Cardiac Disease in Pregnancy

Supervised by:

Dr. Malek Alqasim

Introduced by:

Mohammad Al-Shloul

Shaimaa Almoanis

Hala Al mahasneh

Yosor Al Ajaleen

Introduction

Cardiovascular adaptation to pregnancy

- The primary event is probably peripheral vasodilatation => This is mediated by endothelium-dependent factors, including nitric oxide synthesis upregulated by estradiol and possibly vasodilatory prostaglandins
- Peripheral vasodilation leads to a fall in systemic vascular resistance (SVR) and to compensate for this, the cardiac output increases by around 40% during pregnancy => This is achieved predominantly by an increase in stroke volume but also by a lesser increase in heart rate

CONT....

- ■These changes begin early in pregnancy and by 8 weeks' gestation the cardiac output has already increased by 20%

■The maximum cardiac output is found at about 20–28 weeks' gestation => There is a minimal fall at term

■Although stroke volume declines towards term, the increase in maternal heart rate (10–20 beats per minute [bpm]) is maintained, thus preserving the increased cardiac output

■There is a profound effect of maternal position towards term upon the hemodynamic profile of both the mother and fetus

■In the supine position, pressure of the gravid uterus on the inferior vena cava (IVC) causes a reduction in venous return to the heart and a consequent fall in stroke volume and cardiac output

CONT....

- ■Turning from the lateral to the supine position may result in a 25% reduction in cardiac output => Pregnant women should, therefore, be nursed in the left or right lateral position wherever possible If the woman has to be kept on her back, the pelvis should be rotated so that the uterus drops to the side and off the IVC and cardiac output and uteroplacental blood flow are optimized
- ■Reduced cardiac output is associated with a reduction in uterine blood flow and therefore in placental perfusion; this can compromise the fetus

CVS Changes During Pregnancy

PARAMETER	DIRECTION	TIME COURSE
Heart rate	↑	1st and 2nd trimester (TM)
Blood pressure	↓	Fall in TM 1 and 2, returns to baseline in 3
Cardiac output	↑	45% above baseline by TM 3
Stroke Volume	↑	Peak at weeks 16 to 24
Systemic vascular resistance	↓	Nadir by mid pregnancy
Pulmonary vascular resistance	↓	20- 30% decrease

Pregnancy counselling

- Most women with heart disease will be aware of their condition prior to becoming pregnant
- Ideally, these women should be fully assessed by an obstetrician and cardiologist before embarking on a pregnancy and the maternal and fetal risks carefully explained.
- A plan to optimize medication should be made .
- if there is a possibility that the heart disease will require surgical correction it is recommended that this should be undertaken before a pregnancy

counselling of women with heart disease

- Risk of maternal death .
- Possible reduction of maternal life expectancy .
- Effects of pregnancy on cardiac disease .
- Mortality associated with high risk conditions .
- Risk of fetus developing congenital heart disease .
- Risk of preterm labour and FGR .
- Need for frequent hospital attendance and possible admission .
- Intensive maternal and fetal monitoring during labour .
- Other options – contraception , adoption , surrogacy .
- Timing of pregnancy .

Antenatal management

- •Experienced physicians and obstetricians should manage pregnant women with significant heart disease in a joint obstetric/cardiac clinic.
- •Continuity of care makes the detection of subtle changes in maternal wellbeing more likely.
- •In trying to distinguish between ‘normal’ symptoms of pregnancy and impending cardiac failure, it is important to ask the pregnant woman if she has noted any breathlessness, particularly at night, any change in her heart rate or rhythm, any increased tiredness or a reduction in exercise tolerance

- Routine physical examination should include pulse rate, blood pressure, jugular venous pressure, heart sounds, ankle and sacral oedema and presence of basal crepitations.
- Most women will remain well during the antenatal period and outpatient management is usually possible, although women should be advised to have a low threshold for reducing their normal physical activities .
- Echocardiography is non-invasive and useful in its ability to serially assess function and valves, and an echocardiogram at the booking visit and at around 28 weeks' gestation is usual. Any signs of deteriorating cardiac status should be carefully investigated and Treated Anticoagulation is essential in patients with congenital heart disease who have pulmonary hypertension (PH) or artificial valve replacements, and in those in or at risk of atrial fibrillation. The use of anticoagulants during pregnancy is a complicated issue because warfarin is teratogenic if used in the first trimester, and is linked with fetal
- intracranial haemorrhage in the third trimester. Low- molecular-weight heparin is often used as an alternative to warfarin, especially in the first and third trimester, and can be titrated using factor Xa levels

- High risk cardiac condition

Systemic ventricular dysfunction (ejection fraction <30%, NYHA Class III– IV).

- Pulmonary hypertension.
- Cyanotic congenital heart disease.
- Aortic pathology (dilated aortic root >4 cm,
- Marfan syndrome)
- Ischemic heart disease.
- Left heart obstructive lesions (aortic, mitral stenosis)
- Prosthetic heart valves (metal).
- Previous peripartum cardiomyopathy

fetal risk of maternal cardiac disease

Recurrence (congenital heart disease)

- Maternal cyanosis (fetal hypoxia)
- Iatrogenic prematurity.
- FGR.
- Effects of maternal drugs(teratogenesis, growth restriction, fetal loss)

Heart Failure

- Definition: It is the pathophysiological process in which the heart as a pump is unable to meet the metabolic requirements of the tissue for oxygen and substrates despite the venous return to heart is either normal or increased
- It can occur at any age, but is most common in older people.
- Heart failure is a long-term condition that tends to get gradually worse over time => It cannot usually be cured, but the symptoms can often be controlled for many years

Symptoms

- Fatigue: there isn’t enough oxygen-rich blood to meet the body's energy needs

- Activity limitation: patients become easily tired and short of breath

- Congestion: Fluid buildup in the lungs => coughing, wheezing, and breathing difficulty

- Edema or ankle swelling: Excess fluid can also cause rapid weight gain

- Shortness of breath: Fluid in the lungs makes it more difficult for carbon dioxide to be exchanged for oxygen especially in supine position because gravity allows fluid from below the lungs to travel up the torso

Etiology

- IHD 62%
- Valvular Heart Disease 10%
- Hypertension 4%
- Atrial Fibrillation 3%
- Idiopathic and undetermined (no IHD or angiographic data) 10%

Risk factors for the development of heart failure in pregnancy

- Respiratory or urinary infections.
- Anaemia.
- Obesity.
- Corticosteroids.
- Tocolytics.
- Multiple gestation.
- Hypertension.
- Arrhythmias.
- Pain-related stress.
- Fluid overload.

New York Heart Association classification

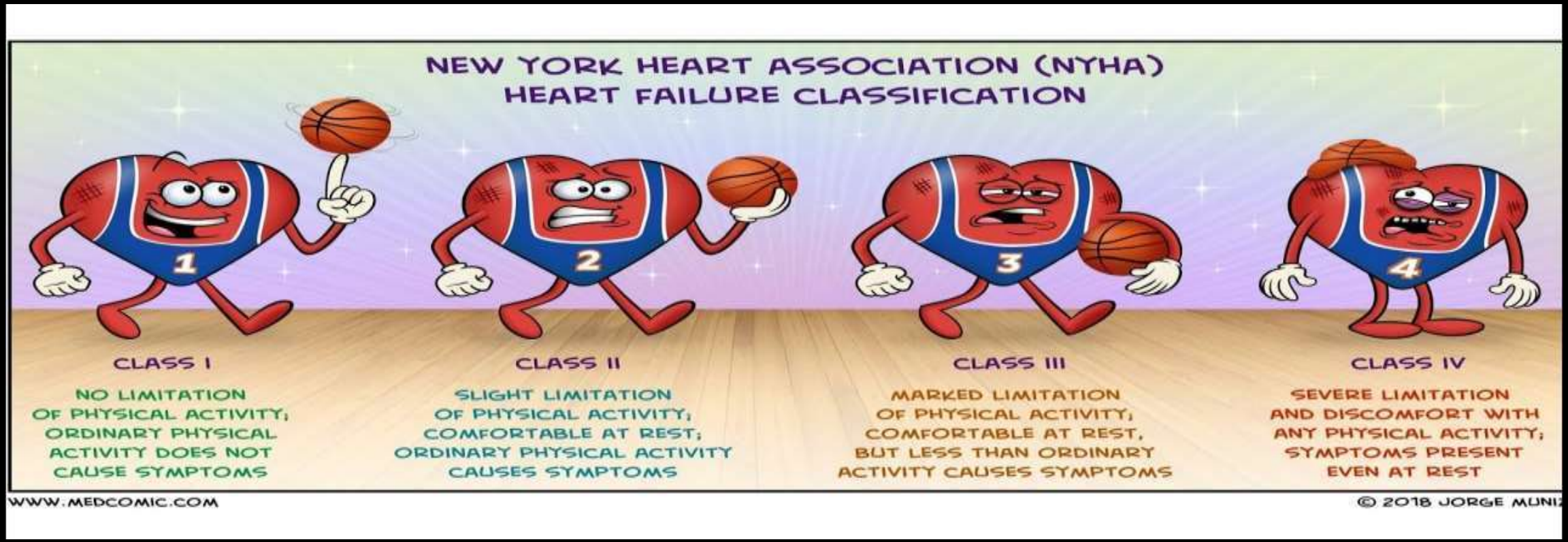


Table 12.2 Toronto risk markers for maternal cardiac events

	Markers
1	Prior episode of heart failure, arrhythmia or stroke
2	NYHA class >II or cyanosis
3	Left heart obstruction
4	Reduced left ventricular function (EF <40 per cent)

0 predictors: risk of cardiac event is 5 per cent; 1 predictor: risk of cardiac event is 37 per cent; >1 predictors: risk of cardiac event is 75 per cent.

Treatment of heart failure in pregnancy

- the principles of treatment are the same as in the non-pregnant individual => The patient should be admitted and the diagnosis confirmed by clinical examination and by echocardiography confirming ventricular dysfunction

- Drug therapy may include : Diuretics, Vasodilators + digoxin +/- Oxygen and morphine
- Arrhythmias also require urgent correction and drug therapy; for example, adenosin

Management

1- antenatal care :

- for fetus :

assessment of fetal wellbeing (fetal growth + regular (CTG)) , If there is evidence of fetal compromise, premature delivery may be considered

- for mother—>

Management of labour in women with heart disease

- Avoid induction of labour if possible.
- Use prophylactic antibiotics.
- Ensure fluid balance.
- Avoid the supine position.
- Discuss regional/epidural anaesthesia/analgesia with senior anaesthetist.
- Keep the second stage short.
- Use Syntocinon judiciously.

2- during labour

First stage

Mostly the aim is onset of normal labour (minimize the risk of intervention)+ maximize the chances of a normal delivery

- • Anesthesia: Epidural is often recommended BUT has a risk of maternal hypotension => senior anesthetist must have a management plan
- Prophylactic antibiotics should be given to any woman with a structural heart defect => reduce the risk of bacterial endocarditis

(amoxycillin 2 g i.v. + gentamicin 1.5 mg/kg i.v)
(if penicillin allergic give vancomycin 1g i.v.)

Second stage

Should be kept short , with an elective forceps or ventouse delivery
(if normal delivery does not occur readily) =>
This reduces maternal effort and the requirement for increased cardiac output

Third stage

active management of the third stage is usually with Syntocinon TM only
(a vasodilator and therefore should be given slowly , with low-dose infusions preferable)

- Don’t give Ergometrine since it may be associated with intense VC , HTN & HF

ONLY do Caesarean delivery if the mother is too unstable to tolerate the physiological demands of labor, because CS is associated with an increased risk of: Hemorrhage, Thrombosis and Infection = > these conditions are less well tolerated in women with cardiac disease

Valvular Heart Diseases

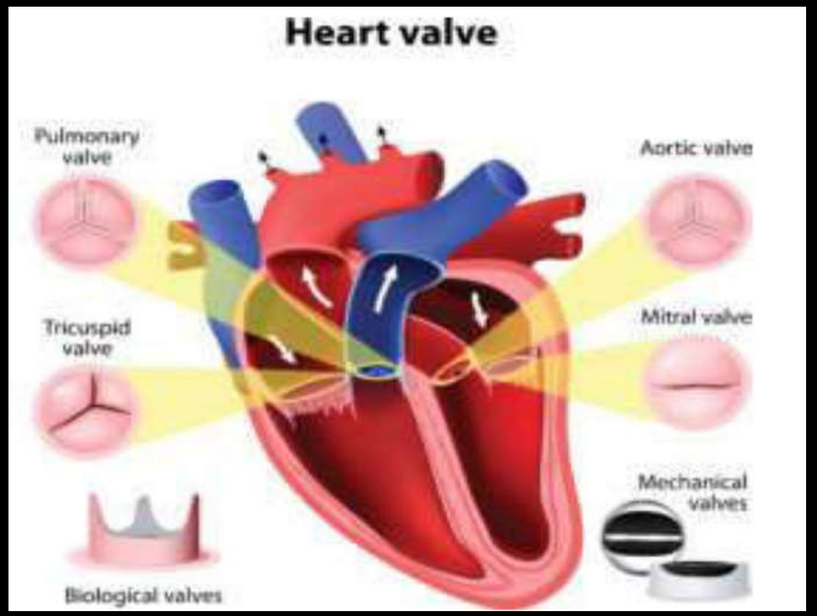
Mitral stenosis

Rheumatic endocarditis causes most mitral stenosis lesions.

- more severe stenosis —> left atrium dilates —> pressure chronically elevated, and significant passive pulmonary hypertension develops.
- Also in stenosis, tachycardia shortens ventricular diastolic filling time and elevates the mitral gradient which lead to more pulmonary edema.
- During pregnancy, there is a progressive increase in blood volume, heart rate and cardiac output which reach their peak levels at 28-32 weeks of gestation.
- MS represents a **fixed obstruction** to left ventricular inflow, the haemodynamic changes of pregnancy usually cause **decompensation.**
- **according to severity** , Patients with mild MS usually tolerate pregnancy and delivery well.

while patients with asymptomatic moderate or severe MS develop symptoms of heart failure, especially in the 2nd trimester of pregnancy , and should be corrected before considering pregnancy & have high rates of pre- term delivery and intrauterine growth restriction.

- They may need correction of their MS during pregnancy, either by **<u>surgical or by percutaneous intervention .</u>**
- most suitable timing for intervention is after the 4th month of pregnancy .



- The most critical clinical aspect of MS patients is the heart rate.

Rapid heart rate shortens diastolic filling time, increases the left atrial pressure and the pulmonary venous pressure and causes heart failure symptoms , That is why controlling the rapid HR greatly improves the symptoms.

- Patients with MS are dependent on atrial contraction. That is why development of atrial fibrillation (AF) is problematic.

ECG

Echocardiography is safe and is needed for diagnosis and follow-up of MS during pregnancy .

• ECG provides information regarding :
1- the area of the mitral valve
2- the size of the left atrium
3- the size and function of the left ventricle and right-sided chambers.

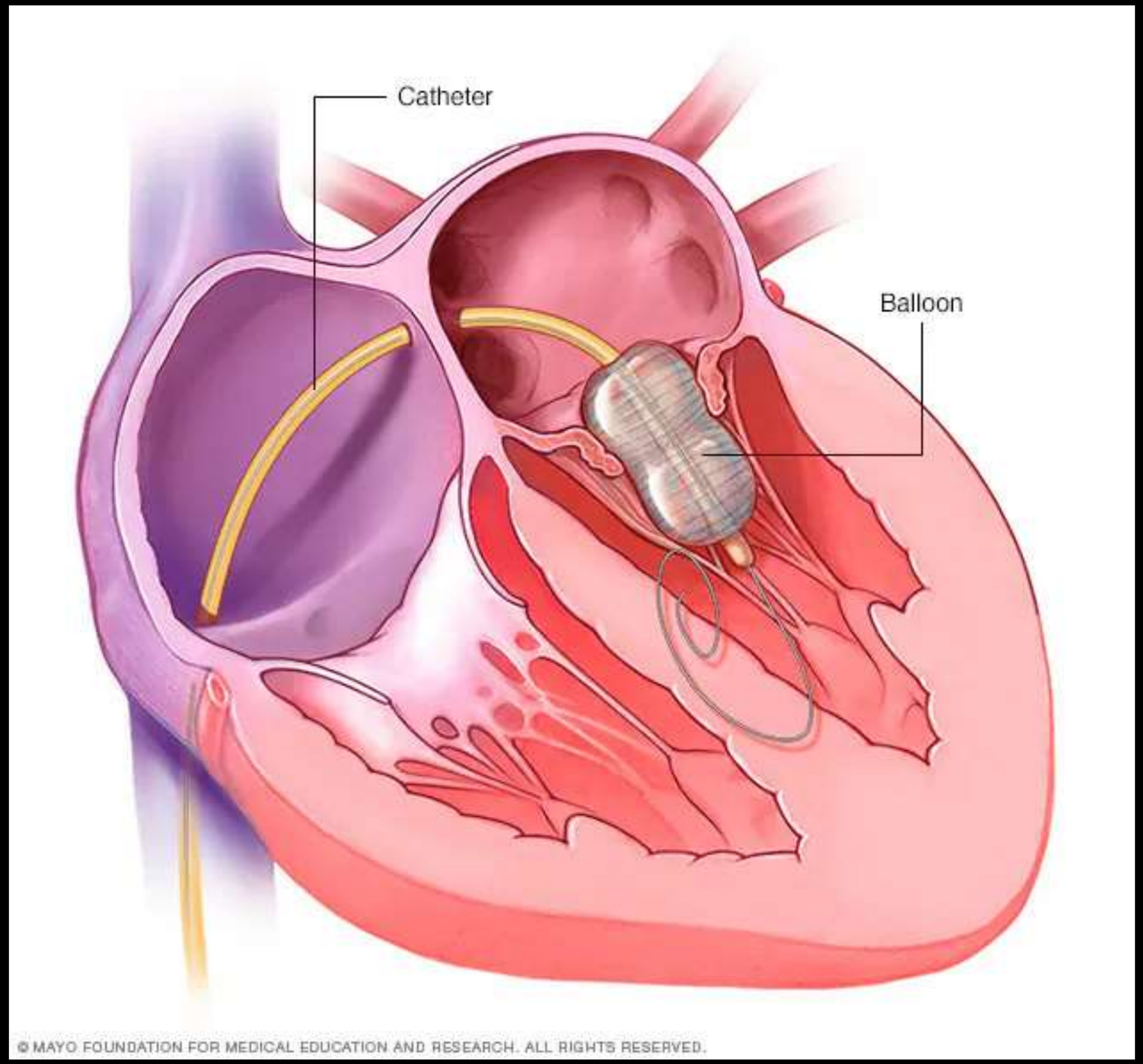
Pregnancy outcomes

- complications are directly associated with the degree of valvular stenosis .
- Woman with mitral valve less than 2 cm2 are at greatest risk.
- Woman with mitral valve less than 1 cm2 increase the risk of fetal growth restriction.

- Prognosis is also related to maternal functional capacity. Among 486 pregnancies complicated by rheumatic heart disease —predominantly mitral stenosis— 8 out of 10 maternal deaths were in women in NYHA classes III or IV.

Management

- 1. Activity Restriction: Limit physical activity; reduce further if symptoms worsen. dietary sodium is restricted , Use diuretics for pulmonary congestion.
- 2. Medication: Beta-blockers to control heart rate. Anticoagulation for atrial fibrillation, left atrial thrombus, or history of embolism.
- 3. Surgical Intervention: Balloon valvuloplasty is preferred for severe symptomatic cases (avoid in the first trimester).
- 4. Delivery Plan: Vaginal delivery is preferred with regional anesthesia. Cesarean is reserved for obstetric reasons or decompensated heart failure.



Aortic stenosis

In women of childbearing age, AS is most commonly due to a congenital bicuspid aortic valve, which may be associated with an aortopathy or coarctation of the aorta.
Adverse maternal and fetal outcomes increase with the severity of AS.

- The risk of hemodynamic compromise and HF is highest during :

1- the second to third trimester
2- during labor and delivery
3- and 24-72 hours after delivery as the cardiac output peaks.

- Although pregnancy-related mortality is low, and reported at zero in some studies
- women with severe AS are more likely to develop HF and atrial arrhythmias and have adverse fetal outcomes such as preterm birth and low birth weight.

Management

- **Asymptomatic Women**: No treatment needed; only close observation.
- **Symptomatic Women**: Activity limitation and diuretics are recommended.
- **Persistent Symptoms**: Consider surgical intervention if symptoms do not improve with rest.
- **Severe Early Symptoms**: Pregnancy termination may be considered.
- **Surgical Risks**: Valve replacement carries a high risk of fetal demise due to cardiac bypass complications.

  - The aortic valve can again narrow, or new aortic regurgitation may develop. The alternative surgical approach—valve replacement—is associated with significant risk of fetal demise because of cardiac bypass.

Delivery

- **Preferred Delivery Method**: Vaginal delivery is preferred for women with moderate to severe aortic stenosis, using assisted second stage if needed.
- **Cesarean Delivery:** May be considered for patients with severe symptoms.
- **Anesthesia**: Regional anesthesia with an epidural is recommended for effective pain control during labor.

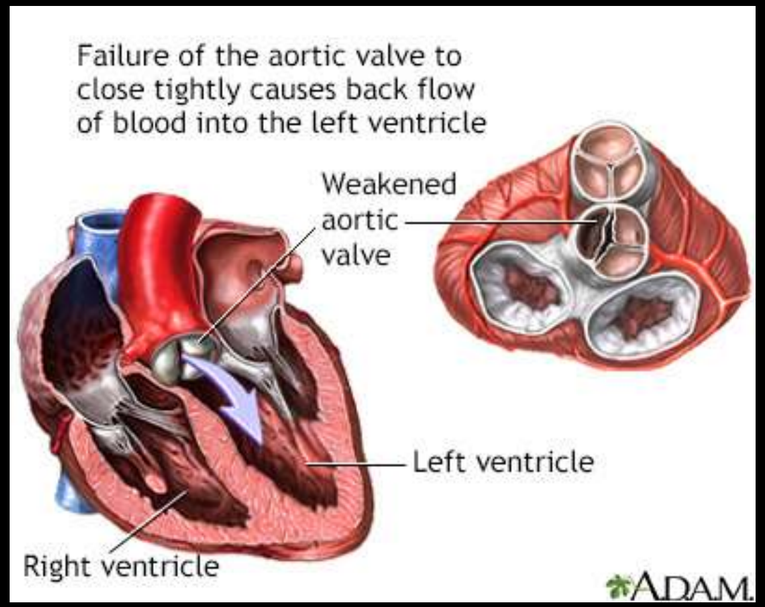
Aortic insufficiency

• Aortic valve regurgitation or insufficiency allows diastolic flow of blood from aorta back into left ventricle.

• Causes : (rheumatic fever, connective tissue abnormalities, congenital lesions and Marfan syndrome)

• generally well tolerated during pregnancy . Diminished vascular resistance is thought to improve hemodynamic function.

• If symptoms of heart failure develop —> diuretics are given, and bed rest is encouraged.

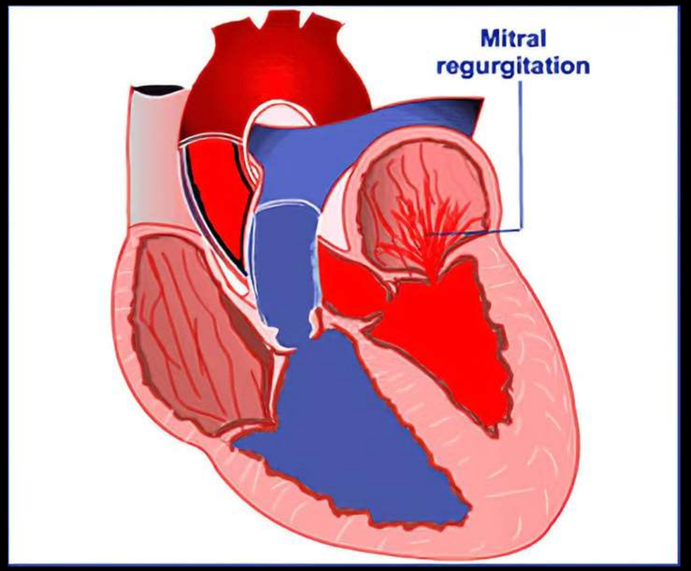


Mitral insufficiency

• In nonpregnant patients, symptoms from mitral valve insufficiency are rare.

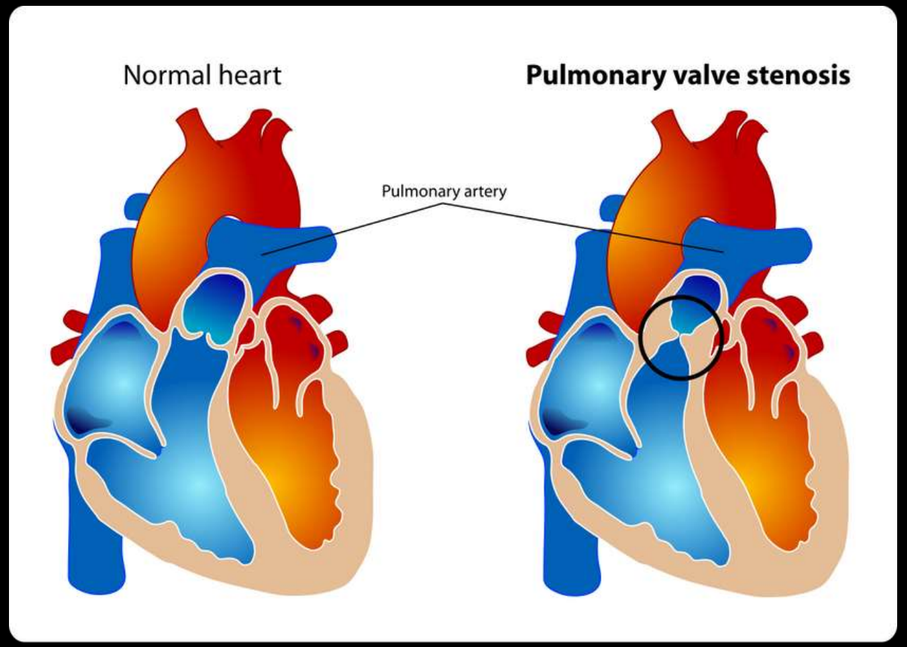
• During pregnancy, MR is similarly well tolerated, probably because the lowered systemic vascular resistance yields less regurgitation.

• Heart failure rarely develops during pregnancy, and occasionally tachyarrhythmias or severely depressed systolic function require treatment.



Pulmonic Stenosis

- This lesion is usually congenital and may be associated with Fallot tetralogy or Noonan syndrome.
- Surgical correction ideally is done before pregnancy, but if symptoms progress, a balloon valvuloplasty may be necessary antepartum.



Patient with prosthetic valve

• **High Risk of Complications**: Pregnant women with mechanical heart valves are at increased risk for thromboembolism, bleeding, and prosthetic valve endocarditis.

• **Prothrombotic State**: Pregnancy induces a hypercoagulable state, marked by elevated clotting factors and reduced protein S activity, lasting 6 to 12 weeks postpartum.

• **Maternal and Fetal Risks**: These women face higher rates of cardiovascular events, obstetric complications (hemorrhage, preterm birth), and fetal issues (growth restriction, miscarriage, stillbirth).

• **Thrombotic Incidence:** There is a 45% rate of thrombotic episodes during pregnancy, with a maternal mortality rate of 1-4%.

- **Anticoagulation**: Effective anticoagulation is essential, necessitating careful risk management for both mother and fetus with various treatment options.

Patient with prosthetic valve

Regime	Maternal thromboembolism (%)	Fetal abnormality (%)	Fetal loss (%)
Warfarin until 38 weeks, followed by elective LSCS	4	6	30
Heparin for 6–12 weeks, followed by warfarin to 38 weeks, followed by LSCS	9	0	30
Heparin throughout pregnancy	25	0	30

LSCS = lower segment caesarean section

Ischemic Heart Disease

🔻Acute myocardial infarction is rare during pregnancy and postpartum , 1 in 10000 pregnancies

🔻The risk of acute myocardial infarction is approximately threefold higher in pregnant women compared with nonpregnant women of similar age.

- Maternal mortality rates of 37–50% have been reported, with most deaths occurring at the time of infarction , Women who sustain an infarction less than 2 weeks before delivery are at especially high risk of death due to the greater myocardial demand of labor and delivery.

- Pregnancy itself has not been identified as a risk factor for acute myocardial infarction.

- However, the increased blood volume, altered haemodynamics and increase in estrogen and progesterone levels are likely to be risk factors for CVD

🔻 So the risk factors for ischemic heart disease in pregnancy are similar to those seen in nonpregnant patients. These include age(>40 yr old), family history of premature CAD, diabetes mellitus, hypertension, dyslipidemia, tobacco use, obesity, and physical inactivity , timing (peak incidence is in the third trimester, in parous women older than 35 years).

🔻 Diagnosis

- Clinical presentation
- ECG changes
- Evidence of myocardial necrosis reflected by elevated serum troponin levels .
- Coronary angiography is considered the diagnostic gold standard and should be expediently performed if acute coronary syndrome—defined as myocardial infarction or unstable angina—is present.

🔻 Treatment of myocardial infarction during pregnancy is the same as that outside of pregnancy; with heparin, beta-blockers and nitrates, Coronary angiography is safe in pregnancy and percutaneous catheter intervention is used as the first-line treatment.

Thrombolysis can cause bleeding from the placental site but is still indicated in the management of acute myocardial infarction

percutaneous transluminal coronary angioplasty (PTCA)
• Although the past fetal and maternal mortality rates, it still used but only when absolutely necessary, avoiding the time when the fetus is most susceptible to radiation (8-15 weeks).

*Statins should be discontinued prior to pregnancy

Arrhythmia

Arrhythmias

- Pregnancy increases the incidence of cardiac arrhythmia. This is the result of:
  - hormonal changes
  - alterations in autonomic tone
  - increased haemodynamic demands
  - mild hypokalemia.

- These factors act to precipitate cardiac arrhythmias not present before pregnancy or to exacerbate preexisting arrhythmias. The risk is highest during labour and delivery

▼ **Atrial and ventricular premature beats** are frequently present during pregnancy. They have no adverse effects on the mother or fetus and require no further investigation.

▼ **Atrial fibrillation and atrial flutter** are rare and can be caused by preexisting congenital or valvular heart disease, thyrotoxicosis or electrolyte imbalance.

▼ **SVT**----- risk of thromboembolism ,due to this risk and the potential detrimental effect on the fetus, early treatment, either with conversion to sinus rhythm or ventricular rate control, is important .

Other causes of SVT encountered in pregnancy are re-entrant tachycardias; for example:

Wolff-Parkinson-White syndrome

Lown-Ganong-Levine syndrome.

Initial treatment in the haemodynamically stable woman to terminate an SVT should involve the vagal maneuver. If this fails, intravenous adenosine can be used safely.

Second-line treatments include digoxin, beta-blockers and calcium channel blockers.

🔻 Ventricular tachycardia ---- must be treated -- risk of heart failure

- Ventricular tachycardia is uncommon in pregnancy. It is usually associated with underlying heart disease but new onset ventricular tachycardia without structural heart disease has been reported.

- Initial therapy with lidocaine or procainamide should be considered in haemodynamically stable women.

- Amiodarone is contraindicated, as it is associated with fetal hypothyroidism, growth restriction and prematurity. Beta-blockers and sotalol are used prophylactically

- Electrical cardioversion is safe in pregnancy and necessary in all women with tachyarrhythmias who are haemodynamically unstable

Congenital Heart Diseases

• During pregnancy, women with congenital heart disease are at increased risk of cardiac events including pulmonary edema and arrhythmias.

• Risk factors include prior history of heart failure, NYHA class III, decreased subpulmonary ventricular EF, severe pulmonary regurgitation, and smoking.

• These women also face increased risks of adverse neonatal outcomes including preterm delivery and infants with growth restriction, respiratory distress syndrome, and intraventricular hemorrhage, additionally, there is an increased incidence of congenital heart disease in children of women with a congenital abnormality.

Minimal risk Defects

- include small ventricular septal defects (VSDs), atrial septal defects (ASDs), Coarctation of the aorta, Tetralogy of Fallot.

- These patients have near-normal physiology with only minimally increased risk during pregnancy and can receive routine care.

Moderate risk Defects

- include transposition of the great arteries, cyanotic heart disease without pulmonary hypertension.

High-risk Defects

- for which patients should be counseled against pregnancy due to the risk of maternal cardiac decompensation and death include, Marfan syndrome, pulmonary vascular disease.

Minimal risk defects

- In pregnancy, increased cardiac output and blood volume are counterbalanced by a decrease in peripheral vascular resistance.
- Left-to-right shunting in women with atrial septal defects (ASDs), ventricular septal defects and PDA is, therefore, reduced.
- In the absence of pulmonary hypertension, pregnancy, labour and delivery are well tolerated.
- Women with ASDs are at risk of atrial arrhythmias and paradoxical emboli.
- As such, there is a low threshold for heparin prophylaxis.

- **Septal defects:** **Young women with uncomplicated secundum-type ASD or isolated VSD usually tolerate pregnancy well**. ASD is the most common congenital heart lesion in adults.

- **ASDs are usually very well tolerated unless they are associated with pulmonary HTN.**

- Complications, such as atrial arrhythmias, pulmonary HTN, and heart failure, usually do not arise until the fifth decade of life and are therefore uncommon in pregnancy.

- VSDs usually close spontaneously or are closed surgically if the lesion is large. For this reason, **significant VSDs are rarely seen in pregnancy**.

- Rarely, uncorrected lesions lead to significant left to right shunts with pulmonary HTN, right ventricular failure, arrhythmias, and reversal of the shunt.

- The incidence of VSD in the offspring of affected parents is 4%; however, small VSDs are often difficult to detect antenatally.

- **Patent ductus arteriosus (PDA):** PDA is not associated with additional maternal risk for cardiac complications if the shunt is small to moderate and if pulmonary artery pressures are normal.

- Moderate to large PDA may be associated with increased volume, left heart failure, and pulmonary HTN or other pulmonary abnormalities.

- Therefore, **pregnancy is not recommended for patients with large PDA and associated complications.**

- **Coarctation of the aorta**: Severe cases of coarctation of the aorta are usually corrected in infancy.

- Surgical correction during pregnancy is recommended only if dissection occurs.

- **Some studies suggest that patients with a history of coarctation have increased rates of preeclampsia, gestational HTN, and preterm labor.**

- Two percent of infants of mothers with coarctation of the aorta may have other cardiac lesions.

- Coarctation of the aorta is characterized by a fixed cardiac output. **Therefore, the patient's heart cannot increase its rate to meet the increased cardiac demands of pregnancy, and extreme care must be taken to prevent hypotension and IUG**

- **Tetralogy of Fallot,** characterized by right ventricular outflow tract obstruction, VSD, right ventricular hypertrophy, and overriding aorta, is associated with right- to-left shunting and cyanosis

- If the defect goes uncorrected, the affected patient rarely lives beyond childhood.

- In developed countries, almost all patients have had surgical correction with good survival rates (85% to 86% at 32 to 36 years) and good quality of life.

- **Pregnancy is generally well tolerated in patients who have had surgical repair, although these women are at increased risk of right-sided heart failure and arrhythmia.**

Moderate risk defects

Transposition of the great arteries

- Transposition of the great arteries is simply that the aorta and pulmonary arteries Has switched places with each other which means oxygen rich blood will be pumped through the pulmonary artery back to the lung and oxygen poor blood would be pumped through the aorta back to the body.

- In such cases there is atrial septal defect or patent foramen Oval that will keep the fetus alive for a couple of weeks.

- Surgery must be done to correct the misplacement.

- Women who have had a good repair are going to tolerate pregnancy well But women who have had complications would be at higher risk in a pregnancy.

High risk defects

Eisenmenger syndrome: occurs when an initial left-to-right shunt results* •

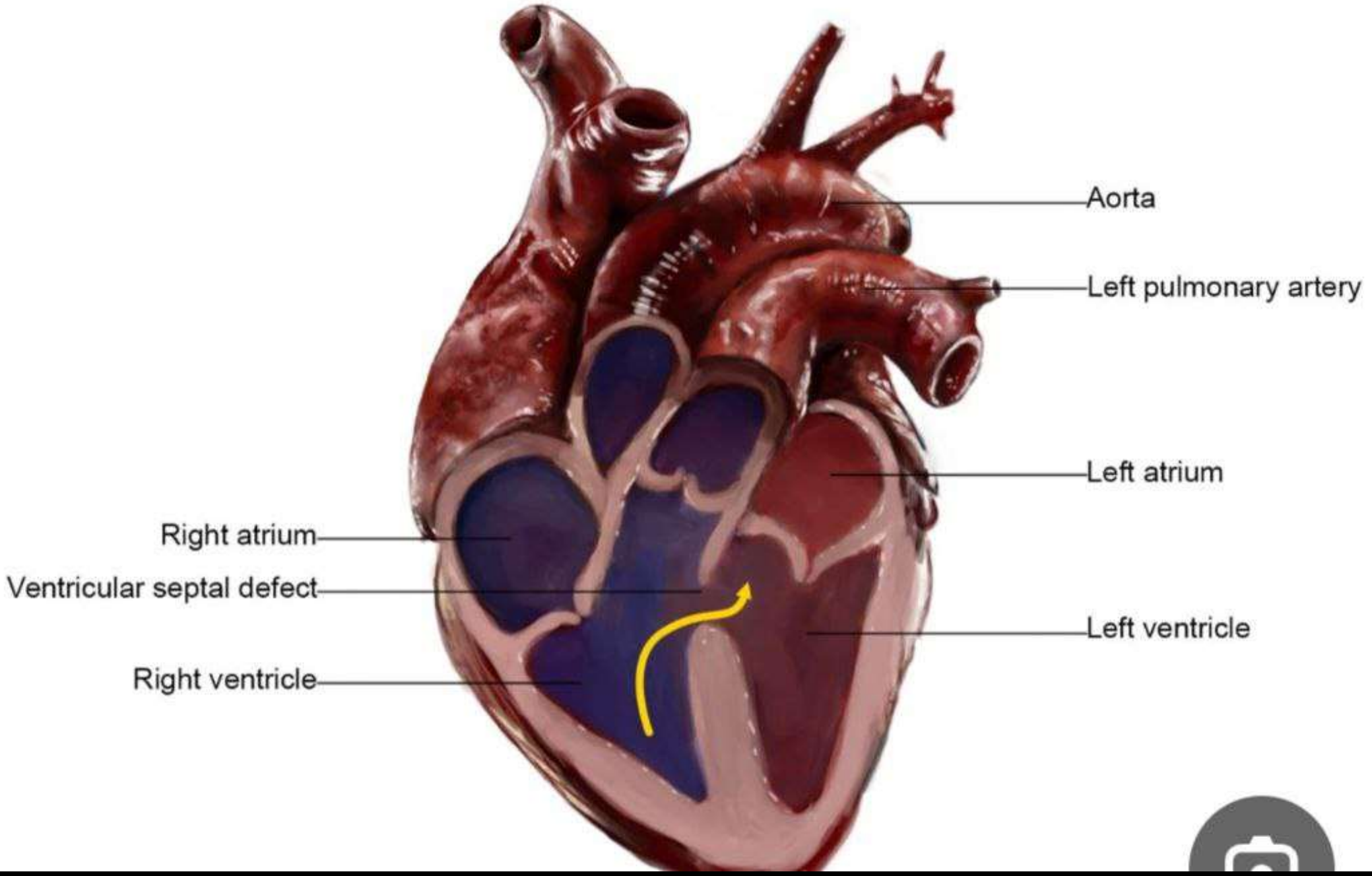
- in pulmonary arterial obliteration and pulmonary HTN, eventually leading to a right-to-left shunt

- This serious condition carries a maternal mortality rate of 50% and a
- fetal mortality rate of more than 50% if cyanosis is present
- In addition, 30% of fetuses exhibit intrauterine growth restriction.
- ***Because of increased maternal mortality, pregnancy is generally)***
- ***(contraindicated, and termination of the pregnancy should be discussed***

- If the pregnancy is continued, special precautions must be taken during the peripartum period-
- Women with Eisenmenger syndrome tolerate hypotension poorly-
- The patient should be monitored with a Swan-Ganz catheter, and care should be taken to- avoid hypovolemia

- Postpartum death most often occurs within 1 week after delivery;
- however, delayed deaths up to 4 to 6 weeks after delivery have been
- reported

Eisenmenger's Syndrome



Marfan syndrome: is an autosomal dominant disorder of the fibrillin *

.gene characterized by connective tissue fragility.

Cardiovascular manifestations include *aortic root dilation and dissection

*MVP, and *aneurysm

Genetic counseling is recommended. According to the 2010

American College of Cardiology(ACC),(patients with a dilated aortic root > 40mm are considered high risk)

If cardiovascular involvement is minor and the aortic root diameter is

smaller than 40 mm, the risk in pregnancy is less than 1%

- If cardiovascular involvement is more extensive or the aortic root is

larger than 40 mm, complications during pregnancy and aortic dissection are increased significantly

-Patients should be monitored with serial physical exams as well as echocardiography

During pregnancy, the hyperdynamic state can increase the risk of aortic dissection and/or rupture, particularly in those patients with an aortic root diameter greater than 40mm.

- Beta-blockade is recommended for patients with Marfan syndrome from the second trimester onward, particularly if the aortic root is dilated.
- Regional anesthesia during labor is considered safe. Women should labor in the left lateral decubitus position with the second stage shortened by instrumental vaginal delivery.
- Cesarean section should be reserved for obstetric indications.

Pulmonary vascular disease

- Pregnancy in the presence of pulmonary hypertension of any cause remains high risk

- Fixed pulmonary vascular resistance prevents any increase in pulmonary blood flow matching the increased cardiac output.

- Pregnancy is poorly tolerated, with a risk of worsening cyanosis and hypoxia, arrhythmias, heart failure and death.

- Most complications occur at term or during the first postpartum week.
- Maternal mortality depends on the underlying cause With secondary pulmonary hypertension being the most fetal then Eisenmenger syndrome then primary pulmonary hypertension.

- Women should be advised of these risks when contemplating pregnancy.

Peripartum cardiomyopathy

- :Definition
- Development of an acute heart failure with reduced
- ejection fraction (systolic heart failure) during the last
- month of pregnancy or within 6 months after delivery

It appears to be a dilatational cardiomyopathy with a •
decreased left ventricular ejection fraction (LVEF < 45%) •

*Epidemiology

Incidence : 1– 4 in 4,000 deliveries

At-risk group: African Americans (account for >40% of cases)

- **Risk factors**
- Multiple gestation pregnancy & Multiparity
- • Cardiovascular risk factors (Age , Hypertension , Smoking , Dm)
- • Pregnancy complicated by hypertension
- • Prolonged use of tocolytics
- • Previous history of PPCM

- ***Pathophysiology**
- • Patients with this condition have no underlying cardiac disease, and
- symptoms of cardiac decompensation appear during the last weeks of
- pregnancy or within 6 months postpartum.
- • The pathophysiology of PPCM is thought to be multifactorial involving
- **inflammatory, hormonal, genetic mechanism**
- #Possible contributing factors :
- • Genetic predisposition
- • Viral infection
- • Autoimmune processes
- • Nutritional deficiencies

Clinical features

Symptoms onset Most common in the first month after delivery and
Can occur during pregnancy in the 3rd trimester

Common manifestation: signs and symptoms of heart failure
Including dyspnea, orthopnea, PND, tachycardia,
.peripheral edema, rales, and elevated JVP

Less common presentations (Due to severely reduced ejection fraction, which can be further complicated by a LV thrombus)
Cardiogenic shock
Thromboembolic event

**The severity of symptoms in patients with PPCM can be classified by the New York Heart Association system

Class I - Disease with no symptom

Class II - Mild symptoms/effect on function or symptoms only with extreme exertion

Class III - Symptoms with minimal exertion

- Class IV - Symptoms at rest

Diagnosis

- PPCM is a diagnosis of exclusion

Must consider differentials including : Pre-existing dilated cardiomyopathy , valvular heart disease ,hypertension heart disease, congenital heart disease ,pulmonary embolism .

Investigations

*ECG

BNP*

CXR*

Cardiac imaging *

- **Complications**
- • Persistent left ventricular dysfunction and heart failure
- • Cardiogenic shock
- • Cardiopulmonary arrest
- • Thromboembolic complications
- • Poor obstetric and neonatal outcomes
- • Death

Management (Overview)

- **Acute and long-term treatment of PPCM is similar to that of HFrEF due to other causes.**
- **Ensure medications are safe to use during pregnancy or lactation.**
- **Multidisciplinary care, involving cardiology, obstetric, neonatology, and intensivist expertise is recommended.**
- **Patients are at increased risk of thromboembolic events and may require anticoagulation.**

Initial management of PPCM

- Acute decompensated PPCM (unstable) : **Urgent stabilization** (Supplemental oxygen , Intravenous furosemide , intravenous norepinephrine)
- **Hemodynamically unstable despite optimal medical therapy: Consider urgent cesarean delivery .**
- Hemodynamically stable patients : Management is similar to that in nonpregnant patients with systolic heart failure & ensure medications are safe for use in pregnancy or lactation.

Anticoagulation in PPCM

- Indications :

1- Confirmed thromboembolic event.

2- LVEF ≤ 30–35% .

* Modifications for pregnancy or lactation:

- Pregnancy: Use Unfractionated heparin or LMWH.
- Lactation: Use UFH, LMWH, or warfarin.

*** Warfarin and DOACs are both contraindicated during pregnancy but during lactation only DOACs are contraindicated .

Delivery

* Acute decompensated PPCM **(despite optimal medical** therapy): Consider **urgent cesarean delivery**
* Stable PPCM: **vaginal delivery at term** , if feasible

Breastfeeding

* Acute decompensated PPCM: Consider delaying breastfeeding until clinically stable
* Stable PPCM: breastfeeding is likely safe and should not be discouraged

Contraception

Contraception counseling is essential and should be done at the time of diagnosis or before hospital discharge.

First-line: progestin-releasing IUDs, subdermal progestin implants, vasectomy, or tubal ligation.

Second-line: depot medroxyprogesterone acetate (Considered safe but less effective than first-line options (

**Estrogen-containing contraceptives have prothrombotic effects and increase the risk of thromboembolism in this group of patients.

**They may also worsen hypertension and increase the risk of heart failure exacerbation and arrhythmias.

Subsequent pregnancies

Preconception counselling:

Persistent LV dysfunction (LVEF<50%) : Advise strongly against pregnancy because of the high risk of deterioration

Recovered LV function (LVEF>50%) : Shared decision-making is advised in consultation with cardiology and obstetrics

** If pregnancy is planned, clinical stability must be ensured and medication adjustments (e.g., discontinuing and/or replacing ACE inhibitors, ARBs, mineralocorticoid receptor antagonists) should be considered prior to pregnancy

**Management of subsequent pregnancies:

-Closely monitor patients for PPCM recurrence during pregnancy and for at least one year postpartum

-Termination of pregnancy should be discussed in high-risk patients

Prognosis

Prognosis depends on normalization of left ventricular size and function within 6 months after delivery

- 95% of patients documented a 5-year survival
- 5% of patients make a spontaneous and full recovery

➢Predictors of recovery :

_ LVEF < 30% at the time of diagnosis
_ Lack of troponin elevation
_ Lower levels of BNP
_ Absence of LV thrombus
_ Breast feeding