

Management of cardiac conditions in pregnancy

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Cardiac disease is the most common cause of death during pregnancy. Women at increased risk require early identification and referral for expert multidisciplinary management in a tertiary centre. Three cases illustrate presentations of pre-existing and acquired cardiac disease that should not be missed and their management.

Key points

- **Cardiac disease is the leading cause of death in pregnant women.**
- **Counselling and assessing women with cardiac disease (preferably before pregnancy) reduces their risk and improves maternal and fetal outcomes.**
- **Chest pain or progressive dyspnoea in pregnancy must always be investigated, as these symptoms may be the first presentation of underlying cardiovascular disease.**
- **A multidisciplinary approach in a tertiary centre is important for management of women with significant cardiovascular disease.**



Cardiac disease is the major cause of morbidity and mortality in pregnant women and affects 0.2 to 4% of all pregnancies in the western world.¹ Data from CEMACH (Confidential Enquiry into Maternal and Child Health) in the UK for 2003 to 2005 show that half of the maternal deaths in pregnancy arose from ischaemic heart disease, cardiomyopathy or aortic dissection. The numbers have increased substantially each triennium since 1985.² Factors contributing to the increase in pregnant women with cardiac disease include the growing number of women with congenital heart disease who survive to childbearing age and the sharp increase in comorbidities such as diabetes, hypertension and obesity among pregnant women.

This article outlines some of the most common cardiac problems in pregnancy: acute coronary syndrome, valvular heart disease (including the presence of prosthetic heart valves) and cardiomyopathy. Three cases are presented that help illustrate these conditions in pregnant women and focus on appropriate management.

Circulatory changes in pregnancy

Pregnancy is a period of considerable haemodynamic change (Box 1). The most marked circulatory changes occur in the third trimester and during labour itself. During the first stage of labour, cardiac output increases 15 to 30% and during the second stage it increases approximately 50%. In the early postpartum phase, cardiac output can increase as much as 80% above baseline because of factors such as autotransfusion of blood into the circulation with uterine contractions and relief of aortocaval compression.³

These circulatory changes can unmask or exacerbate pre-existing cardiac diseases such as congenital, coronary artery and valvular disease and cardiomyopathy. In addition, some cardiac conditions,

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1. Haemodynamic changes in pregnancy

- Blood volume increases 45% (1.2 to 1.5 L) above nonpregnant levels
- Interstitial and plasma volumes increase
- Production of red blood cells increases and red cell mass increases 40%
- Ventricular muscle mass and end-diastolic volume increase
- Ejection fraction is essentially unchanged
- Myocardial contractility improves

such as peripartum cardiomyopathy, are specifically associated with pregnancy, and others, such as spontaneous coronary artery dissection, have a significantly higher incidence during pregnancy.

If the morbidity and mortality of cardiac conditions during pregnancy are to be improved then women at increased risk need to be identified early and referred for management in an appropriate tertiary centre by a skilled multidisciplinary team. Women with pre-existing heart disease can benefit from referral to a multidisciplinary team before pregnancy for preconception planning. The modified WHO classification of maternal cardiovascular risk in pregnancy is shown in Box 2.⁴

Commonly used cardiac drugs and their safety during pregnancy are summarised in the Table.

Acute coronary syndromes

Acute coronary syndromes are the leading cause of mortality in pregnancy. They occur in about one in 16,000 pregnant women and are becoming more frequent.⁵ This is because of the increase in traditional risk factors for coronary artery disease in the general population and the trend for deferring pregnancy until later maternal age. Factors that increase the risk of acute coronary syndromes are listed in Box 3.⁶

Pregnancy also increases the risk of spontaneous coronary artery dissection (SCAD; Figure 1). The reasons are not clear but likely relate to hormonal factors that

2. Modified WHO classification of maternal cardiovascular risk in pregnancy*

Conditions in which pregnancy risk is WHO I[†]

- Uncomplicated, small or mild pulmonary stenosis, patent ductus arteriosus, mitral valve prolapse
- Successfully repaired simple lesions: atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage
- Atrial or ventricular ectopic beats, isolated

Conditions in which pregnancy risk is WHO II or III

WHO II (if otherwise well and uncomplicated)

- Unoperated atrial or ventricular septal defect
- Repaired tetralogy of Fallot
- Most arrhythmias

WHO II to III (depending on individual)

- Mild left ventricular impairment
- Hypertrophic cardiomyopathy
- Native or tissue valvular heart disease not considered WHO I or IV
- Marfan's syndrome without aortic dilatation
- Aorta less than 45 mm in aortic disease with bicuspid aortic valve
- Repaired coarctation

Conditions in which pregnancy risk is WHO III

- Mechanical valve
- Systemic right ventricle
- Fontan circulation
- Cyanotic heart disease (unrepaired)
- Other complex congenital heart disease
- Aortic dilatation of 40 to 45 mm in Marfan's syndrome
- Aortic dilatation of 45 to 50 mm in aortic disease associated with bicuspid aortic valve

Conditions in which pregnancy risk is WHO IV (pregnancy contraindicated)

- Pulmonary arterial hypertension of any cause
- Severe systemic ventricular dysfunction (ejection fraction less than 30%, NYHA class III–IV)
- Previous peripartum cardiomyopathy with any residual left ventricular dysfunction
- Severe mitral stenosis, severe symptomatic aortic stenosis
- Marfan's syndrome with aortic dilatation over 45 mm
- Aortic dilatation over 50 mm in aortic disease associated with bicuspid aortic valve
- Native severe coarctation

Abbreviation: NYHA = New York Heart Association.

* Adapted from Thorne et al. *Heart* 2006; 92: 1520-1525.⁴

[†] Key to risk categories: I = no detectable increased risk of maternal mortality and no/mild increase in morbidity; II = small increased risk of maternal mortality or moderate increase in morbidity; III = significantly increased risk of maternal mortality or severe morbidity. Expert counselling required. If pregnancy is decided on then intensive specialist cardiac and obstetric monitoring is needed throughout pregnancy, childbirth and the puerperium; IV = extremely high risk of maternal mortality or severe morbidity; pregnancy contraindicated. If pregnancy occurs termination should be discussed. If pregnancy continues, care as for class III.

influence the connective tissue of the vessel wall, making it more prone to dissection. SCAD occurs much more often in the third trimester and has a predilection for the left main or left anterior descending (LAD) artery rather than the circumflex or right coronary artery.⁷

The differential diagnosis of chest pain

in pregnancy also includes aortic dissection, which is the second most common cause of maternal death during pregnancy. Typically, aortic dissection in pregnancy involves the ascending aorta, although it can propagate to involve the descending aorta as well. Aortic dissection is markedly increased in patients with Marfan's syndrome or other

Table. Safety during pregnancy of commonly used cardiac drugs

Drug	Use in pregnancy*
ACE inhibitors, angiotensin receptor blockers	D
Atorvastatin	D
Beta blockers	
Labetalol	C
Metoprolol	C
Atenolol	C (D in USA)†
Clopidogrel	B
Digoxin	A
Diltiazem	C
Diuretics	C
Hydralazine	C
Nifedipine	C
Sotalol	C
Warfarin	X

* Key to safety categories in pregnancy:
 A = drugs taken by a large number of pregnant women without an increase in the frequency of malformation or harmful effects on the human fetus;
 B = drugs taken by only a limited number of pregnant women without an increase in the frequency of malformation or harmful effects on the human fetus. Animal studies may be lacking or show harmful effects on fetus but significance uncertain in humans;
 C = animal studies revealed adverse effects on fetus, no controlled studies in women. Give if potential benefits justify potential risks;
 D = positive evidence of human fetal risk;
 X = studies in animals or humans have demonstrated fetal abnormalities, risks outweigh benefits.
 † Atenolol is classed D by the US Food and Drug Administration; it crosses the placenta and causes fetal bradycardia.

connective tissue disorders such as Loeys-Dietz syndrome.

Case 1. Spontaneous coronary artery dissection

Mrs AS is a 31-year-old woman (gravida 3, para 2) who presents at 33 weeks of pregnancy with central chest discomfort of one hour duration. Her ECG shows a sinus rate of 80 beats per minute, with anterior

T-wave inversion. Her serum troponin level is 70 ng/L (reference range, <15 ng/L). Serial ECGs show anterior ST elevation in leads V2 to V5, I and AVL, associated with increasing chest pain, despite treatment with aspirin, metoprolol and morphine (Figure 2).

An echocardiogram shows anterior hypokinesis, and a coronary angiogram confirms spiral dissection of the proximal LAD artery. She is treated with stenting of the LAD artery, which settles both her pain and the ECG changes. Thereafter, she is continued on aspirin and clopidogrel until a planned caesarean delivery at 37 weeks of pregnancy.

Commentary

A high index of suspicion is required to diagnose SCAD. If symptoms and ECG changes resolve with medical therapy then conservative therapy is preferred as the process of coronary angiography may itself cause propagation of coronary dissection. However, coronary angiography is required if the patient has dynamic ECG changes or ST elevation with ongoing symptoms.

If coronary stenting is necessary then aspirin and clopidogrel must be continued throughout the remainder of the pregnancy, and thereafter for at least 12 months, as recommended for nonpregnant patients. For Mrs AS, clopidogrel would be withheld from four days before delivery. Aspirin can usually be continued in high-risk cardiac cases, and neuraxial anaesthesia is not contraindicated for women who are taking aspirin in labour.

Bare metal stents should be used for percutaneous coronary intervention in pregnancy, because the safety of drug-eluting stents has not been substantiated.

Thrombolytic agents are relatively contraindicated in pregnancy because of increased reports of maternal haemorrhage, preterm delivery, fetal loss, placental abruption and postpartum haemorrhage requiring transfusion associated with their use.⁸

Valvular heart disease

Patients with valvular heart disease have higher rates of maternal mortality and hospital admission than patients with

3. Risk factors for acute coronary syndromes in pregnancy^{5,6}

- Advanced maternal age (over 30 years)
- Diabetes mellitus, smoking, chronic hypertension
- Hyperlipidaemia
- Pre-eclampsia, eclampsia, thrombophilia
- Blood transfusion
- Postpartum haemorrhage

congenital heart disease and require increased surveillance and serial echocardiography during pregnancy to anticipate complications.⁹

In general, right-sided cardiac lesions are better tolerated in pregnancy than left-sided lesions. Similarly, left-sided regurgitant valves are better tolerated than stenotic valves (because of a reduction in severity of regurgitation from reduced systemic vascular resistance).

Ideally, women with severe aortic and mitral stenosis should be offered preconception counselling so that the valve lesions can be treated before pregnancy.

Case 2. Rheumatic heart disease

Mrs PT is a 29-year-old Indigenous woman (gravida 2, para 1) who presents at 34 weeks of pregnancy with new-onset rapid atrial fibrillation (AF), orthopnoea and paroxysmal nocturnal dyspnoea. She has a history of rheumatic heart disease, and a recent echocardiogram showed severe mitral and aortic regurgitation with mild global left ventricular dysfunction.

She is treated with metoprolol, digoxin, furosemide (frusemide) and enoxaparin and reverts to sinus rhythm. Three weeks later, she is readmitted to hospital with ongoing symptoms of heart failure. She undergoes induction of labour, resulting in an uncomplicated vaginal delivery monitored in the intensive coronary care unit. A week later, she proceeds to cardiac surgery with valve replacement using a bioprosthetic aortic valve, mitral valve repair and AF ablation.

Commentary

Rheumatic heart disease is generally an uncommon cause of valvular dysfunction in most parts of the western world, but the rate in Northern Australia remains one of the highest, estimated as 15.0 cases per 1000 head of population.¹⁰

In rheumatic heart disease, the valve most commonly affected is the mitral valve, with mitral stenosis occurring in 90% of cases, mitral regurgitation in 7% and aortic regurgitation in 2.5%.¹¹ If the aortic valve becomes regurgitant secondary to rheumatic heart disease then the mitral valve is usually also involved. Aortic stenosis is rarely caused by rheumatic heart disease in women of childbearing age.

Aortic and mitral regurgitation

Aortic regurgitation is usually well tolerated in pregnancy (WHO risk category II to III, Box 2). An individual's risk depends on the severity of the regurgitation, symptoms and left ventricular function. With preserved left ventricular function, the main cardiovascular complication is arrhythmia. Patients with severe aortic regurgitation and left ventricular dysfunction are at increased risk of heart failure.

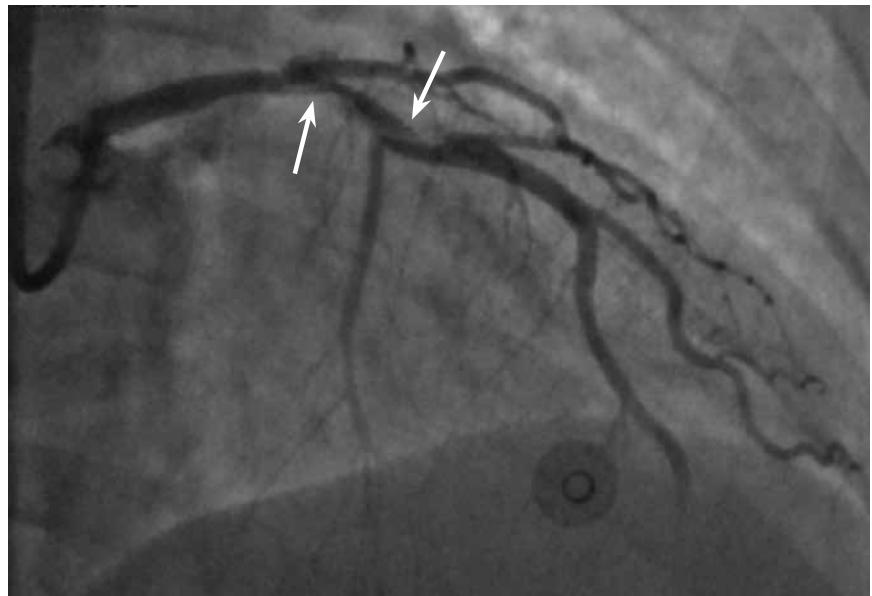


Figure 1. Coronary angiogram showing a spiral dissection (arrows) of the left anterior descending artery in a young woman.

Reproduced with permission from Chaker O, Zied B, Faten J, et al. Management of spontaneous coronary artery dissection causing an acute myocardial infarction in a young female. *Cath Lab Digest* 2013; 21(9): 1,14-15.

Mitral regurgitation is also well tolerated in pregnancy (WHO risk category II to III). As in aortic regurgitation, the main cardiovascular risk is of arrhythmias and heart failure, particularly if there is a degree of left ventricular dysfunction.

Follow up of patients with aortic or mitral regurgitation depends on the individual's status. Vaginal delivery is preferred with epidural anaesthesia and a shortened second stage if women are symptomatic. The risk of peripartum haemorrhage may be increased.

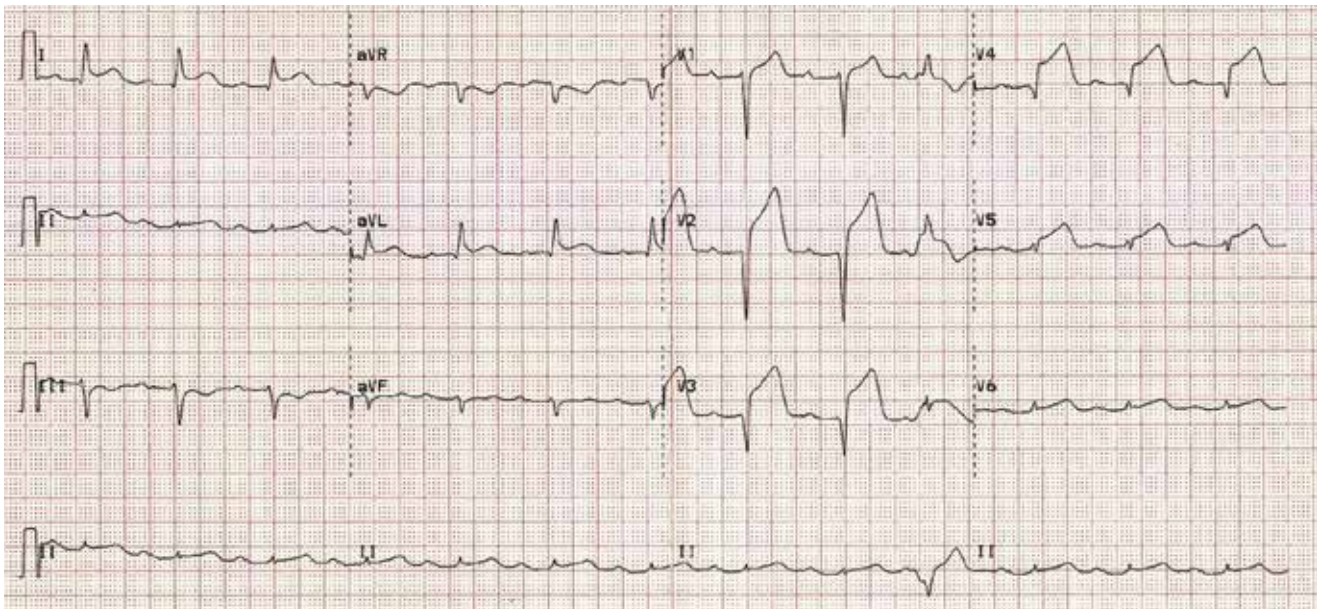


Figure 2. ECG in a patient with acute coronary syndrome, showing progressive ST elevation and Q wave formation in leads V2 to 5, ST elevation in leads I and aVL and some reciprocal ST depression in lead III.

Image courtesy of Dr Ed Burns and Life in the Fast Lane. <http://lifeinthefastlane.com>

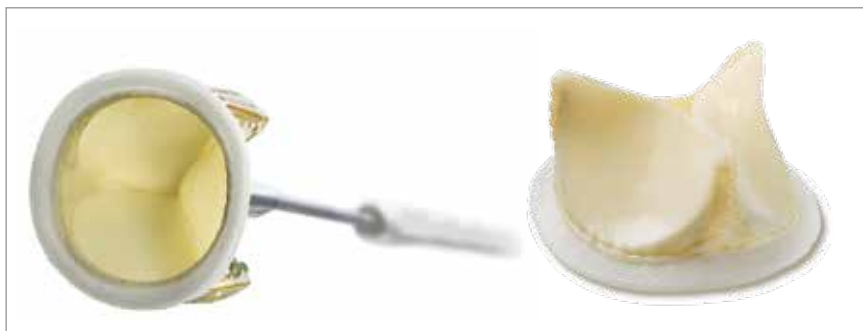


Figure 3. Bioprosthetic heart valves are preferred over mechanical valves in women of childbearing age as they avoid the need for anticoagulation.

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Mitral stenosis

Severe mitral stenosis is the valvular lesion most likely to increase maternal morbidity (WHO risk category IV). Unfavourable maternal outcomes are associated with increased severity (valve area less than 1.5 cm², New York Heart Association [NYHA] functional grade III or IV, significantly elevated right heart pressures with pulmonary artery systolic pressure greater than 50 mmHg and poor NYHA class prior to pregnancy). In addition to the risk of heart failure and arrhythmias, women with mitral stenosis are predisposed to thromboembolism.¹²

Medical treatment of severe mitral stenosis in pregnancy should aim to reduce maternal heart rate with a beta blocker and/or digoxin (if in AF) and to reduce left atrial pressure with judicious use of furosemide. Direct current cardioversion can be safely performed to return patients to sinus rhythm.

Percutaneous mitral balloon valvuloplasty is the treatment of choice for severe symptomatic mitral stenosis if symptoms persist despite optimal medical management. This procedure is usually well tolerated when performed in a tertiary institution with an experienced interventionalist, and fetal mortality is low (0.4% neonatal death).¹³ After valvuloplasty, gradients across the mitral valve are instantly reduced, and patients are usually able to progress to term without significant comorbidities.

Surgical valve replacement should be delayed until the fetus is viable and can be delivered, given fetal mortality rates have

approached 30%.¹³ High fetal mortality resulting from cardiopulmonary bypass relates in part to hypothermia, and if this is avoided and perfusion pressures are maintained at a reasonably high level then fetal mortality rates can be reduced to 10%.¹⁴ Even so, the practice remains to delay cardiac surgery until after delivery of a viable fetus if the mother's health will allow.

Aortic stenosis

Aortic stenosis in women of childbearing age most commonly occurs secondary to a congenital bicuspid aortic valve and may be associated with aortic root dilatation or aortic coarctation. Both of these factors increase the risk of aortic dissection.

Mild-to-moderate aortic stenosis is generally well tolerated, whereas severe aortic stenosis (WHO risk category IV) can lead to haemodynamic and symptom deterioration, resulting in hospitalisation and premature delivery. Despite the increased incidence of morbidity, mortality rates are low. Balloon aortic valvuloplasty is a good option for severe aortic stenosis and is best carried out in the second trimester. This procedure is relatively low risk for both mother and fetus. Definitive aortic valve surgery should be postponed until after delivery of a viable fetus if possible.¹

Prosthetic heart valves and pregnancy

A small proportion of patients with valvular dysfunction have had definitive valve surgery before pregnancy. Women with bioprosthetic valves usually do not require anticoagulation

if they remain in sinus rhythm, which makes these valves an obvious choice for women of childbearing age (Figure 3).

Women with mechanical prosthetic valves require anticoagulation in pregnancy, and their risk of adverse events is considerably higher. The choice of anticoagulant regimen in pregnancy is not straightforward as warfarin offers the best protection against thrombosis but also carries risk for the fetus. Various regimens have been proposed (discussed below). Irrespective of the regimen used, the risks and benefits of each treatment option need to be discussed with the patient, and management should always be in a tertiary centre with close multidisciplinary follow up.

Anticoagulation during pregnancy

The choice of anticoagulation in pregnancy is difficult, as there is no doubt that warfarin offers the best protection against thrombosis for women with mechanical valves (2.4% risk of valve thrombosis and 2% risk of maternal death).^{11,15-17} However, warfarin therapy in pregnancy increases the risk to the fetus of warfarin embryopathy (a constellation of structural birth defects that occur in the first trimester). Warfarin embryopathy appears to be more likely with warfarin doses of 5 mg day or more. Increases in fetal intracranial bleeding persist throughout pregnancy on warfarin.

Whereas warfarin is known to cross the placenta and is classed as category X, neither unfractionated heparin nor low molecular weight heparin (LMWH) cross the placenta and therefore do not increase the risk of teratogenicity or fetal bleeding in pregnancy. LMWH does, however, need dose adjustment during pregnancy and monitoring of anti-factor Xa levels to ensure adequate anticoagulation and prevention of maternal valve thrombosis. Valve thrombosis rates are much lower with LMWH than with unfractionated heparin but are still considerable (9% vs 33%).^{18,19}

Early studies of pregnant women with prosthetic heart valves from the period 1995 to 2004 suggested maternal death rates as high as 2.4%, valve thrombosis in 10.5%, major bleeding in 17.4% and fetal embryopathy in 5%.¹⁵ However, a more recent study

found no episodes of valve thrombosis or thromboembolic complications with prosthetic heart valves when LMWH was used with careful monitoring by repeated measurements of anti-factor Xa levels.¹⁵

Recent guidelines recommend the use of warfarin throughout pregnancy as being the safest management option for women with prosthetic heart valves.¹ A daily dose of warfarin greater than 5 mg/day appears to increase the risk of fetal teratogenicity between 6 and 12 weeks' gestation. For this reason, after thorough counselling, this subset of women should consider changing to unfractionated heparin or LMWH with careful monitoring of anti-factor Xa levels for this period.¹ After 12 weeks' gestation, the safest option to prevent maternal valve thrombosis is to re-commence warfarin and continue throughout pregnancy until the 36th week of gestation. Women taking doses

of warfarin less than 5 mg/day should be able to continue warfarin in the first trimester, with a risk of teratogenicity less than 3%.¹ However, some of these women may prefer to consider unfractionated heparin or LMWH in the first trimester. Careful counselling and informed consent are required.

Anticoagulation in the peripartum period

Regardless of the anticoagulation regimen used earlier in the pregnancy, after the 36th week patients should be managed on LMWH or unfractionated heparin. From 36 hours before induction of labour or planned caesarean delivery, patients should be maintained on unfractionated heparin, which is stopped four to six hours before delivery and restarted four to six hours after delivery if there is no active bleeding. If urgent delivery is necessary then the patient can be given

protamine to partially reverse LMWH, vitamin K (takes four to six hours to have an effect) or fresh frozen plasma to achieve an international normalised ratio of 2 or less.

Cardiomyopathy in pregnancy

Cardiac failure in pregnancy is often difficult to diagnose. The symptoms of exertional dyspnoea and peripheral oedema are common in pregnant women without cardiac disease. Women who develop cardiomyopathy in pregnancy may also complain of palpitations, dizziness, orthopnoea and abdominal discomfort (as a result of hepatic congestion).

A high level of suspicion for cardiac failure is needed in all pregnant women who develop progressive dyspnoea. They may have a pre-existing condition, such as familial, dilated or hypertrophic cardiomyopathy, or may have developed peripartum cardiomyopathy

(in late pregnancy or early postpartum). Alternative causes of dyspnoea in pregnancy should of course also be considered, such as cardiac ischaemia or valvular heart disease, as discussed above, and hypertensive heart disease or pulmonary embolism.

Case 3. Peripartum cardiomyopathy

Mrs KP is a 39-year-old woman (gravida 4, para 4) with type 2 diabetes mellitus. She underwent a normal vaginal delivery at term but is admitted to hospital one week postpartum with orthopnoea, worsening exertional dyspnoea and peripheral oedema. An ECG shows sinus tachycardia (heart rate 110 beats per min), and an echocardiogram shows mild dilatation of the left ventricle with moderate left ventricular systolic dysfunction (ejection fraction, 38%).

She is treated with intravenous furosemide, enalapril and bisoprolol after diuresis is achieved. At follow up in the cardiology clinic six weeks later, she has only mild dyspnoea on marked exertion, and repeat echocardiography shows improvement with residual mild left ventricular systolic dysfunction (ejection fraction, 48%).

Commentary

Peripartum cardiomyopathy is an idiopathic cardiomyopathy resulting in clinical heart failure secondary to left ventricular dysfunction that occurs in the final month of pregnancy or in the months after delivery. The ejection fraction is usually less than 45%.²⁰

The incidence of peripartum cardiomyopathy varies between countries and is thought to be as high as one in 300 in Haiti, and as low as one in 2500 to 4000 in the USA.²⁰ Hypertension, diabetes and smoking are associated with an increased risk of developing peripartum cardiomyopathy. Pregnancy-related factors such as increasing age, multiparity and multiple births also appear to increase the risk.

The pathophysiology of peripartum cardiomyopathy remains ill defined, but various studies have suggested a potential genetic susceptibility or the possibility of an autoimmune basis, with higher titres of autoantibodies against cardiac myosin heavy chains. Particular viruses that cause

peripartum inflammation of cardiac muscle have also been suspected of promoting left ventricular dysfunction in peripartum cardiomyopathy. Other research has focused on the possible role of prolactin in inhibiting cell proliferation, inducing cell apoptosis and impairing cardiac myocyte function.²¹

Differential diagnoses

Patients with pre-existing dilated cardiomyopathy may be asymptomatic until the increased demands on cardiac output in pregnancy unmask underlying left ventricular dysfunction. Differentiating this diagnosis from peripartum cardiomyopathy can be difficult. Patients with dilated cardiomyopathy usually present with symptoms earlier (in the second trimester) than those with peripartum cardiomyopathy, who typically present in the first four months postpartum (78% of cases). Cardiac dimensions may be larger in patients with dilated cardiomyopathy. Peripartum cardiomyopathy remains a diagnosis of exclusion.

Hypertrophic cardiomyopathy can also be unmasked or become more symptomatic in pregnancy. This form of cardiomyopathy is generally well tolerated. Maternal mortality rates in pregnancy for women with hypertrophic cardiomyopathy are mildly elevated above those of the general population but remain low (approximately 10 per 1000 live births).²² Those at particular risk are symptomatic women with moderate-to-severe dyspnoea before pregnancy, a history of syncope, a family history of hypertrophic cardiomyopathy with sudden cardiac death, documented sustained ventricular tachycardia or an elevated resting left ventricular outflow tract gradient of more than 100 mmHg. Women with a strong family history of sudden cardiac death are also at increased risk. The main factors affecting maternal morbidity with hypertrophic cardiomyopathy are worsening NYHA class and arrhythmias.²²

Patient assessment

Early cardiac assessment of women with cardiomyopathy in pregnancy should include an ECG, measurement of B-type natriuretic peptide (BNP) and an echocardiogram.

Cardiac MRI allows more accurate measurement of chamber volumes and ventricular function and assessment of left ventricular thrombus; however, gadolinium (category C) should be avoided until after delivery although it is acceptable while breastfeeding.

Treatment of heart failure in pregnancy

The management of heart failure in pregnancy does not differ markedly from its management in nonpregnant women in regards to use of beta blockers, digoxin, diuretics (usually loop, but thiazide diuretics can be added as second line) and long-acting nitrates. Hydralazine can be used in pregnant women with hypertension and left ventricular systolic dysfunction (moderate to severe) as an alternative to ACE inhibitors or angiotensin receptor blockers, which are contraindicated in pregnancy. Aldosterone antagonists are also not recommended in pregnancy. Anticoagulation is not routinely recommended in women with systolic heart failure as long as they remain in sinus rhythm. Women with atrial fibrillation and moderate-to-severe left ventricular systolic dysfunction, however, should receive anticoagulant therapy to avoid thrombosis.

Conclusion

Physiological changes in pregnancy present a unique challenge to the cardiovascular system in all women. Those with pre-existing or acquired cardiac disease inevitably face even bigger challenges. The importance of management with expert multidisciplinary care and close follow up cannot be overstated. Preconception counselling is also advisable for all women with known cardiac conditions to explain the impact that pregnancy may have on their condition and any potential risks to their children. If all of these measures are addressed then a good outcome can usually be achieved for both mother and child. **CT**

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A list of references is included in the website version of this article (www.cardiologytoday.com.au).

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