CONTAIN :

Left axis deviation 1

- within normal range due to rotation of the heart from the gravid uterus

Inverted or flattened T waves 2

- in leads lll
- Leads V1-V3

Q wave commonly seen 3

- in leads ll,lll and aVF

Atrial , ventricular ectopic beats

on continuous ECG

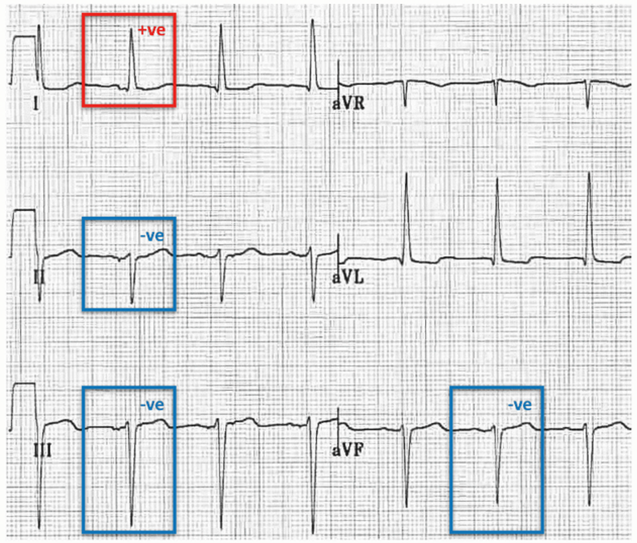
IMPORTANT

ectopic beats are often harmless, sustained arrhythmias like AF or ventricular tachycardia require urgent attention. Pregnancy also unmasks previously silent conditions. Always rule out secondary contributors.

Arrythmogenesis may be related to: combination of

1. Hemodynamic : increased B.V that leads to increase atrial and ventricular stretch
2. Autonomic
3. Hormonal factors : Higher levels of estrogen, may increase alpha-adrenergic receptors, so enhanced adrenergic response

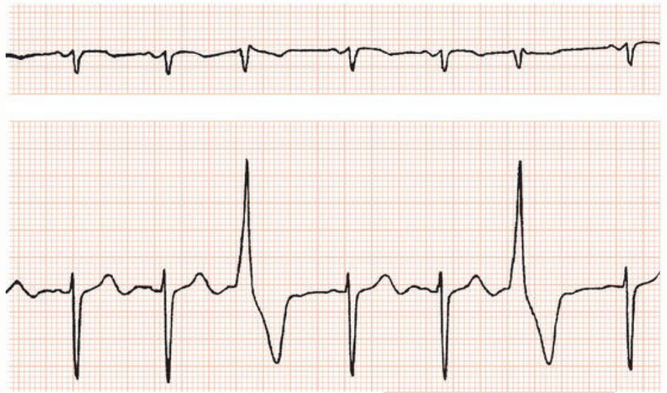
HERE DDX OF PALPITATIONS



LEFT AXIS DEVITION

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



ECTOPIC BEATS

Initial Assessment

History

- onset , frequency and duration ,regularity.
- fast heartbeat, often at rest and particularly when lying down, Or thumping sensation
- compensatory pause from ectopic beats
- family history of sudden cardiac death

examination

- Basic observations: manual palpation of pulse, B.P, R.R, O2 sat. and temp.
- CVS: Auscaltation of heart to elicit any abnormal heart sounds (murmurs)
  a flow murmur is common finding inpregnancy
- RS examination is also useful to determine any features suggestive pulmonary oedema or Lung pathology; infection

Investigations

- is mandatory in the context of presentation with cardiac symptoms to determine who is at risk of arrythmia
- 12- lead ECG (GOLD STANDARD)
- Blood: Hb to exclude anemia
- Thyroid to exclude thyrotoxicosis
- Ambulatory ECG monitoring
- Echocardiogram

Baseline ECG

- An abnormal baseline ECG may indicate underlying pathology and is therefore an essential part of arrhythmia assessment.
- For example, the ECG of a woman with Wolff-Parkinson-White syndrome may show a delta wave; high voltages in the precordial leads with Q wave and ST changes may be seen in hypertrophic cardiomyopathy
  Any variant other than those already described as normal changes in pregnancy should prompt a second opinion from a cardiologist

Ambulatory ECG monitoring :

- Holter monitor, the most used monitor in pregnant women presenting with palpitations
- is an external recorder connected to the woman by skin electrodes. It is commonly used for periods of 24–72 hours
- Interpretation the results of a holter monitoring:
  1. symptoms correspond with sinus tachycardia and other investigations are normal, with no suspicion of PE or sepsis, reassure that it's physiological and no other investigations needed
  2.Infrequent atrial or ventricular ectopic beats, without suspicion of structural heart disease, no need for further investigations
  3. If there's significant arrythmia and the patient is symptomatic, seek urgent advice from cardiologist

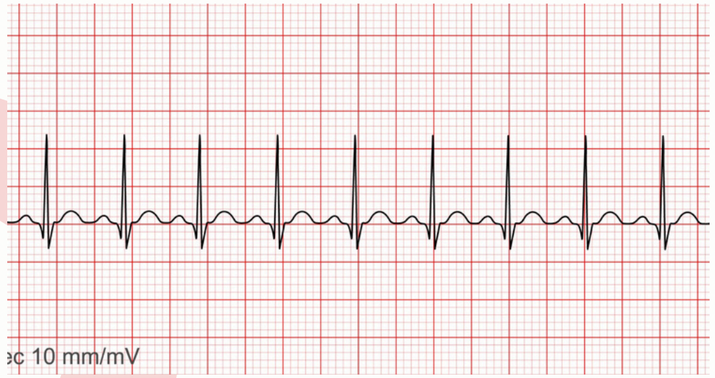
Echocardiogram

- required if structural heart disease needed to be excluded needed when there is
  1. Diagnosed arrythmia
  2. Audible heart murmur
  3. Known structural heart disease
  4. Previous chemotherapy with cardio-toxic agents
  5. Family History of inherited arrythmia, e.g: long QT syndrome, sudden cardiac death

TACHYARRYHMIAS

Sinus tachycardia 1

- Most are benign and do not require treatment, but there are alternative diagnosis: inappropriate sinus tachycardia and postural orthostatic hypotension (POTS) should be considered



Atrial and ventricular premature beats 2

- benign, patient may have palpitations or asymptomatic and very common in pregnancy, detected in more than 50% of pregnant women in a holter monitor study, treatment is not usually required

Supraventicular Tachycardia 3

- the most common non-benign arrhythmia in pregnancy, with a frequency of 24 in 100,000 with a first onset range from 3.8 to 34%
- presents as a palpitation with abrupt onset and offset.
- It comes with syncope or chest pain and related haemodynamic compromise, influenced by structural heart disease if also present
- Most common types: paroxysmal Supraventricular Tachycardia (PSVT)

TACHYARRYTHMIA

Atrial fibrillation Atrial flutter 4

- usually associated with mitral stenosis so women should be anti-coagulated (LMWH). new episodes in pregnancy should prompt investigation and treatment of an underlying cause

- AF : atrial muscle fibres contract independently in an irregular manner, resulting in the absence of P waves and irregular ventricular contraction.

- In atrial flutter, the atria beat regularly at a rate of 300 beats per minute with a ventricular rate of 150 beats per minute (in 1:2 conduction), resulting in a characteristic ‘saw tooth’ appearance on the ECG.

Ventricular tachycardia 5

- VT occurs when the ventricles depolarize rapidly, producing a wide, abnormal QRS complex

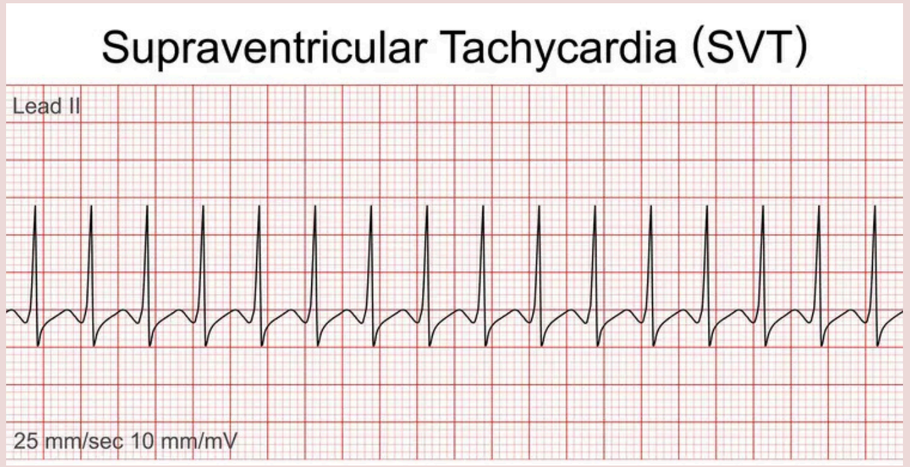
- It is often linked to structural heart disease or primary electrical disorders, which must be excluded in affected women

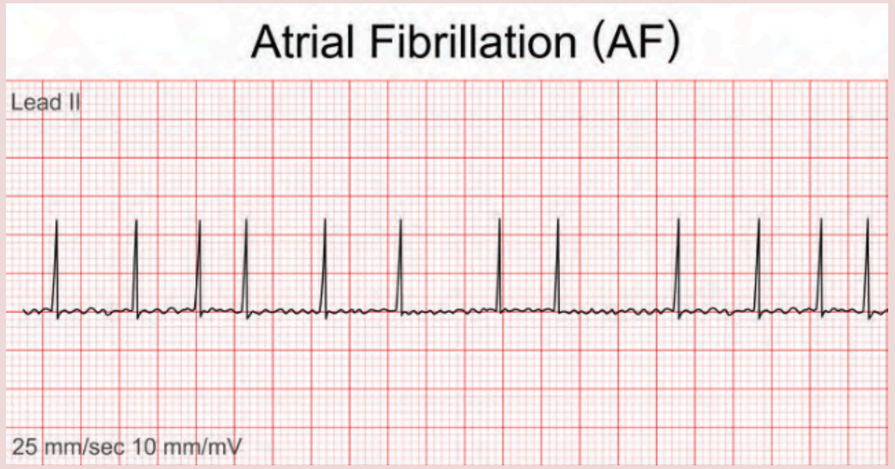
- Idiopathic VT in healthy women usually arises from the right ventricular outflow tract (RVOT), presenting as non-sustained VT with a left bundle branch block pattern; treated with beta-blockers or verapamil.

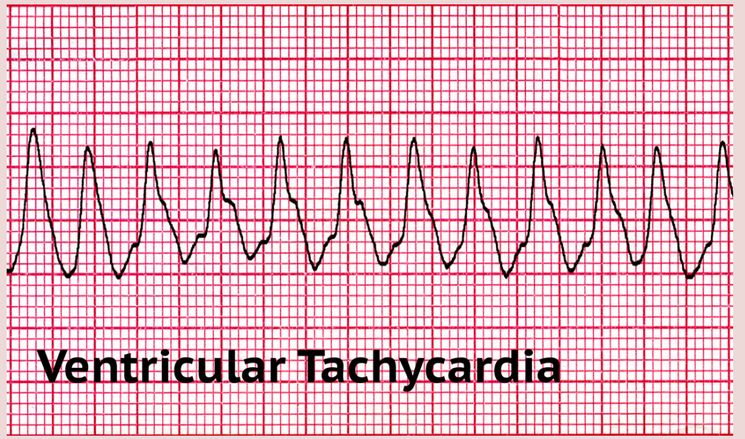
Long QT Syndrome (LQTS) 6

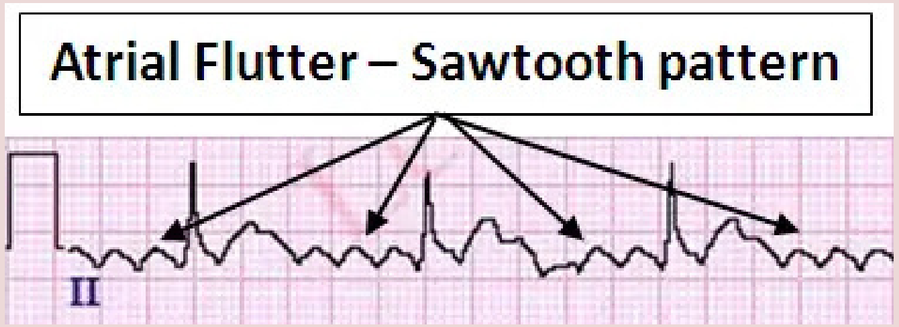
- prolonged QT interval due to abnormal ventricular repolarization, predisposing to ventricular arrhythmias (e.g., torsade de pointes) and sudden death.
- Most commonly linked to mutations in **KCNQ1, KCNH2, and SCN5A.**
- Risk is lower in pregnancy but rises significantly postpartum, especially in type 2 LQTS. Sleep disruption and missed medication further increase risk.
- Women should continue beta-blockers during and after pregnancy LQTS has been associated with sudden infant death; gene variants are found in ~10% of cases.

TACHYARRYTHMIA









KEY CONCEPTS

TACHYARRYTHMIAS

BRADYARRYTHMIAS

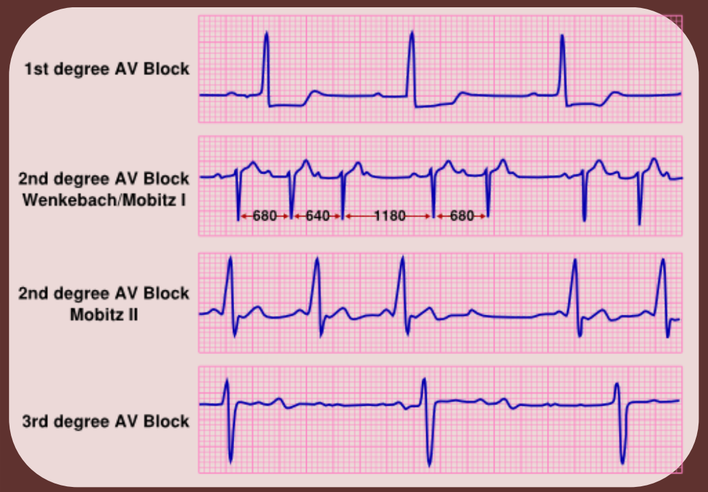
TREATMENT

BRADYARTHYMIAS IN PREGNANCY

- Rare and usually well tolerated.
- In structurally normal hearts:

First-degree block → benign.

• Second-degree (Wenckebach) → usually not symptomatic.



Complete heart block:

• May be congenital or acquired (often after congenital heart surgery).

• Sometimes first diagnosed in pregnancy.

• Asymptomatic → pregnancy and delivery generally well tolerated.

•Symptomatic→pacing recommended (temporary during delivery or permanent, both safe in pregnancy)

TREATMENTS

SVT:

• First line → vagal maneuvers (valsalva's maneuver)

• Adenosine IV (safe, short half-life)

AF/flutter:

• Rate control: Preferred drugs in the acute setting and structurally normal heart: I.V flecainide or butilide

• for Acute episode: direct current cardioversion when cardioversion cannot be achieved or in rate control prophylaxis : first line agent is Beta blocker

Ventricular arrhythmias

If unstable → electrical cardioversion is preferred

If stable → pharmacological cardioversion with drugs like sotalol or flecainide

• Prevention in high-risk women: beta-blockers, amiodarone, or an implantable cardioverter defibrillator (ICD), which may also be used for secondary prevention.

PRE-EXISTING ARRHYTHMIAS MANAGEMENT

- Preconception
  1. Condition-specific device on risk in pregnancy
  2. Review of medication and changes advised if appropriate
  3. Optimisation of condition prior to conceiving; consider referral for accessory pathway ablation

- Antenatal :
  1. Review of medication
  2. Growth scans if on beta-blockers
  3. Anaesthetic review and planning
  4. Planning for birth

PRE-EXISTING ARRHYTHMIAS MANAGEMENT

• Intrapartum

-Vaginal birth usually recommended

-Consider place of birth if risk of arrhythmia high; appropriate facilities

-Consider continuous cardiac monitoring in those at high risk

-Care plan to include advices, ensure drugs/facilities available in advance, and drugs to be avoided

• Postnatal

-Period of inpatient monitoring

-Some conditions high risk of postnatal event, long QT syndrome

-Plans for medication and breastfeeding

-Ensure plans for continuing cardiological care/investigations

ANTIARRYTHMIC 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

PERIPARTUM CARDIOMYOPATYY

Done by: Shahd Ayouben

DEFINITION

The European Society of Cardiology (ESC) defined PPCM as:

- **Idiopathic cardiomyopathy**
- **Presenting with heart failure secondary to left ventricular systolic dysfunction**
- **Towards the end of pregnancy or up to 6 months following delivery**
- **Where no other cause of heart failure is found It is a diagnosis of exclusion.**
- **The left ventricle may not be dilated but the ejection fraction is nearly always reduced below 45%.**

EPIDEMIOLOGY

The estimated incidence in Western health care ranges from 1 in 1000–4000 pregnancies, but certain areas have a greater disease burden.

RISK FACTORS:

1. **Race: The disease more commonly manifests in women of Afro Caribbean lineage and carries a poor prognosis.**
2. **Multiparity**
3. **Multiple pregnancy**
4. **Obesity**
5. **Chronic hypertension**
6. **Pre-eclampsia (There is a close association between pre-eclampsia and PPCM, which is hypothesised to be associated with a synchronous pathophysiology)**
7. **Advanced maternal age(Even though the disease can strike women of any age, those older than 30 years of age are at higher risk)**

2013 meta-analysis:

- Pre-eclampsia in 22% of women with PPCM
- This is 4× higher than the global general population prevalence (5%)

DEFINITION & EPIDEMIOLOGY

RISK FACTORS

PATHOGENESIS

DIAGNOSIS

TREATMENT

A comprehensive understanding of the exact pathogenesis of PPCM continues to elude clinicians and scientists.

The earliest theories proposed to explain the pathophysiology which were later disproven:

VIRAL-INDUCED MYOCARDITIS ×

proved a popular theory; however, subsequent testing of endocardial biopsies at autopsy demonstrated **comparable viral serologies** between PPCM biopsies and control groups.

ADAPTIVE CHANGES TO MATERNAL PHYSIOLOY ×

it has been speculated that the associated haemodynamic stress placed upon the heart may contribute to PPCM.

WHY WERE THEY DENIED ?

(**MRI**) has not demonstrated imaging characteristics consistent with myocarditis.

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 into the postpartum period.
Therefore, the disease timeline does not correlate

PATHOGENESIS

The true overarching pathophysiology is now considered to be multifactorial, with a single theory unlikely to provide unification.

Recent attempts at exploring PPCM aetiology have proposed a ‘two-hit’ model of genetic predisposition combined with a vascular hormonal insult to the maternal heart in and around term, as illustrated in next slides

GENETIC FACTOR

- Strong regional and familial patterns exist in the prevalence of PPCM, implying a strong genetic basis to the condition.
- Multiple genetic targets are associated with the condition including TTN, TTNCI, BAG3, PTHLH and PGC-1a.
- Many of these genes regulate myocyte function, with mutations predisposing to PPCM.

VASCULAR HORMONAL MODELS

1 STAT3 Activation

During pregnancy and postpartum, transcription factor STAT3 is highly activated in the heart

2 Prolactin Increase

Maternal pituitary secretes higher levels of prolactin.

3 Reduced STAT3 Effects

If STAT3 expression is reduced → increased reactive oxidative species (ROS).

- ROS increase levels of cathepsin D.

4 Evidence in Humans and Mice

Reduced STAT3 + higher 16-kDa fragment found in biopsy/transplant samples of women with PPCM.

Bromocriptine (prolactin antagonist) reverses systolic impairment in STAT3-deficient mice.

5 MicroRNA Involvement

16-kDa fragment increases microRNA-146a → blocks signaling pathways → contributes to cardiac myocyte death

6 Prolactin Cleavage

Cathepsin D cleaves prolactin into a 16-kDa fragment → causes cardiac endothelial and capillary dysfunction.

VASCULAR HORMONAL MODELS

Increase sFlt-1 (1)

- The placenta secretes exponentially increasing concentrations of soluble fms-like tyrosine kinase receptor 1 (sFlt-1)

Inhibiting VEGF&PIGF (2)

Raised sFlt-1 alters systemic angiogenesis by inhibiting vascular endothelial growth factor (VEGF) and placental growth factor (PlGF)

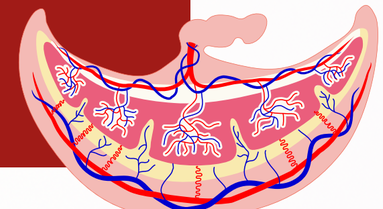
Endothelial damage (3)

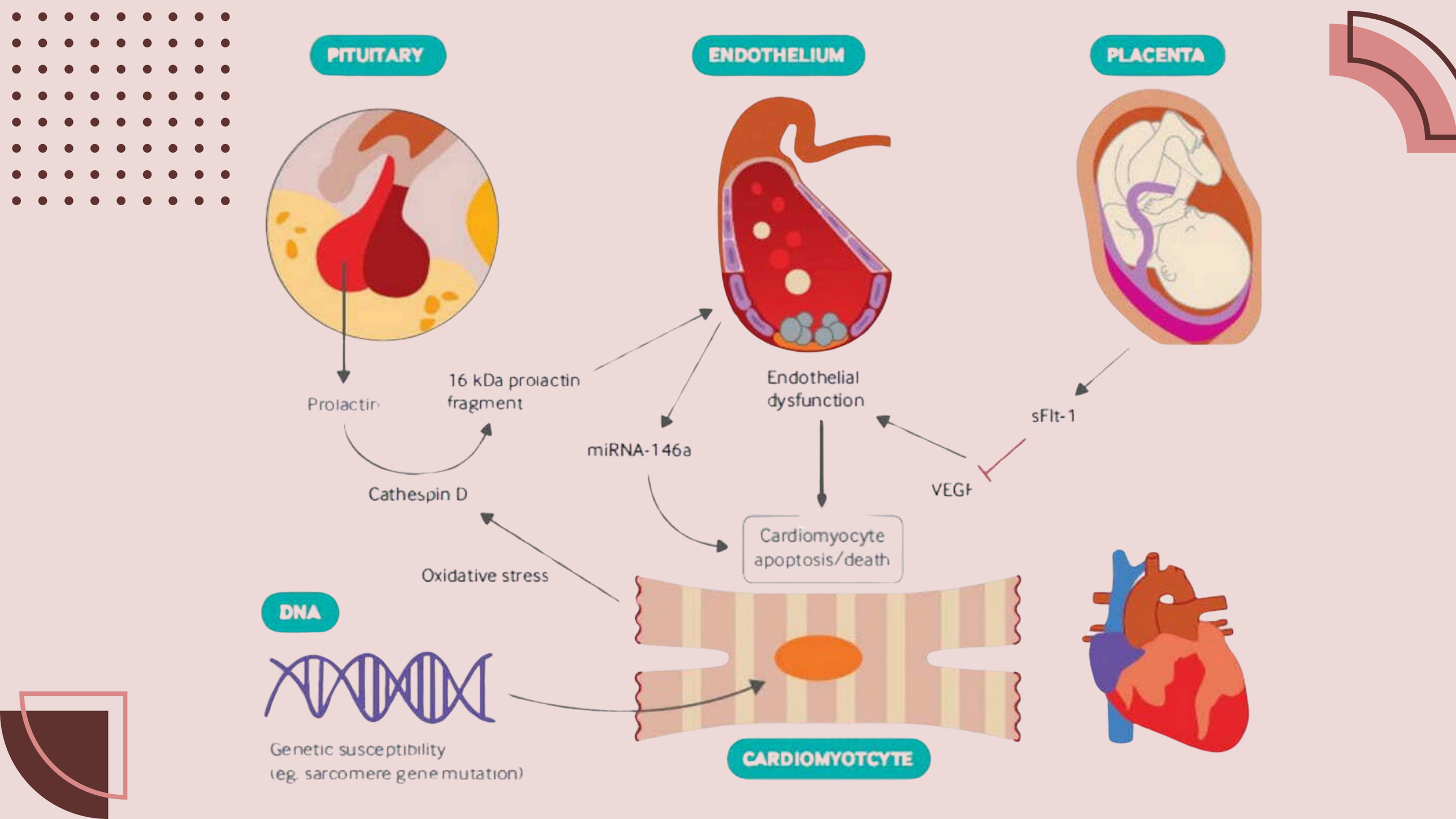
Inhibition of VEGF & PIGF resulting in further endothelial damage.

IMPORTANT

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.




PITUITARY
ENDOTHELIUM
PLACENTA
Prolactin
16 kDa prolactin fragment
Cathespin D
miRNA-146a
Endothelial dysfunction
sFlt-1
VEGF
Cardiomyocyte apoptosis/death
Oxidative stress
DNA
Genetic susceptibility
(eg. sarcomere gene mutation)
CARDIOMYOTCYTE

CLINICAL PRESENTATION

- The presenting features of PPCM can be similar to the physiological symptoms of pregnancy and the postpartum period,which can sometimes lead to a diagnostic delay.

Symptoms of heart failure

- Dyspnoea
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Pedal edema
- Unexplained cough, particularly when lying down
- produces frothy pink sputum
- Dizziness
- Palpitation
- Fatigue
- Chest pain
- Abdominal discomfort (caused by hepatic congestion and precordial pain)

Clinical features

History

- known congenital or acquired heart disease?
- Family history of ischaemic/non-ischaemic heart failure?

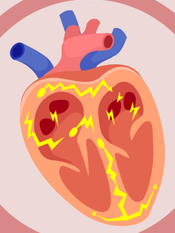
examination

- tachypnoea
- tachycardia
- peripheral oedema
- jugular venous distension.

Ddx

- Benign dyspnea of pregnancy
- cardiomyopathy and valvular disease
- myocardial infarction
- pulmonary embolism
- HIV cardiomyopathy

COMPLICATIONS OF THE DISEASE



1.ventricular arrhythmias

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.develop 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.

INVESTIGATIONS

1.ELECTROCARDIOGRAM

- There are no ECG findings that are specific to PPCM
- common features in these women include sinus tachycardia with nonspecific ST segment and T wave abnormalities
- can be used as discriminating tool for conditions presenting in a similar manner

2.LABORATORY TESTS

- Elevated C-reactive protein and leucocyte count which is non specific
- marker for heart failure Brain natriuretic peptide (BNP) or N-terminal pro-brain natriuretic peptide (NT-proBNP)
- cardiac enzyme troponin T often normal

3.CHEST X-RAY

- nonspecific for PPCM
- may show typical features of cardiac strain, such as cardiomegaly, pulmonary oedema, or pleural effusion

INVESTIGATIONS

ECHOCARDIOGRAPHY

- Most useful tool: Echocardiography is the primary investigation for peripartum cardiomyopathy (PPCM).

Advantages: No radiation risk and widely accessible.

Diagnostic criterion: Left ventricular ejection fraction (LVEF) < 45% is essential for diagnosis.

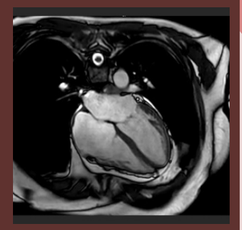
Prognostic value: Provides information on likely recovery and outcomes.

Right ventricular dysfunction: Rare but associated with worse prognosis.

Recovery predictors: LV size and LVEF at diagnosis are the best indicators.

LV end-diastolic diameter (LVEDD) > 6 cm or LVEF < 30% → lower chance of spontaneous recovery, higher risk of mechanical support, transplant, or death.

CARDIAC MRI



• Useful for detailed cardiac imaging.

Accurately measures chamber volumes and systolic function.

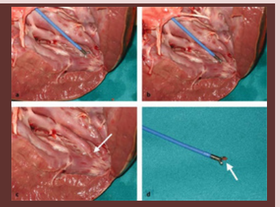
• More sensitive than echocardiography for detecting intracardiac thrombus.

• Assesses tissue injury: intracellular/interstitial damage, hyperaemia, capillary leakage, necrosis, fibrosis.

Limitations: Not first-line in pregnancy due to gadolinium risks and limited availability.

Breastfeeding: Safe after gadolinium administration.

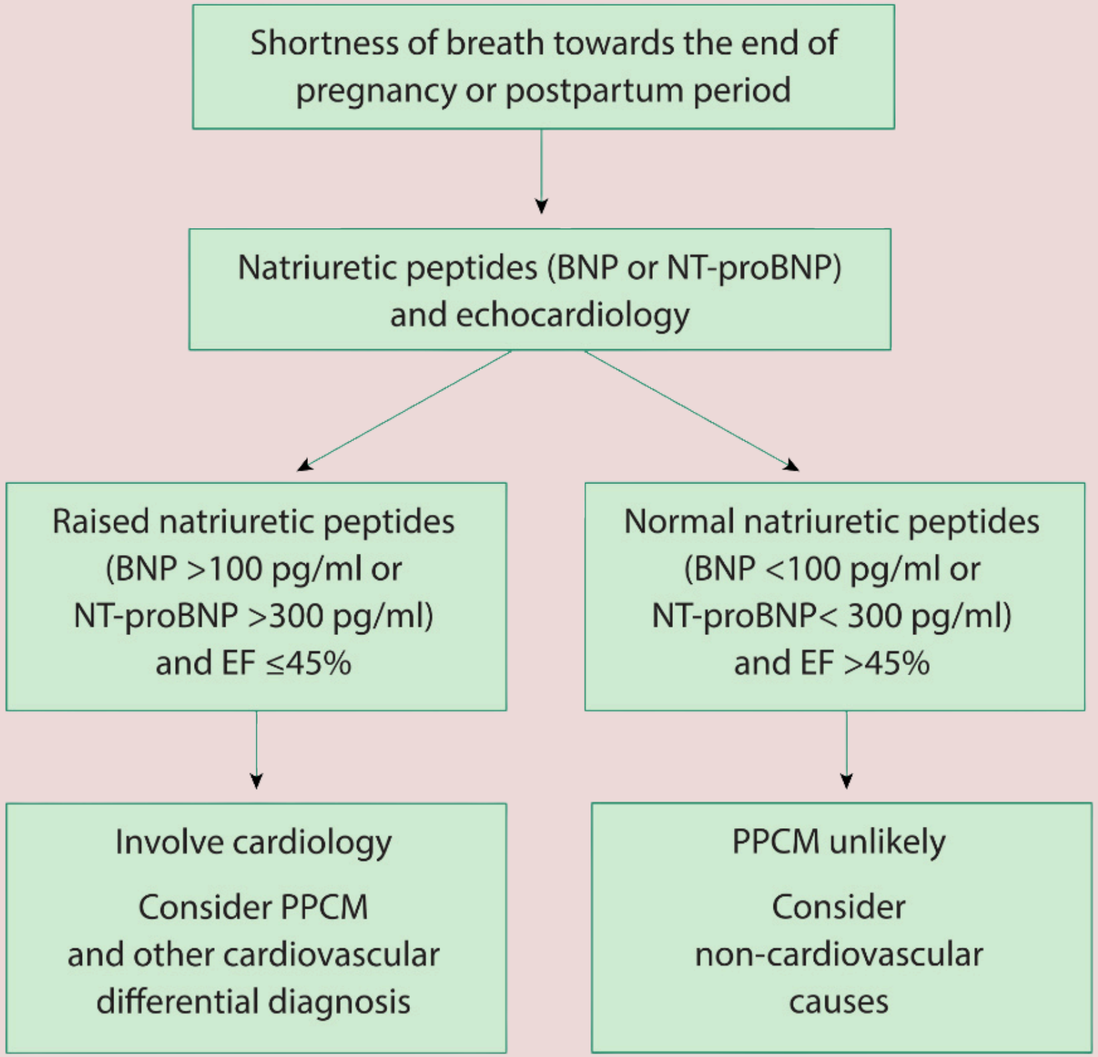
ENDOMYOCARDIAL BIOPSY:



Rarely performed.

Mainly used to rule out other causes of LV dysfunction when diagnosis is uncertain.

INVESTIGATIONS



Case - Differential Diagnoses

Case summary

• 32-year-old woman G2P1, 2 weeks postpartum Symptoms Presents with progressive dyspnoea, orthopnoea, oedema, palpitations.

Examination

tachycardia, raised JVP, basal crackles

Investigation

Echo: LVEF 30%.

Differential Diagnoses

- **Peripartum cardiomyopathy**
- **Idiopathic dilated cardiomyopathy**
- **Valvular heart disease (mitral stenosis, aortic stenosis)**
- **Pulmonary embolism**
- **Amniotic fluid embolism**
- **Myocardial infarction or ischaemic heart disease**
- **Arrhythmias**
- **Asthma**

MANAGEMENT

- ## General Principles

•MULTIDISCIPLINARY TEAM: OBSTETRICIAN, CARDIOLOGIST, INTENSIVIST, NEONATOLOGIST, ANAESTHETIST.

•STABILISE MATERNAL CONDITION BEFORE DELIVERY IF POSSIBLE.

•INVESTIGATE ALL CARDIAC SYMPTOMS THOROUGHLY.

•THERAPY TAILORED TO PREGNANCY STATUS AND HAEMODYNAMIC STABILITY.

MANAGEMENT

Medical Therapy – Pregnancy

- •Salt restriction, loop diuretics for symptom relief (use cautiously).
- •Beta-blockers (prefer metoprolol).
- •Hydralazine + nitrates for afterload reduction.
- •Anticoagulation with LMWH if LVEF ≤35% or AF.
- •Avoid ACEi, ARB, MRA (teratogenic).

MANAGEMENT

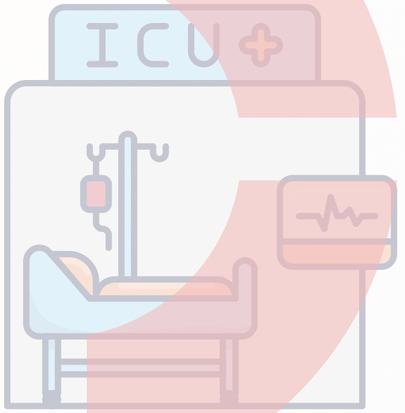
Medical Therapy – Postpartum

- •Breastfeeding: ACEi (enalapril, captopril), ARBs, MRAs, beta-blockers safe.
- •Anticoagulation: LMWH or warfarin safe in lactation.
- •Not breastfeeding: all HFrEF drugs available – ACEi, ARB, ARNI, SGLT2 inhibitors, DOAC.
- •Continue therapy until LVEF recovery (6–12 months).

Your paragraph text

HAEMODYNAMICALLY UNSTABLE

- ADMIT TO ICU – RAPID INTERVENTION REQUIRED.
- MAINTAIN PRELOAD: IV DIURETICS, VASODILATORS IF SBP >110 MMHG.
- OXYGENATION: SPO2 >95%, CPAP OR INTUBATION.
- CIRCULATORY SUPPORT: PREFER LEVOSIMENDAN OVER CATECHOLAMINES.
- URGENT DELIVERY IF UNSTABLE DURING PREGNANCY.
- CONSIDER BROMOCRIPTINE (WITH ANTICOAGULATION).
- MECHANICAL SUPPORT: IABP, IMPELLA, ECMO.



MECHANICAL & ADVANCED THERAPIES

- SHORT-TERM: IABP, IMPELLA, ECMO FOR CIRCULATORY/OXYGENATION SUPPORT.
- LONG-TERM: LVAD/BIVAD – BRIDGE TO RECOVERY OR TRANSPLANT.
- ICD OR WEARABLE DEFIBRILLATOR IF LVEF ≤35%.
- AVOID PERMANENT DEVICES IF EARLY RECOVERY POSSIBLE.

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- AVOID PERMANENT DEVICES IF EARLY RECOVERY POSSIBLE.

PREGNANCY & DELIVERY CONSIDERATIONS

- •Timing: balance maternal status vs fetal prematurity.
- •Vaginal delivery preferred; C-section if obstetric indication or unstable mother.
- •Continuous monitoring (ECG, fluids, oxygen, BP).
- •Regional anaesthesia safe (avoid hemodynamic stress).
- •Third stage: oxytocin infusion preferred; avoid ergometrine/long-acting oxytocin.

BREASTFEEDING

- •Prolactin implicated in pathogenesis – theoretical concern.
- •Stable women (LVEF ≥45%): breastfeeding acceptable.
- •Discourage if symptomatic, unstable, or LVEF <45%.
- •Some drugs safe (enalapril, captopril), others contraindicated (spironolactone, carvedilol).
- •Decision should prioritise maternal recovery.

PROGNOSIS

- •50–80% recover LVEF within 6 months.
- •Mortality improved from 30–50% (1970s) to ~10% now.
- •Afro-Caribbean ethnicity → poorer recovery.
- •Associated hypertension → better recovery.
- •Predictors of poor outcome: LVEF <30%, LVEDD >6 cm.

SUBSEQUENT PREGNANCY

- •Risk of relapse, even if LVEF normalised (20%).
- •Persistent LV dysfunction → 50% chance of worsening, mortality up to 20%.
- •If initial LVEF <25% or incomplete recovery → advise against future pregnancy.
- •Requires pre-conception MDT counselling, close antenatal monitoring, serial echocardiograms.
- •LMWH may be considered during pregnancy if LV dysfunction persists.

Conclusion & Future Directions

- PPCM: a rare but serious disorder in young women.
- Early diagnosis and MDT involvement improve outcomes.
- Bromocriptine shows promise, but more PPCM-specific therapies are needed.
- Gaps remain: optimal treatment duration, biomarkers, long-term prognosis.
- More research and international registries are required.

CONTRACEPTION

- •Reliable contraception essential to avoid unplanned pregnancy.
- •Barrier methods not reliable enough.
- •Preferred: IUD (copper or hormonal), LARC with progestin.
- •Avoid estrogen-containing contraceptives (increase thromboembolic risk).
- •Sterilisation possible but requires careful counselling due to risks and permanence.

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.

Reference

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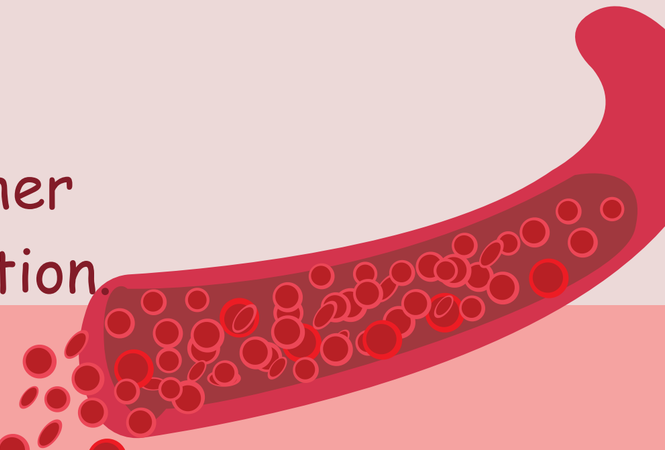
Acute coronary syndromes in pregnancy

Acute Coronary Syndrome (ACS) during pregnancy is rare but a significant cause of maternal mortality.

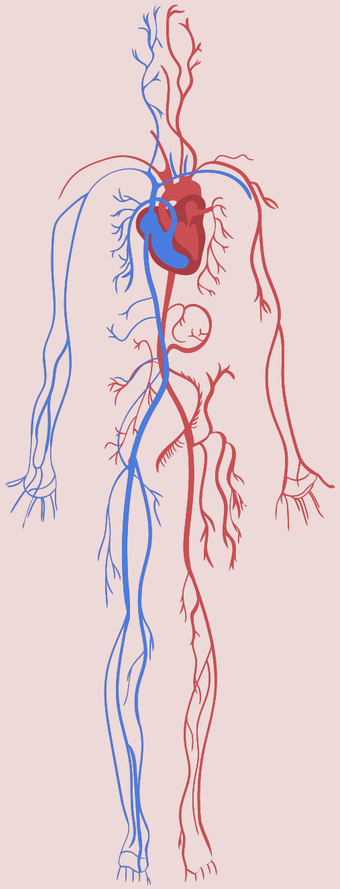
Need for early recognition: Prompt diagnosis and evidence-based multidisciplinary team (MDT) care (obstetricians, cardiologists, anaesthetists, midwives) are essential to improve maternal and fetal outcomes.

Increasing incidence: Largely due to rising maternal age and higher prevalence of cardiovascular risk factors in the pregnant population.



Epidemiology and risk factors

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).



An incidence of 6.2 per 100 000 deliveries, with a mortality rate between 5.1 and 11.0%

Cardiovascular disease was the leading cause of maternal deaths, accounting for 17% of all maternal deaths.

Ischaemic heart disease (IHD)was responsible for around one-fifth of these cases

pregnant women are three to four times more likely to suffer ACS than age-matched nonpregnant women, and the incidence in this population is rising.

Risk factors

Pregnancy-associated spontaneous coronary artery dissection (PASCAD)

occurs more commonly in women with connective tissue disorders such as Marfan syndrome, hypertension and a family history of spontaneous coronary artery dissection

Atherosclerosis:

Advanced maternal age, obesity, smoking, diabetes mellitus, hypertension, dyslipidaemia, personal or family history of ischaemic heart disease (IHD).

Risk factors

Pregnancy-specific prothrombotic factors:

Thrombophilia, hypertensive disorders, blood transfusion, infection, multiparity, sickle cell disease.

increased thrombosis risk associated with ACS

anemia, blood transfusion is probably a surrogate marker for ergometrine use, which may be more associated with spasm

Antenatal ACS is significantly more common in the **third trimester** than at earlier gestations

Can occur at any gestation; more common in the third trimester and postpartum.

Pathophysiology

Pregnancy-related physiological stress: 50% increase in blood volume and cardiac output, higher resting heart rate, dilutional anemia, fluctuating blood pressure and vascular resistance, hypercoagulable state, connective tissue softening

Around delivery, these parameters can shift rapidly. Oxygen consumption in labor increases, with potential subsequent massive blood volume loss. Pregnancy is a prothrombotic state with the risk of thromboembolism rising throughout the pregnancy and into the postpartum period. The woman's connective tissue weakens and stretches in preparation for birth, and this includes the vasculature .Such is the demand placed upon the maternal cardiovascular system that a small reduction in coronary flow can precipitate significant myocardial ischaemia

Types of ACS

ST-ELEVATION MYOCARDIAL INFARCTION (STEMI):

ST ELEVATION OR NEW LBBB WITH TYPICAL ACS SYMPTOMS; USUALLY HAEMODYNAMICALLY UNSTABLE; TIME TO REPERFUSION IS CRITICAL (GOAL <1 HOUR FROM ECG TO FLOW RESTORATION). TYPICALLY CHEST PAIN,SHORTNESS OF BREATH OR VENTRICULAR ARRHYTHMIA.

NON-ST-ELEVATION MYOCARDIAL INFARCTION (NSTEMI):

MORE STABLE THAN STEMI BUT CAN STILL BE UNSTABLE; ECG MAY SHOW ST DEPRESSION/T-WAVE INVERSION OR BE NORMAL; ELEVATED CARDIAC BIOMARKERS.

Unstable angina (UA):

chest pain or SOB at rest ,sometimes with the same ECG changes as NSTEMI but without a biomarker rise.

Stable angina here, although it is not classified as an acute coronary syndrome. Stable angina typically presents as chest pain or shortness of breath that is relieved by rest, with no biomarker rise and – in most cases –a normal ECG.

Aetiologies

The predominant subgroup has been PASCAD

- There is a of the coronary arterial wall as separation of the intimal lining from the outer vessel. Tearing of the intima allows formation of a false lumen, which propagates and compresses the true lumen, creating flow-limiting stenosis or obstruction. Blood flows into the false lumen, clotting and expanding, creating an intramural hematoma. This exerts pressure upon the true lumen, collapsing it and compromising flow. PASCAD usually occurs in the late third trimester and in the early postpartum period
- This timeline suggests an association with the increases in hemodynamic stress and fragility of the vasculature across this period

Aetiologies

Atherosclerosis

has now overtaken PASCAD as the mostprevalent aetiology, probably because of rising maternal ageand prevalence of relevant comorbid risk factors

Atherosclerotic plaquesusually form at areas of haemodynamic shear stress, wherethe endothelium is vulnerable to injury. Following vasculartrauma there is localised accumulation of lipid within theintima, recruitment and proliferation of both immune andsmooth muscle cell invasion with subsequent fibrousconnective tissue deposition. The soft core of lipid beneaththe fibrous cap enlarges, weakening the arterial wall andprecipitating rupture. Upon plaque rupture, blood is exposedto the lesion, leading to rapid platelet recruitment andthrombosis.

Aetiologies

- Thrombosis and thromboembolism within otherwise normal coronary vessels

- Coronary artery spasm:
Rare, can be medically managed

Clinical Presentation:

Typical symptoms: Chest or epigastric pain radiating to neck/arm.

Atypical symptoms: Non-specific pain, nausea, vomiting, back pain, dyspnea, sweating, agitation .Women are more likely than men to describe 'atypical' symptoms.

Differential diagnoses: Pulmonary embolism (PE), aortic dissection, pneumonia, gastro-esophageal reflux, musculoskeletal pain, anxiety

•. Examination may be unremarkable. Vital signs may indicate hemodynamic instability

Investigations

History & examination: May suggest ACS but often non-specific; vital signs may indicate haemodynamic instability

Specific investigations can provide greater clarity:

- **Electrocardiogram (ECG) :** the most familiar of cardiac investigations. Across a normal pregnancy untroubled by cardiac disease ,changes in the ECG can occur. This can include a reduction in the PR interval, sinus tachycardia and left axis deviation
  - There are multiple suggested mechanisms, including maternal organ displacement, changing myocardial conductive properties from higher levels of sympathetic hormonal modulation and left ventricular hypertrophy to support higher cardiacoutput.15 Pathological changes in keeping with acute ACS would include ST elevation, typically within a coronary territory with reciprocal ST depression, ST depression alone, T-wave inversion or new bundle branch block

Cardiac Biomarkers :

Troponin is a highly sensitive, but nonspecific marker for any form of myocardial injury; for example, from myocarditis, renal failure, sepsis, profound hypertension, cardiomyopathy, aortic dissection, PE and takotsubo, as well as pre-eclampsia ,cardiac decompensation and autoimmune diseases with cardiac involvement

Brain natriuretic peptide (BNP)
does not typically have a role in the diagnosis of ACS but may be useful in excluding other differentials, namely cardiac failure.

- **Echocardiogram (echo)** :
  Echo can provide a rapid, noninvasive assessment of cardiac structure and function. maternal physiological changes can be seen on echo. Typically, there is increases in left ventricular mass.

  may demonstrate regional wall motion abnormalities in a specific coronary territory also help to confirm or rule out other differential diagnoses such as PE and cardiomyopathy;

- **Chest X-ray** :There are no specific signs of ACS on chest X-ray (CXR), butthere may be signs of related left ventricular failuremay include pulmonary oedema, venous congestion, pleuraleffusions and septal lines

Echocardiograme

- Echo is a rapid, noninvasive tool to assess cardiac structure and function.

- Normal pregnancy changes include:
  - Increased left ventricular mass and end-diastolic volume
  - Enlarged left atrium
  - Higher flow velocities across valves due to increased cardiac output
  - Left ventricular ejection fraction remains normal

In ACS:

- Echo can show regional wall motion abnormalities in the affected coronary territory (The affected region typically appears less contractile than other unaffected regions)These abnormalities may correspond to ECG changes

- Echo can also help rule in/out other conditions like pulmonary embolism or cardiomyopathy
- normal ECHO doesn't exclude PE.

ETIOLOGY

RISK FACTORS

CLASSIFICATION

INVESTIGATION

TREATMENT

Angiography/Angioplasty

- Gold standard for emergent ACS assessment: cardiac catheterisation and angiography, often proceeding to angioplasty.

- Rapid arterial access allows imaging in ~5 minutes, with immediate intervention if needed.

- During pregnancy:

1. Fetal radiation is low (~0.075 mGy); maternal exposure is higher but usually does not affect clinical decisions.
2. ACS in pregnancy is life-threatening for both mother and fetus.
3. Radial access is preferred over femoral to minimize fetal radiation.
4. Iodinated contrast crosses the placenta but has not been linked to teratogenicity.
5. Lead shielding does not significantly reduce fetal radiation due to scatter exposure

ETIOLOGY

RISK FACTORS

CLASSIFICATION

INVESTIGATION

TREATMENT

Cardiac computed tomograpy

- A noninvasive method for detailed coronary artery assessment.
- Widely used in nonpregnant patients, but limited in emergencies due to:
  - Restricted availability
  - No option for immediate intervention
- Fetal radiation exposure: 1–3 mGy (relatively low)

(So, it is not the first-line choice in acute coronary syndrome).

Cardiac magnetic resonance imaging

- No role in emergency evaluation of pregnant patients.
- Useful for quantifying cardiac structure.
- May be applied in outpatient follow-up or in selected cases of acute myocardial disease.

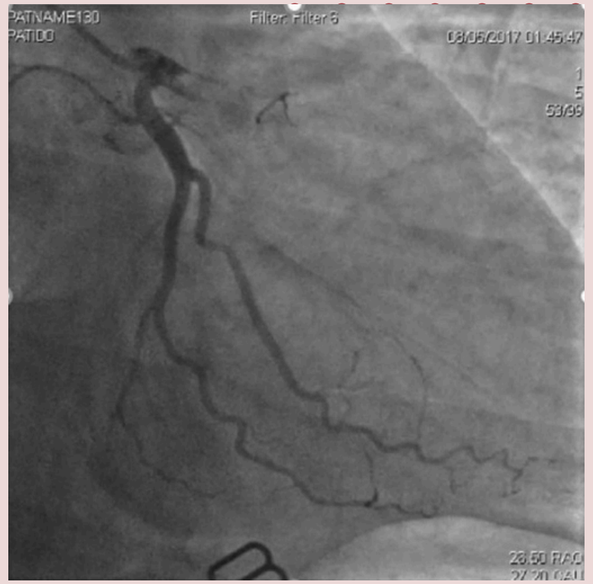
TREATMENT

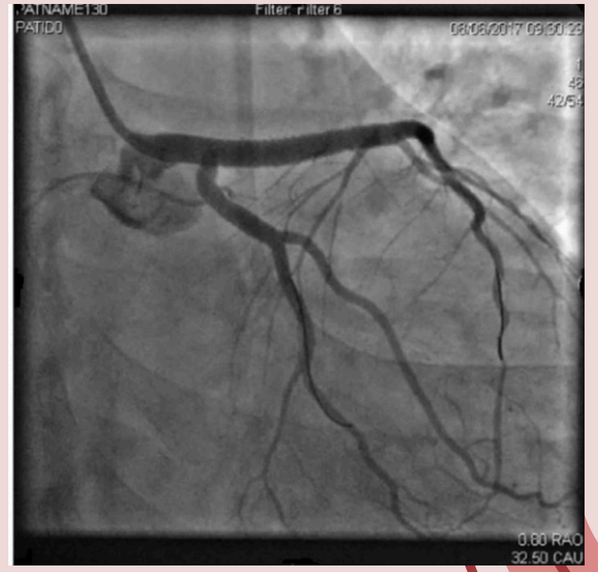
• Percutaneous intervention

■ Procedure Overview

- Diagnostic angiography identifies the obstructed coronary artery.
- Same vascular access is used for PCI—diagnostic catheter is exchanged for a larger guide catheter.
- Wire passage (proximal-to-distal) may restore flow by disrupting thrombus.

- <u>Treatment options:</u>
  - Balloon angioplasty ("monorail" technique).
  - Thrombus aspiration.
  - Local vasodilators/antiplatelet infusion.
  - Stent placement (to maintain patency).





TREATMENT

• Percutaneous intervention

■ Special Considerations in Pregnancy:

- Not all ACS subtypes need PCI (e.g., vasospasm/PASCAD may be managed medically).
- Higher risk of coronary artery dissection during PCI in pregnancy (caution needed, especially with PASCAD).
- Pregnancy is NOT a contraindication for PCI — lifesaving when indicated.

■ Timing of PCI:

- Immediate intervention if hemodynamically unstable.
- If stable, delay until after 4 months gestation (minimizes fetal radiation risk during organogenesis).
- Fetal radiation dose: ~0.0023–0.012 mGy/min during PCI.

TREATMENT

• Thrombolysis

1. Role & Risks of Thrombolysis

- Historical alternative to PCI, still used where PCI is unavailable.
- High bleeding risk: 3× greater than therapeutic anticoagulation.
- Intracranial hemorrhage NNH = 78 (significant concern).
- Pregnancy-specific risk : Major bleeding often from **genital tract (subplacental hemorrhage).

2. Fetal & Obstetric Risks

- 6% fetal loss rate (when used for pulmonary embolism).
- 6% preterm delivery rate.
- drugs do NOT cross placenta (streptokinase, rtPA are safe for fetus directly).

TREATMENT

• Thrombolysis

3. Contraindications in Pregnancy
- Recent surgery (e.g., cesarean delivery).
- No benefit in coronary vasospasm.
- PASCAD (Pregnancy-Associated SCAD): Thrombolysis may extend dissection → vessel rupture → cardiac tamponade.

**(PCI is preferred for ACS in pregnancy)

TREATMENT

- **Pharmacotherapy**

Pregnancy significantly alters drug metabolism and pharmacokinetics, requiring careful medication selection. Cardiovascular drugs fall into three categories: those crossing the placenta and harming the fetus (e.g., ACE inhibitors), those crossing safely (e.g., some beta-blockers), and those not crossing (e.g., heparin). Similar considerations apply during breastfeeding, as some drugs pass into milk while others don't. Many medications lack sufficient safety data, necessitating careful risk-benefit analysis when treatment is essential.

• Pharmacotherapy

Acute Medications & Stabilization in Pregnancy (cardiac event):

1. Priorities

Immediate stabilization of the woman.

Rapid assessment with senior cardiology + obstetrics team involvement.

Consider how timing of delivery affects management.

2. Antiplatelet & Anticoagulation

Aspirin: loading dose 300 mg → then 75 mg daily.

Heparin (therapeutic SC): continued until day before angiography.(If angiography is immediate → hold SC heparin, use IV heparin during procedure).

Clopidogrel: emerging evidence of safety in pregnancy.

Loading dose 600 mg → then 75 mg daily.

TREATMENT

• Pharmacotherapy

Acute Medications & Stabilization in Pregnancy (cardiac event):

3. Symptom Relief

Chest pain → appropriate analgesics.

Nitrates may be used, but can reduce uterine perfusion (caution).

4. If Hemodynamic Instability (despite revascularization):

Consider inotropes.

Consider mechanical circulatory support.

TREATMENT

- **Pharmacotherapy**

Chronic thyerapy

Aspirin (Safe in pregnancy)
Clopidogrel (Recent review → no excess teratogenic).

Secondary Prevention
Beta-blockers
Considered safe, but may cause fetal growth restriction & neonatal hypoglycemia.
Labetalol → widely used for hypertension, good safety data.
Bisoprolol → more cardioselective, preferred in IHD.
ACE inhibitors / ARBs / ARNIs
Contraindicated in pregnancy (teratogenic).
ACE inhibitors safe in breastfeeding, but ARBs & ARNIs should be avoided.

TREATMENT

- **Pharmacotherapy**

Chronic thyerapy

Diuretics

Furosemide → safe in pregnancy & breastfeeding, but use cautiously.

Thiazides & spironolactone → not safe in pregnancy, but safe in breastfeeding.

Statins → avoid (not proven safe).

Anticoagulation

LMWH & UFH → safe in pregnancy & breastfeeding, but risky around delivery → need bridging & possible reversal (protamine for UFH, partial effect in LMWH).

DOACs → insufficient safety data, not recommended.

Warfarin → teratogenic in pregnancy, but safe in breastfeeding.a. pregnancy (teratogenic).

TREATMENT

- **Delivery**

Timing of Delivery

Immediate delivery is rarely indicated (except in extreme cases).
Preferably delay for several weeks after acute event to allow maternal recovery.
Delivery not required before PCI.
Usually recommended before 40 weeks gestation.

Mode of Delivery

Determined mainly by obstetric factors.
Induction of labour is safe.
Prostaglandin E analogues (prostin, propess) and oxytocin → can be used.(oxytocin give iv slowly not bolus)
Avoid prostaglandin F analogues (carboprost) & ergometrine

TREATMENT

- **Delivery**

Labour Management

In 2nd stage: allow up to 2 hours passive descent before active pushing (reduces maternal strain).

Early instrumental delivery not routinely recommended unless obstetric indication.

Emergency Situations

If severe cardiopulmonary shock or cardiac arrest:

Perform CPR with left lateral tilt (relieve aortocaval compression).

If >24 weeks gestation → perimortem C-section if no circulation within 4 minutes.

Immediate neonatal support required.

TREATMENT

• Future pregnancy

Pre-pregnancy Care

- Risk of recurrence of ACS: ~9% in subsequent pregnancies.
- Delay conception: At least 12 months after ACS, even if no residual dysfunction.
- Counselling: Pre-pregnancy counselling and optimisation of cardiac function are essential.
- Medication review: Teratogenic drugs should be switched to safe alternatives.

TREATMENT

- **Future pregnancy**

Contraception:
Must be effective for those not planning pregnancy or still optimising health.

Some contraceptive methods are contraindicated in IHD.

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

TREATMENT

• Future pregnancy

Pregnancy Management

Care should be in a tertiary unit with MDT involvement:
Obstetrician with obstetric medicine expertise.
Cardiologist experienced in pregnancy.
Obstetric anaesthetist.

Risk assessment:

Use mWHO classification.
Women with mWHO III–IV: very high risk → counsel against pregnancy, discuss termination (TOP) if already pregnant.
If TOP chosen: surgical route may be safer (lower failure risk).
Investigations: ECG, echo, biomarkers, cardiac CT, or CMR.
Modifiable risk factors: Must address obesity, smoking, alcohol, diabetes, hypertension.

TREATMENT

Future pregnancy

Antenatal Care

- Close monitoring with regular MDT visits.
- No universal guideline on repeat cardiac investigations in pregnancy, but:
- Any new/worsening symptoms → urgent specialist assessment.
- Medication review at pre-conception or first antenatal visit.
- Women should not stop cardiac drugs abruptly without specialist advice.

“هل ما زال أحد يذكرني ؟!“

اللهم فكّ أسر أسرانا
وتقبّل شهداءنا
لا حول لنا ولا قوة إلا بك!