Cardiac disease in pregnancy

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Abstract

Cardiac disease is a significant cause of maternal mortality. In the UK in the last maternal mortality report, it was the leading cause of maternal death (2.3 deaths per 100,000 maternities). Some mothers are leaving having a family until later in life and present with a variety of acquired heart conditions and there are increasing numbers of patients with corrected congenital heart disease surviving to childbearing age. It is essential to have a good knowledge of the cardiac conditions, how pregnancy affects these conditions and an understanding of the physiological changes during pregnancy. These patients can turn up on any delivery suite or in any hospital at any time.

Keywords Cardiac disease; cardiomyopathy; maternal mortality; pulmonary hypertension

Royal College of Anaesthetists CPD Matrix: 1A01, 2B06, 2C04, 3B00.

Learning objectives

After reading this article, you should be able to:

- list five predictors of cardiac events during pregnancy
- explain the physiological cardiovascular changes in pregnancy
- describe WHO classification of maternal cardiovascular risk

Physiology

The physiological changes to the cardiovascular system during pregnancy take place to facilitate the increased demand for oxygen and nutrients by the utero-placental unit, and that of the growing fetus. Blood flow to the uterus increases from 50 ml/minute at 10 weeks' gestation to 850 ml/minute at term. This is accomplished by a 50% rise in cardiac output (CO) and blood volume. There is a drop in systemic and pulmonary vascular resistance due to vasoactive prostaglandins and nitric oxide production, thus preventing a rise in pulmonary artery pressure from the increased circulating volume.[1] The systolic and diastolic pressures fall, reaching their lowest values during the second trimester, before increasing as term approaches, although never reaching pre-pregnancy values.

During labour CO increases by a further 25–50% with up to 500 ml of blood returned from the intervillous spaces during contractions. The CO increases again after delivery associated with a relative hypervolaemic state. This can result in a clinical deterioration in patients with cardiac disease due to the associated rise in ventricular filling pressure and end-diastolic volume. There is a 15% reduction in colloid osmotic pressure during pregnancy, further increasing the risk of pulmonary oedema in these patients.

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General approach to cardiac disease in pregnancy

Ideally preparation should occur before pregnancy occurs. The responsible cardiologist and obstetrician should undertake preconceptual counselling. Risks involved to the patient should be discussed openly so that they can make an informed choice. Cardiac function should be optimized, which may include surgical interventions such as valve replacement. Known teratogenic medications should be discontinued. Women should be seen early in established pregnancy in joint cardiology and obstetric clinics. A multidisciplinary care plan can be made and considerations of what facilities are required at the unit where delivery will take place such as cardiology, cardiothoracic surgery and cardiac or general intensive care. An anaesthetist should review the woman at an early stage to make a plan for delivery and post-partum care.

In general, predictors of a primary cardiac event during the pregnancy include:[2]

- prior cardiac event or stroke
- prior arrhythmia
- New York Heart Association (NYHA) functional class greater than 2
- left-sided heart obstruction (mitral valve area <2 cm^2, aortic valve area <1.5 cm^2 or outflow tract gradient >30 mmHg)
- left ventricular ejection fraction less than 40%.

Congenital heart disease

In most cases of congenital heart disease the diagnosis, functional status and any therapeutic strategies will be well established pre-pregnancy. Depending on the condition pregnancy could be well tolerated or be classified as risk class IV in the modified WHO classification of maternal cardiovascular risk (Table 1).

Acquired heart disease

Acute coronary syndromes

As the average maternal age increases, the number of women at risk of an ischaemic event will also increase. Acute coronary syndromes (ACS) are estimated to occur at 3–6 per 100,000 deliveries and are related to the characteristic risk factors of smoking, hypertension, hyperlipidaemia, diabetes and a positive family history. Spontaneous coronary artery dissection is more common in pregnancy, usually occurring in the peri-partum period. Maternal mortality after an ACS is estimated at 5–10%, which has improved with the increased availability of primary percutaneous coronary intervention (PCI).

Modified WHO classification of maternal cardiovascular risk[2]

Risk class	Risk of pregnancy
I	No detectable increased risk of maternal mortality and no/mild increase in morbidity
II	Small increase risk of maternal mortality or moderate increase in morbidity
III	Significantly increased risk of maternal mortality or serious morbidity. Requires expert counselling and intensive specialist input throughout
IV	Extremely high risk of maternal mortality or severe morbidity, pregnancy contraindicated. If becomes pregnant termination should be discussed, if pregnancy continues care as class III

Table 1

The diagnostic criteria of an ACS are similar to those of non-pregnant patients. There must be a low threshold for further investigation of parturients and post-partum women that present with chest pain. A history of chest pain, ECG changes and troponin rise are the hallmarks, although inverted T waves may occur in pregnancy without underlying ischaemia. PCI is the preferred therapy of choice and stenting has been performed successfully during pregnancy. Tissue plasminogen activator does not cross the placenta, however it may cause catastrophic bleeding around the placenta and should only be used in life-threatening ACS where PCI is not available.[3] Low-dose aspirin and β-blockers are safe to use in pregnancy. If clopidogrel is indicated it should be used for the shortest duration possible and stopped prior to delivery.

Valvular heart disease

Stenotic valvular disease carries a higher risk than regurgitant lesions during pregnancy.[4] In mitral stenosis (MS) heart failure occurs frequently in those with valve areas less than 1.5 cm^2 even when previously asymptomatic. Pulmonary oedema may occur, particularly if atrial fibrillation occurs. The third stage of labour may also precipitate pulmonary oedema in these patients, which should be managed in the same way as non-pregnant patients.

Those with moderate or severe MS should be advised to delay pregnancy until balloon dilatation or valve replacement is performed. In those already pregnant, β-blockers are used to control the heart rate and allow left atrial emptying, and diuretics can be used to control symptoms. In those who are still symptomatic, percutaneous balloon dilatation can be performed. Open surgery to the valve should be avoided if at all possible. Vaginal delivery is appropriate for most patients, however those with NYHA class 3 or 4, or where balloon dilatation has failed, should be considered for caesarean section.

Aortic stenosis (AS) is primarily caused by a congenital bicuspid valve and is often well tolerated if mild/moderate in an asymptomatic patient. Patients with symptomatic AS and impaired left ventricular function are at the greatest risk of heart failure and arrhythmias from the increase in cardiac output associated with pregnancy. A general anaesthetic for caesarean section is often utilized for patients with severe AS in order to avoid the drop in systemic vascular resistance associated with a regional technique.

Mechanical heart valves and anticoagulation

Mechanical heart valves require lifelong anticoagulation, there is concern of valve thrombosis during pregnancy. The safest strategy for the mother is to continue warfarin as this is associated with the lowest rate of valve thrombosis. However, warfarin is associated with a risk of embryopathy, stillbirth, and fetal intracerebral haemorrhage. Unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH) are alternative methods of anticoagulation but carry a higher risk of valve thrombosis. LMWH may be used throughout pregnancy or in the 6–12-week period to avoid embryopathy associated with warfarin. Close monitoring of the anti-Xa levels, keeping peak levels at 0.8–1.2 U/ml, is essential as increased volume of distribution and renal clearance often lead to an increase in the dose requirement. Choice of anticoagulation regimen depends on valve type, previous thrombosis, dose of warfarin, and maternal preference. It should be explained to the woman that the risk of embryopathy is low when the daily dose of warfarin is less than 5 mg. However, she should also be given the option to replace it with LMWH during weeks 6–12. The use of LMWH throughout the entire pregnancy is not recommended although warfarin should be switched to LMWH prior to delivery.

Endocarditis prophylaxis

The 2008 National Institute for Health and Clinical Excellence guidelines on antibiotic prophylaxis for infective endocarditis advice against offering routine antibiotic prophylaxis to women during childbirth. The American Heart Association suggests that patients with conditions posing a high risk for infective endocarditis (i.e. prosthetic valve, congenital heart disease, previous infective endocarditis, cardiac transplant) should receive an antibiotic active against enterococci (ampicillin, vancomycin or piperacillin) during operative procedures or prior to urinary tract manipulation.

Aortic disease

Various conditions may predispose to aortic disease (Box 1.) Recommendations include counselling about the risk to the mother antenatally, imaging the aorta and close monitoring of aortic size during the pregnancy. If the ascending aorta is more than 4.5 cm a caesarean should be considered. Aortic dissection is a potential risk and there were seven deaths in the last maternal mortality report. Measures to minimize cardiovascular stress at the time of delivery are important in patients with a dilated aorta, these include β-blockade and/or regional anaesthesia.

Pulmonary hypertension

There is a high maternal mortality rate associated with pulmonary hypertension, previously up to 50% in historical series, and more recently published data suggests mortality figures of 15–30%. Death occurred in the third trimester or first months