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Ward Rounds in Obstetrics & Gynecology

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Foreword
Sir Sabaratnam Arulkumaran

JAYPEE

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CHAPTER

Heart Diseases and Pregnancy: Cardiologist's Perspective

Nagesh CM

CASE 1

A 22-year-old female patient with 5 months of amenorrhea presented to us with history of exertional dyspnea and palpitations of 2 months duration. Also gives history of one episode of paroxysmal nocturnal dyspnea. No history of swelling of limbs, abdominal distension or facial puffiness. No previous history of rheumatic fever or penidure prophylaxis. On examination, pulse, blood pressure and jugular venous pulse (JVP) were normal cardiovascular examination revealed loud first heart sound with mid-diastolic murmur in mitral area and loud pulmonary component of second heart sound. Rest other systems were normal.

Q. 1. What is your diagnosis?

Rheumatic heart disease: Severe mitral stenosis, pulmonary hypertension in sinus rhythm, NYHA class II, with no signs of infective endocarditis.

Q. 2. How did you diagnose RHD in this patient without history of rheumatic fever?

In 50% of patients may not have history of rheumatic fever or penidure prophylaxis in the past. Most common valve lesion in pregnancy is mitral stenosis and etiology of MS is rheumatic in 99% of cases.

Q. 3. What are the normal hemodynamic changes during pregnancy?

Shown in the Table 1.

Table 1: Physiological changes during pregnancy and childbirth

Pregnancy	*Childbirth*
• Increase in cardiac output by 30–50% by the end of T1 and continues to rise thereafter • Augmentation of stroke volume resulting in a systolic murmur of grade 2/6 early pregnancy • Blood volume increase by a maximum of 40% by 24 weeks • Heart rate increase by 1–30 beats in late pregnancy • Reversible chamber dilatation in up to 30% of women • Vena caval compression–decreased venous return-reduction in preload	• Cardiac output increases by another 15% in early 1st stage, by 25% in late 1st stage and by up to 50% during expulsive part of 2nd stage • Heart rate and BP may increase due to anxiety and pain
	After childbirth
	• Acute increase in preload due to elimination of vena caval compression, edema resolution and backflow from involuting uterus—partially compensated by blood loss during delivery
• Systemic vascular resistance decreases secondary to placental circulation being low resistance and also vasoactive substance change in levels	

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Q. 4. What are the differentiating symptoms and cardiac findings during pregnancy between physiological and pathological changes?

Symptoms and signs of pregnancy may mimic cardiac disease due to the physiological changes (Tables 2 and 3).

Table 2: Signs and symptoms common for both pregnancy and cardiac disease

Symptoms	*Signs*
• Easy fatigability • Palpitations • Pedal edema • Syncope, urinary frequency • Breathlessness	• Tachycardia • Collapsing pulse • Precordial heave • S3 being heard • Systolic murmur up to grade 3 • Dilated neck veins

Table 3: Signs and symptoms that should rise the suspicion of cardiac disease

Symptoms	*Signs*	
• Dyspnea at rest • Orthopnea and PND • Progressive symptoms • Repeated syncope attacks • Above ankle and persistent edema	• Bradycardia <50 • Pulmonary rales • Severe tachycardia >150 • Raised JVP • Diastolic murmur • Grade 4 onwards systolic murmur	• Cyanosis • Clubbing • Pulmonary rales • Cyanosis • Clubbing • Cardiomegaly

Q. 5. What are etiologies of valvular heart diseases in women of child-bearing age?

Given in Table 4.

Table 4: Etiology of cardiac disease in women of reproductive age group

Valve	*Congenital*	*Rheumatic*	*Cardiomyopathy*	*Others*
Mitral	• Stenosis • MVP	• Stenosis • Regurgitation	• Tethered valve • Hypertrophic CMP anterior leaflet motion during systole	• Prior infective endocarditis • Prosthetic valve
Aortic	Bicuspid valve	• Stenosis • Regurgitation		• Marfan • Ehler danlos • Turner • Prior Infective endocarditis • Prosthetic Valve • Connective tissue disorder • Ross procedure
Pulmonary	Stenosis Noonan's syndrome Postoperative —TOF			• Postoperative residual regurgitation • Prosthetic valve
Factors altering the outcome			• Arrhythmias • IHD • Prior heart failure • Anticoagulation • Ejection fraction <40%	• Severe pulmonary hypertension • Aortic coarctation • Aortic root dilatation • History of stroke or TIA

Q. 6. How does the hemodynamic changes of pregnancy affect mitral stenosis?

The increase in heart rate limits the available time for left ventricle (LV) filling, resulting in increasing left atrial and pulmonary pressures leading to pulmonary edema.

Increased volume load and tachycardia cause patient to deteriorate and advance from one NYHA to higher.

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The risk of maternal death is greater during labor and postpartum period due to increase in preload from autoperfusion of uterus which flood central circulation leading to pulmonary edema.

Q. 7. What are the causes for decompensation of stable valvular heart disease (MS) in pregnancy?

- Intercurrent infections
- Anemia
- Pulmonary embolism
- Atrial fibrillation
- Severe preeclampsia.

Q. 8. How do you investigate this patient?

- Routine investigations
  - Hb for anemia
  - Total and differential counts, ESR-infections
  - Renal function tests and electrolytes
  - Thyroid function tests
- ASLO (antistreptolysin antibodies), CRP (C-reactive protein)
- Electrocardiogram (ECG)
- Echocardiogram
  - To confirm rheumatic heart disease etiology
  - Assess valve lesions and their severity
  - Assess valve suitability for PTMC
  - Any clot in left atrium or appendage.
  - Other investigations routinely done in pregnancy along with an anomaly scan.

Q. 9. How do you monitor pregnancy with valvular heart disease?

Patient with known valvular heart disease must undergo proper prepregnancy counseling.

Patients decompensate usually during 20–22 weeks and around 30 weeks when cardiac output is high, so regular follow-up recommended before pregnancy, after pregnancy confirmation.

Regular penidure prophylaxis every 3 weeks to be continued.

Patient should be monitored for precipitating factors like anemia, thyrotoxicosis, infections, atrial fibrillation and recurrent rheumatic activity, preeclampsia and promptly treated.

Q. 10. How do you manage the case of mitral stenosis (MS) during pregnancy?

- *Mild-to-moderate MS:* Only medical management. Pregnancy is tolerated well unless decompensation occurs.
- *Symptomatic moderate-to-severe MS.*

Medical management (Valvular heart disease)

Table 5 summarizes the medical management of all valvular lesions.

Table 5: Summary of medical management of valvular lesions in pregnancy

Mitral stenosis	*Mitral regurgitation*
Beta-blockers—Metoprolol, atenolol (Class D) rarely propranlol Diuretics—Thiazide, and loop diuretics Anticoagulants Prophylaxis with antibiotics	Nitrates and hydralazine as vasodilators Diuretics rarely Anticoagulants Prophylaxis with antibiotics
Aortic stenosis	*Aortic regurgitation*
Beta-blockers—metoprolol, atenolol (Class D) rarely propranolol Diuretics—Thiazide, and loop diuretics (LVF) Anticoagulants Prophylaxis with antibiotics	Diuretics Vasodilators (ACE and ARBs not to be used)

Invasive Management

PTMC (Percutaneous trans mitral commissurotomy)

- Done in symptomatic MS refractory to medical therapy, forestall need for MVR (mitral valve replacement)
- Second trimester is the best time

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- Most accepted, reported success up to 100%
- Done using shielding of abdomen to prevent radiation.

Advantages: Maternal mortality rates for percutaneous mitral valvuloplasty were 0.2%, and fetal mortality rates were 2%, which included elective terminations; overall, the procedural success rate was 98%.

Mitral Valve Replacement

Usually deferred until postpartum, unless hemodynamically compromised not responding to medical or PTMC.

Disadvantages
- Maternal mortality—3%
- Fetal loss—12–20%

Obstetric Management
- Cesarean section indicated only for obstetric reasons.
- Endocarditis prophylaxis guidelines do not recommend antibiotic prophylaxis for vaginal delivery in women with VHD.
- Invasive monitoring including pulmonary artery pressure (PAP) and systemic vascular resistance (SVR) should be considered.
- After delivery oxytocin 10–20 units in 1,000 units of crystalloid administered to lower SVR and PVR, resulting in lower cardiac output.

Q. 11. What is the role of digoxin nowadays?

Digoxin is recommended only to the patients with atrial fibrillation for rate control in RHD and heart failure patients due to its inotropic effect. No mortality benefit noted with digoxin.

Q. 12. How do you manage this case?

As described earlier since patient has severe MS and second trimester is ideal time for PTMC, I would ask her to undergo PTMC to prevent further complications.

Q. 13. When do you refer for surgery?

- Deferred until postpartum, unless hemodynamically compromised not responding to medical or PTMC.
- Patient developing acute severe mitral regurgitation after PTMC.

14. What are the potential risks of valvular heart disease in pregnancy?

Table 6: The answer is depicted

Valve lesion	*Potential risks in pregnancy*
Tricuspid regurgitation	Associated with other cardiac lesions but usually well tolerated
Pulmonic stenosis/ insufficiency	Usually well tolerated or asymptomatic prior to pregnancy. May be of greater concern after surgery for congenital heart disease
Mitral stenosis	Atrial fibrillation, cerebral or systemic thromboembolism, pulmonary edema
Mitral regurgitation	Well tolerated and may decrease with decreased afterload
Aortic stenosis	Arrhythmia, heart failure, or syncope
Aortic insufficiency	Well tolerated and may decrease with decreased afterload

Q. 15. How do you manage aortic stenosis (AS) during pregnancy?

The etiology of AS in pregnant mother is usually congenital, bicuspid aortic valve.

Many asymptomatic women with AS have symptom onset during pregnancy due to increased systemic metabolic demands and limited ability to increase stroke volume.

Congestive heart failure is the most common complication.

Patient education and frequent maternal monitoring are used in asymptomatic patients.

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In symptomatic, treatment options include bedrest, oxygen supplementation and beta-blockers.

Balloon aortic valvuloplasty and aortic valve replacement in severe AS who failed medical therapy.

Q.16. How do you manage regurgitant lesions mitral and aortic regurgitation during pregnancy?

In theory the decrease in the SVR (systemic vascular resistance) and shortened diastole of pregnancy decreases the regurgitation severity and are well tolerated.

17. How do you manage right sided valve lesions?

- *Pulmonary stenosis:* Usually congenital. Mild to moderate lesion well tolerated. if severe and symptomatic pulmonary balloon valvuloplasty can be performed safely during second trimester.
- *Tricuspid stenosis:* Rare, but treatment with percutaneous balloon commissurotomy has been successful.
- *Right sided regurgitant lesions:* Well tolerated.

18. How do you advise prophylaxis in this rheumatic fever?

As shown in the underlying Table 7.

Table 7: Secondary prophylaxis for patients with rheumatic fever

Condition	*Prophylaxis*
Rheumatic fever with carditis and residual valvular lesions	For 10 years or till the age of 40 years (whichever is longer) or lifetime
Rheumatic fever with carditis and residual valvular lesions	For 10 years or till the age of 21 years (whichever is longer)
Rheumatic fever without carditis	For 5 years or till the age of 21 years (whichever is longer)

Table 8: Drugs used for secondary prophylaxis

Drug used	*Dosage*
Benzathine penicillin	Weight <27 kg—6L units IM 4 weekly Weight >27 kg—12L units IM 4 weekly
Penicillin V potassium	0.25 g orally twice daily
Sulfadiazine	Weight <27 kg—0.5g orally once a day Weight >27 kg—1g Orally once a day
Macrolide or azalide (For penicillin allergic patients)	Varies

Q. 19. How do you grade a pregnant patient with valvular heart disease?

NYHA classification of cardiac disease in pregnancy

NYHA class I—No limitation of routine activities.
NYHA class II—Slight limitation of routine activities.
NYHA classs III—Severe limitation of activities.
NYHA class IV—Symptomatic at rest.

20. What is the role of invasive monitoring in mitral stenosis and pregnancy?

Invasive cardiac monitoring like radial artery cannulation and pulmonary catheter are beneficial in assessing the cardiac output, pulmonary artery pressure and for guiding fluid and drug therapy, especially in NYHA III and IV patients.

CASE 2

Cardiomyopathies

Patient Mrs X wife of Mr Y, resident of Karnataka, belonging to lower socioeconomic class, is 29-year-old primigravida, para one with one living issues, with 33 weeks period of gestation with regular ANCs referred to cardiac OPD with complaints of:

- Amenorrhea since 8 months

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- Swelling of lower limbs since 7 days
- Breathlessness since last 7 days.

Patient complains of swelling of both lower limbs since 7 days which has gradually increased to involve up to mid thigh region. Pedal edema increases during evening hours and reduces on lying down. Patient also complaints of breathlessness since last 15 days. Patient gets breathlessness on immediately on lying down. It is not associated with palpitations or any chest pain. There is no history of fever, cough or decreased urine output. There is no history of asthma or chronic cough or previous cardiac diseases. No history of penidure prophylaxis.

Obstetric History

She is gravida 1,uneventful.

Past History

There is no history of cardiac disease or no history of fever, joint pain or recurrent sore throat in the past.

Personal History

My patient is a housewife. She does not have any kind of addiction.

Family History

There is no history of similar complaints in any of the family member.

Socioeconomic History

She belongs to lower middle class according to modified Kuppuswamy scale.

On Examination

My patient is conscious and well oriented to time, place and person. She is thin built.

Her height is 5 feet and weight is 50 kg. Her gait is normal.

Vitals

- Her pulse rate is 100/min regular, good in volume, bilateral synchronous without any radiofemoral delay.
- Her BP is 90/60 mm Hg
- Her JVP is raised. She is afebrile
- Her respiratory rate is 30/min

General Physical Examination

- There is no pallor or icterus
- There is bilateral pitting pedal edema.

Systemic Examination

Cardiovascular: Apex beat is present in 5th intercostals space and is heaving S1S2 normal.

Pan systolic murmur grade III is heard best over mitral area radiating to left axilla.

Respiratory: Air entry equal on both the sides. No added sounds or crepts heard.

CNS: No abnormality detected.

Q. 21. What is the final diagnosis ?

This patient with no history of past cardiac disease has presented with symptoms suggestive of biventricular failure and hence peripartum cardiomyopathy diagnosis should be kept in mind.

Q. 22. Define peripartum cardiomyopathy (PPCMP)?

Pericardiac cardiomyopathy is cardiac failure occurring in the last few weeks of pregnancy to the first five months after delivery in a pregnant lady usually with no pre-existing cardiac disease.

Q. 23. What are the risk factors associated with PPCMP?

Several risk factors are associated with PPCM include the following:

- Elderly pregnant women
- Multiparous women
- Multiple pregnancy
- Race (Negroid)
- Hypertensive disorders of pregnancy
- Cocaine exposure
- Some of the tocolytics
- Family history.

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Q. 24. How do you diagnose PPCMP?

National Heart, Lung and Blood Institute (NHLBI), with the National Institutes of Health (NIH), have given the following criteria for the diagnosis of PPCMP.

Cardiac failure occurring in a pregnant woman in the last few weeks of pregnancy or within the first five months after delivery who gives no history of previous cardiac disease and in whom no other cause for heart failure can be identified. Along with these, left ventricular malfunctioning has to be demonstrated with a fall in the ejection fraction to <45% or end diastolic dimensions of the left ventricle ≥ 2.7 cm/m^2

Cardiac Disease in Pregnancy Risk Score—One point for each 1. History of past cardiac event or arrhythmia 2. Cyanosis or NYHA >II 3. Mitral valve area <2 cm^2/aortic valve area <1.5 cm^2/ LV outflow gradient >30 mm Hg 4. LV ejection fraction <40% The cardiac disease in pregnancy (CARPREG score)	Chance of cardiac complication: 0 points = 5% 1 point = 27% ≥2 points = 75% Contraindication for pregnancy

Q. 25. What is the clinical presentation of PPCMP?

Early signs and symptoms of PPCM may often mimic normal physiological findings of pregnancy and include:

- Pedal edema, dyspnea on exertion, orthopnea, PND, and persistent cough.
- Abdominal discomfort secondary to hepatic congestion, dizziness, precordial pain, and palpitations, and, in the later stages, postural hypotension can occur. Arrhythmias and asymptomatic LV dysfunction may occur.

Signs of biventricular failure:

- Edema
- Raised JVP
- Resting tachycardia
- Hypertension
- Basal crepitations
- Signs of pulmonary/systemic embolism
- Cardiac signs: S3 gallop, loud P2, arrhythmias, murmur of mitral regurgitation.

In the majority of patients, symptoms develop in the first 4 months after delivery (78%). Only 9% of patients present in the last month of pregnancy.13% present either prior to 1 month before delivery, or more than 4 months postpartum.

Q. 26. What is the etiopathology of PPCMP?

The etiopathogenesis of PPCMP is not understood and hence various factors have been thought to be responsible, some of them being:

- Inflammatory etiology—substantiated by increase in CRP, IL6, IFN, etc.
- Autoimmunity-association with hypertension, familial occurrence, etc.
- Increased apoptosis—elderly women, multiparous women showing more risk
- Endothelial dysfunction—association with hypertensive disorders of pregnancy
- Genetic—racial and familial occurrence
- Cerium
- Nutritional aspects
- Increased soluble forms like tyrosine kinase levels—Association with preeclampsia
- *Peripartum oxidative stress:* Proinflammatory and proapoptotic factor formation (16kDa prolactin). This hypothesis has led to potential therapeutic strategy with blockade of prolactin by bromocriptine.

Q. 27. What investigations will you order in a case ?

Ans: Following Investigations are done in this case

- CBC, urine routine, LFT, RFT, electrolytes along with magnesium and calcium levels
- ESR, CRP

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- T3, T4, TSH
- Chest X ray
- Echocardiogram-clinches the diagnosis by demonstrating LV dysfunction.

Neither cardiac catheterization nor biopsy is required.

Q. 28. How do you manage this case?

PPCMP is a type of dilated cardiomyopathy and managed in the same lines as the other CMPs.

The principles of management are:

- Improve hemodynamic status
- Minimize the patient discomfort
- Improve long-term outcome.

The following interventions are useful in the treatment:

- Sodium restriction
- Inotropes—digoxin
- Diuretics—furosemide, spironolactone
- Vasodilators—hydralazine, nitrates, beta-1 selective blockers
- Anti-coagulation especially with low EF or when bromocriptine is used
- Bromocriptine.

Newer Modalities

- Devices to assist left ventricle—as a bridge before transplantation
- Implantable cardioverters for resynchronization.

Transplantation: More of rejection anticipated

Experimental therapy: Immunosuppressive and immunomodulating therapy (pentoxifylline).

Q. 29. What are the complications of PPCMP?

Maternal Complications

- Low output state can lead to chronic hypoxia, slow circulation leading to thromboembolism, arrhythmias
- Delay in diagnosis as it may be misdiagnosed as preeclampsia
- It can be undertreated because of fetal concerns.

Fetal Complications

- Fetal distress due to maternal hypoxia or drug induced hypovolemia.

Q. 30. How do you manage during labor?

Route of Delivery

As cesarean delivery is more often associated with infections and embolic events, vaginal delivery is the most preferred.

Other interventions are:

- Adequate pain control
- Take care of infection
- Manage anemia, preeclampsia energetically
- Sodium restriction is helpful
- Confining to bed in pregnancy helps to maintain uteroplacental circulation.

Q. 31. What is the prognosis and follow-up data on PPCMP?

- Recovery—50%
- Maintained on drugs—25%
- Deterioration—25%.

ACE inhibitors and β-blockers are continued for at least 1 year after normalization of ejection fraction.

Q. 32. What is the risk of subsequent pregnancy?

It is known to recur in 30% and hence further pregnancies need to be discouraged.

Q. 33. What are the other types of cardiomyopathies seen in pregnancy?

Pregnancy may be associated with other varieties of CMPs like dilated cardiomyopathy (DCM) and hypertrophic cardiomyopathy (HCM).

Q. 34. How do you manage DCM during pregnancy?

Only women with EF >50% can attempt pregnancy, ones with EF between 40–50% if with poor exercise tolerance pregnancy is

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contraindicated. If <30% pregnancy should never be attempted.

Other pregnant women with DCM are symptomatically managed along the same principles as PPCMP is managed.

Q. 35. How do you manage HCM during pregnancy?

Prepregnancy symptomatic patients do not do well in pregnancy, otherwise HCM patients tolerate pregnancy better than others.

They are managed with beta-blockers of increased dose (keeping in mind the fetal side effects).

Labor is cut short in the second stage and CS done only for obstetric indications.

CASE 3

Coronary Artery Disease

A 35 years female of Indian origin in her twenty-fifth-week of pregnancy presented to the emergency department with acute onset progressive chest pain and diaphoresis. She did not have any documented cardiovascular risk factors and denied any complications during prior normal vaginal pregnancy 7 years ago. She was taking her multivitamin pills and no other prescribed medications. Her parents and sibling did not have CAD and DM.

Initial assessment revealed an afebrile pregnant lady with blood pressure 117/79 mm Hg, pulse 88 beats per minute, pulse oximetry 99% on room air. There were no signs of congestive heart failure with clear lungs and no jugular venous distension. The precordial auscultation revealed normal sounds with physiological splitting. Electrocardiography (EKG) showed ST-segment elevation in anterolateral leads V1–V4, I and aVL with reciprocal ST depressions in inferior leads II, III and AVF. Coronary catheterization was performed through right radial artery access after shielding the patient's back and abdomen with lead aprons to minimize fetal radiation exposure. Angiogram showed total occlusion of the proximal left anterior descending (LAD) artery with normal right and left circumflex coronary arteries. Patient received 325 mg aspirin and loading dose of (600 mg) clopidogrel. Mechanical thrombectomy was performed, and a bare metal stent was deployed in the LAD. Intra-arterial heparin was used to maintain anticoagulation during coronary intervention. Post-angioplasty EKG showed resolution of ST-segments. She had an uncomplicated hospital course. Pre-discharge transthoracic echocardiography revealed normal left ventricular function without any wall motion abnormality. She was discharged on 81 mg aspirin, 75 mg clopidogrel, 25 mg metoprolol tartrate twice a day regimen. She delivered on due date at 38 weeks period of gestation without any complication, and underwent cesarean section for labor arrest. Antiplatelet regimen was not interrupted during labor and delivery in our patient. She remained asymptomatic during follow-up with optimal medical management. Atorvastatin was added to her regimen 6 months after pregnancy.

Q. 36. What are the options to manage acute MI and pregnancy?

MI in any patient has two options of management:

1. Thrombolysis and
2. Coronary angiography.

Thrombolytics are better avoided in pregnancy without the correct information about coronary vascular anatomy. This may increase the risk of hemorrhage and further dissection of atherosclerotic plate in coronary vessels.

The main concern with coronary angiography is radiation exposure, but the following facts would clarify the concerns.

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- The radiation exposure is to the tune of 0.02–0.1mGy which is far below the teratogenic threshold.
- Utilizing radial artery approach than femoral minimizes the risk of radiation.
- It helps to differentiate between organic obstruction or spasm of the vessel.
- It helps to place a stent and thus it is therapeutic too.

The main controversy is about continuing antiplatelet therapy, its duration, management during labor. Some recommend dual antiplatelet therapy combining clopidogrel and aspirin for at least 4 weeks.

Q. 37. What care to be taken during labor?

Post-MI a patient ideally should deliver at least 2–3 weeks later if feasible.

Vaginal delivery can be considered except for obstetric reasons, women on clopidogrel, or the ones in failure.

Ergots should be avoided in them. The ones who are planning pregnancy should do so after an year of MI, after evaluation for revascularization especially after weaning them from the teratogenic drugs.

Q. 38. What are the risk factors for CAD in pregnancy?

- Chronic hypertension/preeclampsia
- DM
- Dyslipidemia
- Smoking
- Family history of CAD
- Previous use of combined OC pills.

Q. 39. What are the causes for myocardial infarction in pregnancy?

- Atherosclerosis
- Coronary spasm/dissection
- Coronary vascular thrombosis
- General vasculitis
- Autoimmune factors associated with collagen vascular diseases
- Amniotic fluid embolism
- Tumor of adrenal medulla (pheochromocytoma)
- Cocaine abuse.

Q. 40. What time during pregnancy is MI common?

MI tends to occur mostly in women in labor or in the postpartum period.

It is more commonly found in the ones who are hypertensives and diabetics.

CASE 4

PPI

A 27-year-old G1P0 presented for prenatal care in the first trimester of pregnancy. She had a significant past medical history of bradycardia, hypotension and syncope that required permanent cardiac pacemaker placement 6 years earlier. In the early second trimester, the patient began experiencing light-headedness and breathlessness with exertion. Pacemaker interrogation done and reprogrammed, with resolution of the patient's symptoms. The remainder of the pregnancy progressed unremarkably. The patient underwent primary cesarean section at 39 weeks gestation with delivery of a healthy term infant. Preoperative anesthesia consultation was obtained. The postoperative course was uneventful. Pre-pregnancy pacemaker settings were re-established after the postpartum period.

Q. 41. How do you manage patient with PPI with pregnancy?

Management during Pregnancy

- Obtain detailed history regarding symptoms like palpitations, syncope, dizziness, seizures, exersional dyspnea.

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- Interrogate the pacemaker preferably as a part of prepregnancy counseling or at least in the first trimester specially to know the patient dependency on the device.
- Detailed cardiac evaluation by ECG and ECHO preferably before or at least in the first trimester of pregnancy has to be done.
- Every visit ask for symptoms
- Though they tolerate pregnancy well, decompensation may occur in third trimester
- May present with RVF, AF, any of these may result inintrauterine fetal demise also.

Q. 42. Which mode of delivery is better in patients on PPI?

PPI perse does not indicate an option for route of delivery. Decision is based on obstetric indications. Otherwise PPI patients can have a vaginal delivery.

Q. 43. What are the anesthetic considerations in patients with PPI?

Epidural is preferred over subarachnoid block.

Anesthetist should be aware of the details of pacemaker including durability and battery life.

Close monitoring throughout surgery and in the immediate postoperative period is crucial to pickup any dysfunction of pacemaker and resulting reduction in cardiac output. The signs are not well appreciated in an anesthetized patient and ECG interpretation is very important.

Any other factor which can alter the heart rate like potassium level fluctuations on either side should be monitored. Associated hypertension, use of antihypertensive like beta-blockers, calcium-channel blockers which can have an effect on the heart rate needs to be considered throughout the monitoring.

Q. 44. What other surgical precautions need to be taken?

- Avoid monopolar cautery as this can cause asystole.
- Use small bursts of bipolar cautery. Keep the cautery grounding plate as away as possible from the pacemaker, but as close to the surgical area as possible
- If the pacemaker is still affected then the pacemaker may be converted to a continuous asynchronous pacing mode (VOO) by programming or by placement of a magnet over the device.

Post-surgery monitoring:

Post-surgery change the settings of pacemaker if the pacemaker was reprogrammed before.

CASE 5

Pregnancy and Hypertension

A 35-year-old lady at 32 weeks of gestation in her first pregnancy goes to your office for a minor upper respiratory tract infection. Incidentally, her blood pressure is found to be 155/90 mm Hg with a pulse rate of 85/min. The cardiovascular examination and chest examinations are otherwise unremarkable.

Q. 45. What is your first impression about this case?

This could be a case of a pregnancy with any form of hypertension. This needs to be considered as hypertensive disorder of pregnancy is defined as any systolic reading above 140 mm Hg and diastolic reading above 90 mm Hg on 2 occasions 6 hours apart.

But here only one reading has been taken, so repeat recording for confirmation is to be done.

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But sometimes if the initial reading itself is >160/110 mmHg, strong consideration of the diagnosis has to be made as therapy becomes very essential at this level of blood pressure.

Q. 46. How do you classify hypertension in pregnancy?

Table 9: Classification of hypertension in pregnancy

Hypertensive type	*Definition/description*
1. Chronic hypertension	Hypertension BP 140/90 mmHg before pregnancy or that is diagnosed before the 20th week of pregnancy
2. Gestational hypertension	• New HTN with BP>140/90 mm Hg on two separate occasions without proteinuria after the 20th week of gestation or BP normalizes by 12 weeks postpartum • It is often a retrospective diagnosis
3. Preeclampsia superimposed on chronic hypertension	Increased BP above the patients baseline, a change in proteinuria or new onset proteinuria after 20 weeks, or evidence of end organ dysfunction
4. Preeclampsia-Eclampsia	Proteinuria >0.3g during 24 hours or grade ++ in two samples in addition to new hypertension after 20 weeks of gestation. In the absence of proteinuria, the disease should be suspected when increased BP is associated with headache, blurred vision, epigastric pain, nausea and or vomiting, low platelets, or abnormal liver enzymes

Q. 47. What are the antihypertensive drugs commonly used in pregnancy?

Antihypertensive used in hypertensive disorders of pregnancy belong to different categories:

- *Centrally acting*: Methyl dopa is the drug that has extensively been used from a long time, safe in pregnancy both for the mother and the baby. It may be associated with postpartum psychosis in some and hence it is not preferred by all after delivery.
- *Beta-blocker:* Though beta-blockers are the commonly used drugs in nonpregnant hypertensive, it is not the first line drug as it is associated with FGR, neonatal hypoglycemia, bradycardia, etc.
- *Calcium-channel blockers:* Nifedipine is the drug from this group that is used in HDP, but is the second line drug, used in addition to alpha-methyldopa or labetalol.
- *Labetalol:* Which is both alpha and beta blocker is nowadays the most commonly used drug in HDP. But, its long-term effects are not studied and safety not proved beyond doubt.
- *Diuretics:* Diuretics are not used in hypertension as they worsen the hemo-concentration associated with HDP and may compromise uteroplacental circulation. Their use is restricted to situations like massive edema especially postpartum and in the emergency management of pulmonary edema or initial stages of acute renal failure associated with hypertension.
- *Emergency use:* IV labetalol, oral nifedipine, can be used as emergency management option. If available hydralazine also may be used.

A detailed discussion of HDPs is available in Chapter 8.

Arrhythmias and Pregnancy

Q. 48. What are the mechanisms of arrhythmias in pregnancy?

Mechanisms implicated are:

- Increased heart rate and cardiac output
- Reduced SVR
- Increased plasma catecholamines and adrenergic receptor sensitivity
- Atrial stretch

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- Increased intravascular volume and end diastolic volumes
- Hormonal and emotional changes.

Q. 49. What are the diagnostic modalities used in pregnancy to diagnose arrhythmias?

- Resting ECG
- Echocardiogram
- Pharmacological testing. For example adenosine in narrow QRS tachycardias.
- Electrophysiological testing-rarely done in pregnancy.

CASE 6

Congenital Heart Disease and Pregnancy

Congenital heart diseases could belong to any of the following group:

- Acyanotic heart diseases
- Cyanotic heart diseases
- Left to right shunts.

A 29-years-old lady with history of 30 weeks of amenorrhea incidentally detected to have ejection systolic murmur (ESM) in pulmonary area on regular antenatal check-up was referred for cardiac evaluation. Patient was asymptomatic. On examination, hemodynamically stable. Cardiac examination revealed wide, fixed split S2 with grade 2 ESM. Other systems were normal.

Q. 50. What is your diagnosis?

Probably atrial septal defect (ASD).

Q. 51. What are the types of ASDs?

ASD may be classified as:

- Ostium secundum ASD—common type
- Ostium primum ASD
- Sinus venous type
- Coronary sinus type—rare type.

Q. 52. How do you investigate this patient?

ECG is to be done and depending on the findings the ASD may be categorized:

- *Incomplete right bundle branch block:* Ostium secundum ASD.
- *Left axis deviation:* Ostium primum ASD.
- *Inverted P waves:* Sinus venosus ASD.

Q. 53. How do you manage a case of ASD and pregnancy?

- ASD is well tolerated usually
- Presence of pulmonary hypertension/ atrial fibrillation worsens the prognosis
- Postpartum DVT and paradoxical embolism should be kept in mind and prevented.

Q. 54. What are the other shunt lesions seen and their effect on pregnancy?

a. *Ventricular septal defect (VSD):* VSD is well tolerated in pregnancy, but pregnancy is contraindicated in women with reversal of shunt and pulmonary hypertension.
b. *Patent ductus arteriosus (PDA):* The pregnancy outcome depends on pressure gradient across, and small communications with normal pressures are not detrimental to a pregnant mother, but again pulmonary hypertension contraindicates pregnancy.

Coarctation of Aorta (CoA)

Aortic coarctation reveals most often as secondary hypertension in pregnancy.

Lower extremity impairment in circulation is the cause of renal hypertension, can cause IUGR, occasional fetal death due to uteroplacental insufficiency.

Anti-hypertensives, surgery, stenting may have to be considered in these patients.

Cyanotic Congenital Heart Disease (CCHD)

Q. 55. What is the effect of pregnancy on cyanotic heart disease?

Normal physiological changes in pregnancy which decreases the peripheral resistance worsens the right to left shunt. Hypoxia, cyanosis will cause polycythemia, increases the risk of thrombosis in the mother and fetal growth restriction in the baby.

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Q. 56. What are potential complications of common congenital heart diseases seen during pregnancy?

Cardiac lesion	*Potential complications during pregnancy*
Atrial septal defect	• Paradoxical embolus • Eisenmenger's physiology and cyanosis
Ventricular septal defect	• Endocarditis • Eisenmenger's physiology and cyanosis
Patent ductus arteriosus	• Eisenmenger's physiology and cyanosis
Eisenmenger's syndrome	• Cyanosis • Fetal growth retardation • Maternal mortality
Coarctation of the aorta	• Decreased uterine perfusion • Hypertension • Aortic dissection • Intracranial hemorrhage • Bicuspid aortic valve pathology
Ebstein's anomaly	• Increased tricuspid insufficiency • Right ventricular failure • Arrhythmias; heart block

Complex Cyanotic Congenital Heart Disease

With the advances in the management of congenital heart disease and successful childhood surgeries, many of the girls are reaching the age of childbearing and the outcome of pregnancy is uniformly good. Early interventions have resulted in these women with normal pulmonary artery pressures and absence of cyanosis which are the predictors of good prognosis.

Post repair patients may show tachyarrhythmias, bradyarrhythmias, heart blocks, which may necessitate antiarrhythmic therapy.

Complex cyanotic CHD

- Tricuspid stenosis atresia
- Double-outlet right ventricle
- Single ventricle
- Transposition of the great arteries
- A variation of these entities

Infective Endocarditis (IE)

This is one of the grave complications during pregnancy in a woman with pre-existing cardiac disease-maternal mortality 29% and fetal mortality 23%.

It is the infection of the endocardium, endothelium of thoracic great vessels, prosthetic valves or diseased valves.

Incidence is 0.006%, more common on the left side.

Hence women with high risk lesions are usually given prophylaxis for infective endocarditis.

Prophylaxis is recommended for those with a prosthetic valve, a history of infective endocarditis, cyanotic congenital heart disease and cardiac transplant recipients with valvulopathy.

Anticoagulation and Pregnancy

Q. 57. What are the indications for antithrombotic agents in pregnancy?

- Acute VTE
- Mechanical prosthetic valves
- Prevention of pregnancy-related complications in women with antithrombin deficiency, antiphospholipid antibody (APLA) syndrome, or other thrombophilia's who have had a prior VTE.

Q. 58. Why there is increased risk of thrombosis during pregnancy?

Normal pregnancy is associated with a hypercoagulable state, which arises from the following:

- *Increased serum levels of procoagulants:* Including fibrinogen and factors II, VII, VIII, X and XII.
- Decreased protein S levels.
- *Increased resistance to activated protein C:* Observed in the second and third trimesters of pregnancy.

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- Increased serum plasminogen activator inhibitor-1 (PAI-1) and placental PAI-2 levels lead to a decreased fibrinolytic state.
- *Venous stasis:* Resulting from pressure of the gravid uterus on the inferior vena cava and decreased venous tone.
- Increased platelet adhesiveness.

Q. 59. What are the complications of anticoagulation during pregnancy?

- Use of anticoagulants in the breastfeeding mother: Heparin and LMWHs are not secreted into breast milk, and 2 reports have shown that maternal administration of warfarin does not induce an anticoagulant effect in the breastfed infant. Thus, women using these agents can safely breastfeed.
- Fetal complications of anticoagulants during pregnancy:
  - Warfarin crosses the placenta and can cause fetal bleeding and teratogenicity, later occurring mainly during the first trimester.
  - Neither UFH nor LMWH cross the placenta, therefore, these agents do not cause fetal bleeding or teratogenicity, although bleeding at the uteroplacental junction and fetal wastage are possible.
- Maternal complications of anticoagulants during pregnancy:
  - Major bleeding in 2%.
  - Immune thrombocytopenia (heparin-induced thrombocytopenia [HIT]) in 3%, which predisposes them to venous and arterial thrombosis.
  - Heparin-induced osteoporosis causes vertebral fracture in 2–3% of patients, and significant reduction in bone density is seen in about 30% of patients receiving long-term UFH. LMWH causes less osteoporosis and HIT than UFH.

Q. 60. How do you maintain anticoagulation in pregnant patient with mechanical prosthetic valves?

The main principle of management depends on:

- *Type of the valve:* Ball in cage highest risk followed by tilting disc then bi-leaflet valves.
- *Valve position:* Prosthetic valve at tricuspid region has highest risk of thrombosis followed by mitral and aortic valve.

Q. 61. What are the advantages and disadvantages of various anticoagulant drugs use in pregnancy?

- *Heparin:* Heparin is safe for fetus, does not cross placenta, and provides appropriate anticoagulant effect at appropriate doses. Disadvantage being osteoporosis and heparin induced thrombocytopenia.
- *Warfarin:* More efficacious than UFH for thromboembolic prophylaxis of pregnant women with mechanical valves. Unfortunately, warfarin therapy in the first trimester of pregnancy is associated with a substantial increase in fetal anomalies, fetal wastage (30%), prematurity (45%), and low birth weight (50%).
- Adverse effect of warfarin is dose dependent with dose >5 mg/day, fetal complications occurred in 88% and embryopathy in 8% compared with lower dose <5 mg/day which is 15% and embryopathy 0%.
- Low molecular weight heparin-attractive alternative to heparin because of ease of its use and superior bioavailability. It does not cross placenta, so no embryopathy. However, its use is associated with valve thrombosis and maternal deaths.

Q. 62. Which type of anticoagulation is better—heparin or warfarin?

In a systematic review of fetal and maternal outcome of pregnancy in patients with

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mechanical heart valves, the regimen associated with the lowest risk of valve thrombosis (3.9%) was warfarin throughout pregnancy. Using it through an entire pregnancy, however, was associated with warfarin embryopathy in 6.4% of live births. This risk was eliminated when heparin was substituted for warfarin at or prior to 6 weeks and continued until 12 weeks, although using heparin only from 6–12 weeks gestation was associated with an increased risk of valve thrombosis (9.2%).

Q. 63. What are the various regimens for anticoagulation during pregnancy?

Regimen 1: Stop warfarin from 6–12weeks gestation and anticoagulate with unfractionated heparin. Continue warfarin up to 36 weeks with goal of PT/INR 2.5–3.0. Restart heparin 2 weeks prior to planned delivery.

Regimen 2: Recent concept is if dose of warfarin required is, 5 mg/day continue the same throughout pregnancy. Change over to heparin 2 weeks prior to planned delivery.

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Ward Rounds in
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Medical education is unique in the sense that it not only involves theoretical knowledge transfer but also skill transfer. Bedside clinics, grand rounds, daily ward rounds, case discussions all aim at this skill acquisition by the learners. This book aims at providing this skill transfer exercise in the form of detailed case discussions, which are of extensive use for both undergraduates and postgraduates. The contributors are undergraduate and postgraduate examiners of various universities and experts in their respective fields.

One more uniqueness of this book is the discussion on intensive care unit (ICU) rounds, postoperative ward rounds, examination of a sexual assault victim, cardiac disease in pregnancy discussed by both cardiologist and the obstetrician, some frequently encountered issues like vulval hematoma, fever in pregnancy, role of bedside ultrasound in obstetrics, discussion on invasive fetal procedures, etc.

We are hopeful that this book will be very much useful for the students of obstetrics and gynecology, including DNB students. It may be a very useful book for practitioners and consultants too.



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