Recent Advances in Obstetrics & Gynaecology

Edited by
Mausumi Das • Togas Tulandi

Contents

Chapter 4

Pregnancy and heart disease

Shrilla Banerjee

INTRODUCTION

The normal heart is put under pressure by the effects of pregnancy, so if the heart is already compromised, pregnancy may result in a suboptimal outcome for both fetus and mother. Cardiac disease is the leading cause of maternal death and accounts for 23% of the causes of maternal death in the UK [1]. Therefore, pregnancy must be planned and anticipated in a patient with an existing heart condition, pre-conception.

Cardiac disease as a first presentation in pregnancy or acquired in pregnancy is associated with poor outcomes. Acquired cardiac disease is more common with increasing maternal age [2], smoking [3,4], diabetes [5], hypertension [6], and obesity [7,8].

The cardiac causes of maternal death include those shown in **Figure 4.1** [1].

All patients with existing heart disease should be assessed before conception. The assessment should include a full history (including exercise capacity, history of heart failure or arrhythmia), an exercise test and echocardiogram [looking at pulmonary pressures, valve dysfunction, and left ventricular (LV) dimensions and function].

The patient should be assessed during each of the trimesters and at any time a change of symptoms occurs.

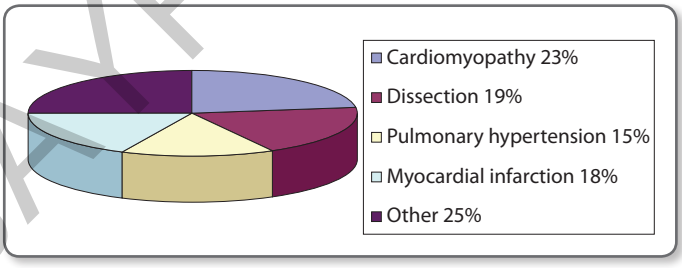

Figure 4.1 The cardiac causes of maternal death.

Shrilla Banerjee MBChB MD FRCP, Consultant Cardiologist at NHS, UK
Email: Shrilla.Banerjee@nhs.net

PHYSIOLOGICAL CHANGES ASSOCIATED WITH PREGNANCY

The changes in the cardiovascular system occur to meet the increased demands of the mother and fetus. The clinical signs and symptoms of a normal pregnancy are shown in **Box 4.1**. The total blood volume increases by 45% on average, and cardiac output increases by up to 50%, peaking in the second trimester [9,10].

Cardiac output

Stroke volume × *heart rate* = *cardiac output*

Stroke volume, heart rate, and cardiac output increase in pregnancy [11,12]. Cardiac output increases rapidly by about 30–50% in the first two trimesters and remains elevated and peaks until 30 weeks, but then may decrease slightly because of the enlarging uterus on the vena cava [12–15]. There is some debate whether the cardiac output increases, reduces, or stays the same in later pregnancy. During labour, cardiac output increases by a further 30% due to the auto-transfusion of blood from the utero-placental circulation to the maternal systemic circulation [9,13]. Following delivery, the cardiac output falls to about 15–25% above normal levels and gradually declines until normal levels are reached at about 6 weeks post-partum [13].

Systemic vascular resistance

The systemic vascular resistance (SVR) falls by 30–50% in the first 8 weeks and there is a further drop to 40% at 20–26 weeks which contributes to the hyper-dynamic circulation of pregnancy. The drop in SVR, triggers the renin–angiotensin–aldosterone system to retain sodium and water, manifests by peripheral oedema seen in pregnancy [16]. The drop in SVR corresponds to a reduction in mean arterial pressure, which starts to recover to normal pre-pregnancy levels from 26 to 28 weeks [13].

Heart rate

The rise in cardiac output is mirrored by the rise in heart rate and is triggered by the lower systemic blood pressure (BP) and the reduction in afterload. The heart rate rises between 15 and 25% during pregnancy [11,12].

Box 4.1 Clinical signs and symptoms in a normal pregnancy

- Shortness of breath and dyspnoea caused by hyperventilation
- Visible, elevated jugular venous pressure
- Displaced cardiac apex, palpable right ventricular apex
- Loud first heart sound, split second sound
- Ejection systolic murmur
- Pallor due to anaemia
- Weight gain

Blood pressure

Both systolic and diastolic BPs drop by about 10 mmHg reaching trough levels at about 20 weeks. However, by term, BP levels return to pre-pregnancy levels.

Left ventricular dimensions

The heart itself is displaced to the left and upwards, by the enlarging uterus. This results in apparent cardiomegaly on chest radiography. However, there is also a true increase in LV cavity size, caused by the increase in preload and contractility of the left ventricle secondary to increased catecholamine release results. These changes are accompanied by a degree of functional mitral valvular regurgitation and an increase in left atrial diameter.

Electrocardiogram changes

Non-specific ST-segment, Q-wave (II, III, and aVF) and T-wave (inverted/flattened T-waves leads III, V_1–V_3) changes can be seen in pregnancy, along with a resting sinus tachycardia. The cardiac axis may be slightly left-deviated due to the displacement effects of the uterus on the heart [17].

HYPERTENSION IN PREGNANCY

Hypertension complicates 10–15% of pregnancies and is responsible for up to 25% of all antenatal admissions [18]. There are recognised varieties of hypertension in pregnancy:

- Chronic hypertension
- Pregnancy-induced hypertension
- Pre-eclampsia

Risk factors for gestational hypertension include nulliparity, multiple pregnancy, previous pre-eclampsia, chronic hypertension, diabetes, obesity, and maternal age over 35 years [18].

The end effects of each of these varieties of hypertension are similar, in that they contribute significantly to maternal and fetal mortality and morbidity.

Chronic or pre-existing hypertension

If hypertension is diagnosed in the first trimester, it is likely to have been a pre-existing disorder. However, this can only be confirmed 3–6 months post-partum if the baseline BP fails to return to normal. It is defined as a systolic BP of 140 mmHg or greater and/or a diastolic BP of 90 mmHg or more.

As with all new diagnoses of hypertension, no assumption of primary or essential hypertension should be made without the exclusion of important causes of secondary hypertension, such as renal or cardiac disease, coarctation, Cushing's syndrome, Conn's syndrome, or phaeochromocytoma [19].

Examination findings to look for include radio-femoral delay (coarctation) and renal bruits (renal artery stenosis). Investigations should include urine dipstick, urea and electrolytes, renal ultrasound scan, and urinary catecholamines.

If a patient with pre-existing hypertension becomes pregnant, it is imperative to review medications [20,21].

First-line therapy – methyldopa

This is a superb drug for BP control and is known to be safe for the fetus. It can cause depression, so should be changed immediately post-partum (within 2 days of delivery). Other effects include sedation (that eventually patients become tolerant to) and postural hypotension, liver function abnormalities, and haemolytic anaemia.

Second-line therapy – calcium antagonists

Used in conjunction with methyldopa, calcium antagonists (e.g., slow release nifedipine) and oral hydralazine are often helpful in those in whom monotherapy with methyldopa has failed. Alpha-receptor blockers (doxazosin) are also useful in conjunction with methyldopa.

Third-line therapy – beta-blockers

Beta-blockers are generally well tolerated by pregnant women in addition to being effective anti-hypertensive agents. However, beta-blockers are known to cause intra-uterine growth retardation and hence patients need regular scans to assess for fetal growth. It is advisable to avoid atenolol in pregnancy. Labetalol in both tablet and parenteral form is a favoured formulation, preferably given in the second or third trimester. Also consider bisoprolol but with careful fetal growth monitoring.

- *Angiotensin-converting enzyme (ACE) inhibitors* can cause significant fetal abnormalities including oligohydramnios, renal failure, and hypotension. Patients on ACE inhibitors should be changed to a different type of medication immediately as ACE inhibitors are associated with congenital kidney abnormalities
- If a patient on ACE inhibitors becomes pregnant this is not an indication for termination, as the structural malformations caused are not related to the first trimester. The patient should be promptly converted to methyldopa. Methyldopa must be changed within 2 days of delivery due to increased risk of post-natal depression. Post-partum, ACE inhibitors can be restarted as their use in breastfeeding mothers is safe
- *Angiotensin receptor blockers* similar to ACE inhibitors. Suggest avoid in pregnancy
- *Diuretics* – not the drugs of choice in this physiological state, unless there is evidence of fluid overload, for instance in conditions such as heart failure, pulmonary oedema, or idiopathic intracranial hypertension

Management of hypertension in pregnancy

Blood pressure should be taken with the patient seated or semi-reclining but not recumbent. Tight BP control is key. Target levels lower than 150/100 mmHg, but ideally <140/90 mmHg.

- *Urine testing* for proteinuria regularly, if more than 1+ of protein, then arrange a spot urinary protein:creatinine ratio to quantify proteinuria. If >30 mg/mmol, treat as per pre-eclampsia. Consider placental growth factor (PIGF)-based testing to help rule out pre-eclampsia
- *Fetal ultrasound* at 28–30 weeks and 32–34 weeks to assess fetal growth and amniotic fluid levels

Pregnancy-induced hypertension

This usually appears in the second half of pregnancy and resolves within 6 weeks of delivery. It can persist for up to 3 months post-partum. It is a complication seen in about 5–10% of pregnancies.

Pregnancy-induced hypertension is defined as hypertension without proteinuria or other features of pre-eclampsia. The main difference between the two conditions is that the outcome is much worse with pre-eclampsia. The later the presentation in the pregnancy, the less likely the progression to pre-eclampsia (40% before 30 weeks vs. 7% after 38 weeks).

Management is similar to that of hypertension in pregnancy [19].

Pre-eclampsia

Pre-eclampsia is defined as pregnancy-induced hypertension and proteinuria and/or fetal growth restriction and/or biochemical and haematological abnormalities. The risks of developing pre-eclampsia are shown in **Box 4.2**. It is a multi-system disorder, due to diffuse vascular endothelial dysfunction. The first abnormalities occur in the first weeks of pregnancy, with abnormal placenta formation due to remodelling of the uterine spiral arteries, resulting in reduced vascular resistance, and leading to systemic endothelial dysfunction, hypertension, and renal impairment [22].

Women may present with headache, visual disturbance, epigastric or right upper quadrant pain, nausea, vomiting, or rapidly progressive oedema. The most common cause of death is cerebral haemorrhage and adult respiratory distress syndrome [23]. Fetal effects are intra-uterine growth retardation, placental abruption, and intra-uterine death [22].

Management

The aim should be to manage the patient supportively, with delivery at or after 34 weeks' gestation, if at all possible. Recommended investigations are shown in **Table 4.1**. BP control has to be a balance between reduction of maternal BP, whilst protecting the utero-placental

Box 4.2 Risks of developing pre-eclampsia

- First pregnancy
- Age 40 years or more
- Body Mass Index (BMI) of 35 or more at presentation
- Family history of pre-eclampsia (mother or sister)
- Multiple pregnancy
- 10 years or more since last pregnancy

Table 4.1 Investigations in pre-eclampsia

Urinalysis	**Microscopy and culture**
Bloods	FBC (HELLP syndrome – platelets < 100×10^9) U + Es LFTs (ALT or AST > 70 IU/L)
Clotting studies	
24-hour urine collection	Quantify protein loss
Fetal ultrasound	Fetal growth and assessment of amniotic fluid volumes
Cerebral imaging	Only if focal neurology, to exclude haemorrhage or prolonged coma

ALT, alanine transaminase; AST, aspartate transaminase; FBC, full blood count; HELLP, haemolysis, elevated liver enzymes and low platelet syndrome; LFT, liver function test.

Box 4.3 Management of hypertension in pre-eclampsia

- Mild hypertension: 140–149/90–99 mmHg
  - Monitor BP four times daily
  - Check bloods for FBC, renal function, and LFTs twice weekly
- Moderate hypertension: 150–159/100–109 mmHg
  - Monitor BP four times daily
  - Start anti-hypertensives (labetalol/methyldopa/nifedipine)
  - Target BP < 150/80–90 mmHg
  - Check bloods for FBC, renal function, and LFTs three times weekly
- Severe hypertension: >160/110 mmHg
  - Monitor BP four times daily
  - Check bloods for FBC, renal function, and LFTs three times weekly
  - Fetal ultrasound
  - Cardiotocography to assess fetal movements

BP, blood pressure; FBC, full blood count; LFT, liver function test.

circulation and maintaining fetal blood flow. BP levels over 140/90 mmHg should provoke consideration of treatment. It is mandatory for BPs over 170/110 mmHg to be treated. Headache and epigastric pain should be assessed for at every BP assessment. Management depends on severity of BP – mild, moderate, or severe, as shown in **Box 4.3**.

Management of acute severe pre-eclampsia

Delivery of the fetus is the ultimate resolution of the situation. This has to be balanced against extending the gestational period to be beyond 34 weeks if possible.
Blood pressure management includes the use of:

- Hydralazine (intermittent intravenous bolus)
- Labetalol (oral continuous intravenous infusion)
- Nifedipine tablets (oral only – never use sublingually)

If using hydralazine or nifedipine as second-line agents, the concomitant use of methyldopa reduces the side effects of headache and tachycardia.

Seizure management

Magnesium sulphate is the drug of choice to prevent and control seizures in pre-eclampsia [24]. However, when used in conjunction with nifedipine, profound hypotension may ensue.

Fluid balance

Fluid restriction is advised to reduce the risk of overload and pulmonary oedema. Pulmonary oedema is a significant contributor to maternal death.

Post-partum

- Careful BP monitoring is essential to time change/reduction of medications
- Stop methyldopa (risk of post-partum depression)

- Blood tests – monitor full blood count (FBC), liver function tests (LFTs) and creatinine 72 hours post-partum
- Offer ACE inhibitor/angiotensin II receptor blocker (ARB) medication to manage BP, even if breastfeeding
- Black (African-Caribbean) women are better managed with nifedipine or amlodipine

Prognosis

- Recurrence rate of 15% in women who had pre-eclampsia in first pregnancy [25]
- Increased risk of hypertension and heart disease [26]
- Maternal mortality rate of eclampsia is 1.8% [27]
- Low-dose aspirin (from 12 weeks' gestation) has been shown to reduce risk in those at high-risk of pre-eclampsia [28–30]

PALPITATIONS AND ARRHYTHMIA

Palpitations are a common complaint and can occur in up to 50–60% of the pregnant population [31]. Holter data for pregnant women were analysed in one study, which showed that 76% of those experiencing palpitation were found to be in sinus tachycardia alone. Only 24% of the Holter tests demonstrated any arrhythmia, most of which were benign [32]. The physical and hormonal changes associated with pregnancy may produce a pro-arrhythmic state. Increasing cardiac output results in myocardial stretch and increases left ventricular end-diastolic (LVED) pressure volumes. These changes can promote arrhythmogenesis. Arrhythmia is the most common pregnancy-related cardiac complication. The increased circulating volume in pregnancy, can contribute to increased atrial and ventricular stretch, and combined with autonomic and hormonal factors, makes pregnancy a time of increased arrhythmogenesis [31,32].

Most palpitations experienced will be due to isolated atrial and ventricular ectopic beats. In **Box 4.4** the features that may warrant further investigation are shown. Treatment includes reassurance and avoidance of precipitants. Episodes of tachycardia are more symptomatic in pregnancy and can occur in any of the trimesters.

Investigations

A thorough history, baseline 12-lead electrocardiogram (ECG), ambulatory ECG monitoring, and echocardiogram should be considered.

Box 4.4 Areas in history that suggest further investigation is necessary

- Fast and irregular palpitation
- Palpitations waking from sleep
- Pre-syncope in association with palpitations
- Pre-existing cardiac condition
- Family history of cardiac disease
- Worsening symptoms with exertion
- Associated chest pain, shortness of breath, and syncope

Sinus tachycardia

This is common in normal pregnancy. However, if the tachycardia is over 25% greater than the normal pre-pregnant heart rate, then consider hyperthyroidism, or other causes such as anaemia, hypovolaemia, sepsis, or respiratory or cardiac pathology. Cardio-respiratory causes include pulmonary embolism, phaeochromocytoma, and postural orthostatic tachycardia syndrome (POTS).

Atrial and ventricular ectopic beats

Again, it is very common in pregnancy. It is found in about 50% of pregnant women on ambulatory monitoring [32]. At low burdens (<7.5% of total beats) are considered benign and rarely require treatment.

Supraventricular tachycardia

Paroxysmal supraventricular tachycardia (SVT) is the most common non-benign arrhythmia encountered in pregnancy. A pregnant patient has a risk of developing new SVT 34% higher than at other times in her life [33]. Most are caused by atrioventricular nodal re-entrant tachycardia (AVNRT) [34]. SVT classically has an abrupt symptomatic onset and cessation. Management of an AVNRT is similar in pregnant and non-pregnant patients. Vagal manoeuvres should be the first choice. If successful in terminating the arrhythmia, no further treatment is necessary. However, if vagal manoeuvres fail, then it is worth considering intravenous adenosine that is safe in these circumstances, but whilst the women is monitored [35]. Subsequent strategies can include intravenous adenosine, propranolol, or metoprolol that are also safe to use in pregnancy. Some suggest that adenosine use in late pregnancy should be used with fetal heart rate monitoring. Flecainide and amiodarone are best avoided. Both carry the risks of teratogenesis.

If the arrhythmia is resistant to all therapies suggested, direct current (DC) cardioversion is safe, especially if performed within 48 hours of onset obviating the need for anticoagulation [36,37].

Beta-blockers are helpful agents for prophylaxis of paroxysmal SVT.

Curative catheter ablation is also possible in pregnancy, preferably in the second trimester with radiation shields placed over the abdomen. Pulsed fluoroscopy should help to limit radiation exposure. In patients with pre-existing tachycardia, a catheter ablation should be planned prior to conception if possible.

Atrial fibrillation and flutter

Atrial fibrillation (AF) and atrial flutter are rarely seen in isolation in pregnancy. These rhythms are often seen in conjunction with congenital heart disease (Ebstein's anomaly), rheumatic heart disease (mitral stenosis), or hyperthyroidism [37,38].

In patients with permanent AF, adding beta-blockers (not atenolol) and digoxin can optimise rate control. AF increases the risk of systemic thromboembolism. Anticoagulation should be considered in those at high risk of stroke such as women with permanent AF, structural heart disease, rheumatic mitral valve disease, and those with previous emboli. Heparin is safe in early pregnancy (before 12 weeks) and this can be replaced with warfarin in the second and third trimesters. It is imperative that there is collaborative planning of the delivery between cardiologists, haematologists, and obstetricians [39].

Ventricular tachycardia

A broad complex rapidly conducted heart rhythm with haemodynamic compromise should be treated as ventricular tachycardia (VT), until otherwise proven. VT is often associated with structural heart disease [hypertrophic cardiomyopathy (HCM), peri-partum cardiomyopathy (PPCM), arrhythmogenic right ventricular cardiomyopathy (ARVC)] or a primary electrical problem [long QT syndrome (LQTS)] [40].

Right ventricular outflow tract (RVOT) tachycardia is the most likely source of VT seen in pregnancy. RVOT tachycardia is known to occur in patients without recognised structural heart disease. Most episodes are related to stress and are probably catecholamine driven, given that they respond to beta-blocker therapy. One study showed that in 11 women with VT in pregnancy, 73% had monomorphic VT originating from the right ventricle [i.e. left bundle branch block (LBBB) morphology], with an inferior axis [41]. The tachycardia resolves post-delivery and the prognosis for mother and baby is good.

An echocardiogram and ECG are necessary to determine if there is any evidence of structural heart disease. The antiarrhythmic of choice for VT in pregnancy is lidocaine and electrical DC cardio-version should be performed if haemodynamic compromise occurs. DC cardio-version is safe in pregnancy and is a quick and useful tool for rapidly terminating potentially troublesome tachyarrhythmias [40].

Implantable cardioverter defibrillators

Women with implantable cardioverter defibrillators (ICDs) implanted should not be discouraged from becoming pregnant. The data is favourable for both maternal and fetal outcomes in the presence of cardiac conditions necessitating ICDs. A study of 44 pregnant women with structural heart disease and ICDs, found that none received inappropriate shocks during pregnancy. Indeed, 75% received no shocks at all. Fetal outcomes were: 89% born healthy, 4% small for dates, and 2% stillborn [42].

Long QT syndrome

Inherited LQTS is seen more commonly in women. However, women who are pregnant experience most cardiac events in the immediate post-partum period (9%), as opposed to during pregnancy (1.8–4.5%) [43]. Especially relevant to those with type 2 LQTS. The recommendation for this group is therefore to continue beta-blocker therapy throughout the pregnancy and post-partum period. There is a small risk of beta-blocker entering breast milk and causing neonatal bradyarrhythmia. However, the maternal risk is greater and takes precedence.

Cardiac Risk in the Young (CRY) a charity for cardiomyopathy and sudden cardiac death in the young has published a list of medications to avoid in LQTS [SADS: drugs to avoid. https://www.sads.org.uk/drugs-to-avoid/] [44].

Gene defects coding for cardiac ion channels have been identified as associated with inherited LQTS. There is an association with LQTS and sudden infant death syndrome, so referral for genetic review should be a priority in the post-partum period.

The options available in antiarrhythmic therapy and safety in pregnancy and breast-feeding are shown in **Table 4.2**.

Table 4.2 Antiarrhythmic medications in pregnancy and breastfeeding

Drug	Safe in pregnancy	Safe during breastfeeding
Adenosine	✔ Fetal toxicity with high doses	✔
Amiodarone	✔ Suitable for emergency use only	✖ Avoid as risk of neonatal hypothyroidism
Beta-blockers	✔ Possible association with IUGR	✔
Digoxin	✔ Fetal toxicity with high doses	✔
Flecainide	✔ Insufficient data likely to be safe	✔ Not known to be harmful
Lidocaine	✔ Fetal toxicity with high doses	✔
Verapamil	✔ Avoid rapid injection	✔

IUGR, intrauterine growth restriction.

Bradyarrhythmias

Patients with symptomatic bradycardia or congenital complete heart block should have a permanent pacemaker implanted prior to conception [45,46]. Most arrhythmias in women with a structurally normal heart are not associated with concerns around pregnancy or delivery [37]. The placing of temporary wires should be discouraged peri-delivery [45,46].

ISCHAEMIC HEART DISEASE

Acute coronary syndrome (ACS) is a rare presentation in women of reproductive age, but pregnancy itself increases the risk of an acute myocardial infarct (MI) by three to four times. The incidence is 6 per 100,000 deliveries in the Western World, but is increasing, probably in the current climate of increased maternal age, smoking, and obesity. Mortality from MI in pregnancy is in the range of 37–50% [47].

Factors such as maternal age over 40 years, diabetes, hypertension, thrombophilia, transfusion, smoking, and peri-partum infection are significant risk factors for acute MI in pregnancy [48]. Indeed, the odds of MI in one study were 30-fold increased for a maternal age of 40 years and above [49], and cardiac death four times increased when compared to those for maternal age of <20 years [50,51].

The presentation of ACS is three times more likely to be as an ST-elevation MI (STEMI) as opposed to a non-ST-elevation MI (NSTEMI) [52]. Most occur in the third trimester or post-partum, making management difficult as thrombolysis is contraindicated pre-delivery and for 10 days post-delivery. Hence, the treatment of choice is immediate (primary) angioplasty, although there is little evidence to support this choice. Atherosclerotic disease accounts for a significant proportion of ACS in pregnancy, although other causations include coronary artery thrombotic occlusion and coronary artery spasm and spontaneous coronary artery dissection (SCAD) [53].

Spontaneous coronary artery dissection is a process of coronary artery dissection, due to intramural haematoma resulting in vessel occlusion. It is more common in young women, without conventional coronary artery risk factors. The classic presentation in pregnancy is with an anterior STEMI. It is hypothesised that the hormonal effects of pregnancy result in changes in vessel structure, and that, combined with the increased haemodynamic burden in late pregnancy, puts stress on the vessel architecture.

Management of ACS in pregnancy

This is a medical emergency, so all care should be coordinated with an interventional cardiologist, obstetrician, and obstetric anaesthetist. The management of ACS in the pregnant woman should be the same as for the non-pregnant woman.

Patients should be given aspirin 75 mg stat and also clopidogrel (no data to suggest fetal toxicity) as dual anti-platelet therapy (DAPT). The fetus in the pelvis should be shielded using lead aprons and a radial arterial approach followed to allow optimal positioning of the mother to prevent vena caval compression, reducing venous return and further haemodynamic compromise (left lateral position). Consideration should be given to the type of stent used as drug-eluting stents require longer DAPT (>3 months) than bare metal stents (1 month).

Pregnancy in women with existing coronary artery disease

Generally, maternal outcomes are favourable in this group [54], but registries have shown that there are higher than expected pre-term deliveries and lower birth weights. However, if there is evidence of significant impairment of LV function, pregnancy should be discouraged [37]. The suitability and safety of ACS medications in pregnancy and breastfeeding are shown in **Table 4.3**.

Table 4.3 ACS cardiac medications in pregnancy and breastfeeding [55]

Drug	Pregnancy	Dose adjustments	Delivery	Breastfeeding
Aspirin	• Yes • Crosses placenta and can cause pregnancy loss and congenital defects	Nil – 300 mg stat and then 75 mg od maintenance	Yes	Yes
Clopidogrel	• Yes • Crosses placenta	Nil – 300 mg stat and then 75 mg od maintenance	Discontinue 7 days pre-delivery	Not recommended
Ticagrelor	Not recommended			Not recommended
Heparin (unfractionated UFH)	• Yes • Increased maternal bleeding	Nil	Discontinue during delivery or spinal anaesthesia	Yes
Heparin (low molecular weight LMW)	• Yes • Increased maternal bleeding	Dose according to booking weight	Discontinue during delivery or spinal anaesthesia Consider switching to UFH in last weeks of pregnancy	Yes
NOAC	Not recommended			Not recommended
Beta-blockers	• Yes • Risk of IUGR and fetal bradycardia • Atenolol not recommended	Nil	Yes	Yes
Statins	Not recommended			Not recommended
ACE inhibitor/ARB	Contraindicated in pregnancy			Yes

ACS, acute coronary syndrome; ARB, angiotensin II receptor blocker; NOAC, non-vitamin K oral anticoagulant.

CARDIOMYOPATHY

Cardiomyopathy is a congenital or acquired disease of the heart muscle. It describes a group of disorders in which the heart muscle is structurally and functionally abnormal in the absence of coronary artery disease, hypertension, valvular disease, and congenital heart disease, sufficient to cause the observed abnormality. Cardiomyopathy exists in different forms: dilated, hypertrophic, ARVC, peripartum, restrictive, and LV non-compaction cardiomyopathies [55].

Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) can be idiopathic or secondary to a number of different conditions including myocarditis, alcohol or other toxins, endocrine and autoimmune disorders, and nutritional factors. Pregnancy in women with DCM is associated with poor outcomes, especially those with more than moderate LV impairment [LV ejection fraction (LVEF) < 45%]. The haemodynamic changes of pregnancy, specifically the increase in cardiac work, may provoke ventricular failure, pulmonary oedema, and fetal loss in women with DCM. Those with symptoms New York Heart Association (NYHA) class III and above should be counselled against pregnancy as there is an associated maternal mortality of 7% [56,57].

Adverse events occurring in pregnancy with DCM include heart failure, VT, aborted sudden death, AF, transient ischaemic attack, and stroke and death. Women with no history of previous cardiac events, and a good NYHA class with only mild LV impairment has a reasonable chance of an event-free pregnancy [56].

Some medications for ventricular failure are teratogenic: patients should be counselled pre-pregnancy regarding the risk of both poor maternal and fetal outcomes and any ACE inhibitor or ARBs should be withdrawn if the decision is made to continue with pregnancy. The cardiac function should be reassessed prior to conception, and advice against pregnancy given if the LVEF further deteriorates.

Beta-blockers should be maintained throughout pregnancy, with the appropriate fetal monitoring for intrauterine growth restriction (IUGR). Other drugs that can be used are diuretics (to manage fluid balance), nitrates, and hydralazine (to reduce preload).

Further management is discussed below with PPCM.

Peri-partum cardiomyopathy

Peri-partum cardiomyopathy is heart failure secondary to LV systolic dysfunction, with an EF <45%, presenting between the last month of pregnancy and 5 months post-partum in a woman without previously known structural heart disease. The aetiology is unknown. The LV systolic dysfunction that occurs can be reversible [58].

Cardiomyopathy presenting earlier in pregnancy is defined as pregnancy-associated cardiomyopathy. Worldwide there is a huge variation in the incidence of PPCM with high rates in Haiti (1 in 299 livebirths) [59] and only 1 per 4,000 livebirths in the USA [60]. The lowest rates are found in Scandinavia and Japan [61,62]. The mortality rates are reported between <2 and 50%. Risk factors for developing PPCM can be seen in **Box 4.5**.

There are many hypotheses as to the aetiology of PPCM and include causes such as selenium deficiency, immune-mediated mechanism, myocarditis, and viral triggers (such as enteroviruses – *Coxsackie*, *Parvovirus B19*, adenovirus, and herpes). The role of fetal microchimerism (fetal cells enter the maternal circulation apparently to enhance maternal

Recent Advances in Obstetrics & Gynaecology 28

Recent Advances in Obstetrics & Gynaecology, volume 28, features an outstanding collection of comprehensive reviews of the latest advances in this rapidly developing specialty. It brings together 17 chapters written by expert authors who are world renowned in their field.

Featuring topics such as Noninvasive Prenatal Testing, Preterm Labour and Vaginal Microbiomes, Pregnancy and Heart Disease, Infections in Pregnancy, including the SARS-CoV-2 virus, Preeclampsia, Recurrent Miscarriage, Current Management of Endometriosis, Management of Hydrosalpinx in Women with Infertility, Immunotherapy for Ovarian Cancers, Early Cervical Cancer, Managing the Menopause, Fertility Preservation in Cancer Patients, and Precision Medicine Applications in Ovarian Cancer, each chapter provides a comprehensive overview and highlights the latest developments of relevance to clinical practice.

The chapters are presented in a user-friendly and easy to read format, making this latest volume an invaluable resource for both busy clinicians and trainees preparing for postgraduate examinations.

- 17 chapters focusing on important latest developments in the field of Obstetrics and Gynaecology
- Comprehensive review of topics written by established pioneers and world-renowned experts
- Practical and relevant content for clinical practice in a user-friendly format
- Effective revision tool for candidates sitting for postgraduate Obstetrics and Gynaecology examinations

JP medical publishers

www.jpmedpub.com

ISBN 978-1-78779-173-2