Introduction

Heart disease is the primary cause of nonobstetric mortality in pregnancy, occurring in 1-4% of pregnancies\(^1\,^2\) and accounting for 10-15% of maternal mortality in developed countries.\(^1\,^2\) Cardiac disease contributes to 40.2% of maternal deaths in South Africa.\(^4\) Soma-Pillay et al noted that rheumatic valvular heart disease complicated 63.5% of pregnancies in Pretoria.\(^4\) Mitral regurgitation (MR) and mitral stenosis (MS) represent the most common causes of valvular disorders requiring surgery during pregnancy, with an incidence of 27.5% and 20.8%, respectively.\(^4\) Arnoni et al\(^n\) noted that 93% of cardiac surgeries during pregnancy were for valve disease, of which 70% were for mitral valve disease.\(^1\,^5\) Cardiovascular maternal morbidity and mortality during pregnancy correlate strongly with maternal functional status.\(^1\,^2\,^5\) Pregnant women tolerate cardiopulmonary bypass (CPB) surgery as well as non-pregnant women do,\(^2\,^4\,^6\) but foetal morbidity and mortality are high,\(^2\,^6\,^8\) due to increased uterine activity and contractions that reduce placental perfusion.\(^5\,^7\) Untreated maternal heart disease also places the foetus at risk,\(^2\) and if open heart surgery is required, it is best undertaken in the second trimester.\(^2\,^6\,^8\) This case study describes positive maternal and foetal outcomes after CPB surgery in a parturient with severe mitral valve disease.

Case study

A 17-year-old, 28 weeks pregnant patient with a body mass index of 22 kg/m\(^2\) was admitted to the Dr George Mukhari Hospital with a diagnosis of severe MS and moderate MR. Being short of breath and complaining of easy fatiguability, she was classified as New York Heart Association (NYHA) class III and World Health Organization (WHO) heart failure stage C. Her blood pressure was 90/60 mmHg and her heart rate was 82 beats per minute and regular. She had a tapping apex beat, loud first heart sound, loud pulmonary component of the second heart sound and a grade 3/4 diastolic murmur heard loudest at the apex. The echocardiograph confirmed the diagnosis of severe MS with a mitral valvar area of 0.88 cm\(^2\), a mean gradient of 1.85 m/s or 15 mmHg, MR, right ventricular pressure of 42 mmHg and ejection fraction of 52%.

The patient was hospitalised until 33 weeks’ gestation. During this period she was haemodynamically stable, maintaining blood pressures of 80/50 to 90/60 mmHg and heart rates of 60-84 beats per minute, and she did not develop heart failure. Management included bed rest and foetal monitoring by non-stress tests and ultrasound. Penicillin VK 250 mg was administered twice daily for prophylaxis, and she was anticoagulated with enoxaparin 40 mg subcutaneous injection.

Although maximal physiological changes usually occur by 32 weeks and there were no signs of further functional deterioration, the patient’s functional status remained very poor clinically with very limited exercise tolerance, independent of the echo result. This precluded a vaginal delivery, which would have significantly increased her cardiac output and heart rate, exposing the patient to the risk of haemodynamic compromise. Further multidisciplinary discussions took into consideration that Caesarean section does not relieve the haemodynamic stress of the puerperium, with a potential for functional deterioration and relatively
poor outcomes associated with premature foetuses in our institute. The risk-benefit ratio in our setting was therefore weighted towards performing a mitral valve replacement (MVR) prior to delivery. Informed consent for an MVR was provided by the patient’s mother, as the parturient was a minor.

Preoperatively the patient was haemodynamically stable with a blood pressure of 112/69 mmHg and a heart rate of 101 beats per minute. Ultrasonography showed a viable foetus of 33 weeks’ gestation and an estimated weight of 1 900 g. Premedication consisted of diazepam 5 mg, sodium citrate 30 ml orally and intravenous metoclopramide 10 mg. After preoxygenation, general anaesthesia was induced with the patient supine and with left lateral tilt to avoid aortocaval compression, using intravenous fentanyl 5 µg/kg for three to five minutes, followed by etomidate 0.3 mg/kg. Tracheal intubation was facilitated with suxamethonium 1.5 mg/kg. Anaesthesia was maintained using a continuous fentanyl infusion at a rate of 0.1 µg/kg/hour supplemented with midazolam 0.2 mg/kg, vecuronium 20 µg/kg, sevoflurane 1%, and air and oxygen at FiO₂ of 0.7. Intraoperative monitoring included electrocardiography, pulse oximetry, end-tidal carbon dioxide measurement, rectal temperature probe, urinary catheter for fluid balance, intra-arterial blood pressure monitoring and central venous pressure measurement. Foetal heart rate was monitored using cardiotocography. Inotropic support with dobutamine infusion at 5 µg/kg/minute and 500 ml of salvaged blood was administered after weaning from CPB surgery and continued postoperatively in the intensive care unit. Foetal bradycardia was noted during the initiation of CPB surgery, which resolved with restoration of maternal temperature and normalising of circulation. During sternal closure, heparin was neutralised with 180 mg of protamine. The duration of anaesthesia and surgery was three hours and 30 minutes. The patient was extubated after six hours in the intensive care unit, after which she received oxygen by facemask and a six-hourly regimen of subcutaneous enoxaparin 60 mg was begun. She remained haemodynamically stable and the foetal heart rate varied between 130 and 140 beats per minute. Postoperative recovery was uneventful and the postoperative monitoring data are summarised in Table II.

After administering heparin 1 800 IU, we monitored the patient’s anticoagulation status using activated clotting times. The mitral valve was replaced in a CPB surgery time of 50 minutes and an aortic cross-clamp time of 39 minutes. Perfusion flow rates ranged between 3.6–4.1 l/m²/minute. Rectal temperature was 32.5–35.6 °C.

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### Table I: Perioperative parameters for the mother and foetus

| Parameter | Pre-induction | Post-induction | Pre-CPB | CPB | Post-CPB | Conclusion |
|-----------|--------------|----------------|---------|-----|----------|------------|
| bMAP/flow rate | 80 | 85 | 72 | 60–100/3.6-4.1 | 65 | 60 |
| cHR (beats/minute) | 101 | 90 | 76 | 106 | 100 |
| dCVP (mmHg) | 5 | 5 | 12 | 16 | 16 | 17 |
| eFiO₂ (%) | 1.0 | 0.7 | 0.7 | 0.7 | 0.7 |
| fSaO₂ (%) | 99 | 99 | 99 | 99 | 99 |
| gPaO₂ (kPa) | 39.0 | 39.0 | 39.0 | 39.0 | 37.6 |
| hETCO₂ (mmHg) | 36 | 36 | 36 | 32.5–35.6 | 35.7 | 35.5 |
| iTemperature (°C) | 37 | 37 | 36 | 36 | 35.7 | 35.5 |
| jpH | 7.43 | 7.43 | 7.38 | 7.42–7.55 | 7.47 | 7.47 |
| kBE | -3.6 | -3.6 | -5.0 | -0.4–2.0 | 0.5 | 0.5 |
| lPotassium (mmol) | 4.7 | 4.7 | 4.8 | 5.7–8.7 | 4.7 | 4.7 |
| mHaemoglobin (g/dl) | 11.5 | 11.5 | 11.2 | 6.6–8.7 | 8.0 | 8.0 |
| nACT(seconds) | 114 | 114 | 386 | 405–480 | 116 | 116 |
| oFHR (beats/minute) | 110–120 | 110–120 | 110–120 | 50–70 | 130–145 | 130–140 |

### Table II: Postoperative parameters for the mother and foetus

| Parameter | 0 h | 12 h | 24 h | 48 h |
|-----------|-----|------|------|------|
| aMAP (mmHg) | 78 | 70 | 75 | 70 |
| bCVP (mmHg) | 10–14 | 14–18 | 8–13 | 8–13 |
| cPaO₂ (kPa) | 60.6 | 14.1 | 22.6 | 14.1 |
| dPaCO₂ (kPa) | 3.6 | 4.98 | 3.07 | 4.94 |
| pH | 7.43 | 7.43 | 7.42 | 7.42 |
| eBE | -1.1 | -1.2 | -5.5 | -0.1 |
| Potassium (mmol) | 3.6 | 5.1 | 5.9 | 4.1 |
| Lactate (mmol) | 3.8 | 1.5 | 1.8 | 1.2 |
| Haemoglobin (g/dl) | 3.1 | 7.0 | 5.9 | 5.9 |
| Glucose (mmol) | 15–120 | 110–130 | 110–144 | 130–150 |
| fFHR (beats/minute) | 110–120 | 110–120 | 110–120 | 50–70 | 130–145 | 130–140 |

a= mean arterial pressure, b= central venous pressure, c= partial oxygen pressure, d= partial carbon dioxide pressure, e= base excess, f= foetal heart rate
Case Study: Positive maternal and foetal outcomes after cardiopulmonary bypass surgery

Eleven days postoperatively, the non-stress test indicated probable foetal distress and an emergency Caesarean section was performed under general anaesthesia. Neuraxial block was contraindicated, because less than 24 hours had elapsed since the previous dose of enoxaparin. A healthy male baby with a birthweight of 1910 g, a length of 45 cm, a skull circumference of 33.5 cm and Apgar scores of eight, nine and 10 at one, five and 10 minutes, respectively, was delivered. Anaesthesia and surgery were uneventful, as well as the postoperative course. The baby was managed in the neonatal unit for problems regarding prematurity, craniosynostosis and neonatal jaundice. He was subsequently discharged, healthy, 21 days after delivery.

Discussion

Open-heart surgery is best avoided during pregnancy because of many potential adverse effects on mother and foetus.1,2,5-7 These include maternal and foetal death,1,2,5-7 intrauterine growth restriction, low postnatal birthweight and congenital malformations.6 Sustained uterine contractions reduce uterine blood flow (UBF), which results in foeto-placental insufficiency and subsequent foetal hypoxaemia.2,5 Foetal bradycardia, an indicator of foetal asphyxia,2 may occur during CPB surgery initiation and emergence therefrom,1,2,5,7 and may potentially be caused by the following factors: reduced systemic vascular resistance, low UBF, haemodilution, hypoventilation, particulate or air embolism, obstruction of venous drainage during inferior vena cava cannulation, activation of inflammatory processes or maternal narcotic administration.2,5-7 High foetal mortality is attributed to the above factors, which can affect foetal oxygen delivery during CPB surgery.1,5-7 Survival of healthy infants is as high as 56% post-CPB surgery in parturient women with severe cardiac disease.8,9 Intraoperative foetal monitoring can help to correct some of the potential hazards10,11 that result in inadequate foetal oxygen delivery. During CPB surgery in the case study, the haemoglobin values decreased to a minimum of 6.6 g/dl, which could be explained by intraoperative bleeding and haemodilution with subsequent decrease in foetal oxygen delivery. Foetal bradycardia was noted, which resolved with restoration of maternal temperature and normalisation of circulation. Foetal protection strategies included foetal monitoring, a short CPB surgery time, high inspired maternal oxygen concentration (FiO₂ 0.7-1.0), high pump flow rates of 3.6-4.1 l/min and perfusion pressures of 70-100 mmHg (although it momentarily went as low as 60 mmHg), to compensate for the increase in cardiac output that normally occurs during pregnancy.

The indications for cardiac surgery in this case were severe MS (mitral valve area less than 1 cm²) and possible worsening functional status (NYHA class III)1,2 imposed by pregnancy. In general, the “rule of one class” applies; that is, during pregnancy, the patient’s symptomatic status will increase by one NYHA class.13 Patients presenting with severe symptoms (i.e. NYHA classes III and IV) have poor outcomes if treated medically.13

Prevention and treatment of tachycardia is central to the periooperative management of patients with MS.13 During her stay at both the obstetric and cardiothoracic wards, the patient was in sinus rhythm and had a normal pulse rate of 60-84 beats/minute and blood pressure variation of 80/50-90/60 mmHg. The medical therapies for patients with MS in sinus rhythm are relatively limited; however, beta blockers are useful to prevent tachycardia.12 Our patient had an acceptable heart rate range that was unlikely to have limited left ventricular (LV) preload, and therefore a beta blocker was not administered. The increase in heart rate directly preoperatively was attributed to patient anxiety, after other causes for tachycardia, such as infection, had been excluded. Her anxiety was treated with a small dose (5 mg) of diazepam preoperatively, avoiding sedative-induced hypoventilation and jeopardy of the patient’s limited LV preload.13 A short-acting beta blocker such as esmolol might be useful in this situation for heart rate control. However, we were concerned that the use of a beta blocker in our patient, who had a low baseline blood pressure, might cause a significantly reduced cardiac output, compromising organ perfusion. Intraoperatively her heart rate was controlled with a narcotic-based anaesthetic.

Maternal life was prioritised over that of the foetus, considering that there was the possibility that the patient would decompensate severely from the cardiovascular stress imposed by pregnancy6,8,12 with subsequent death of mother or foetus, unless surgery took place. It is generally thought that Caesarean delivery should be reserved for obstetric indications only and that the presence of heart disease should not influence that decision.3 The obstetric approach was also to mature the foetus as far as possible, with a target of 37 weeks, and to avoid complications related to prematurity. Thus, steroids were prescribed for maturation of the foetus’s lungs. The cardiothoracic surgeons’ concerns were those of sepsis and bleeding from the uterus2,8 should a Caesarean section be done prior to open-heart surgery. Uterine atony, resulting from smooth muscle relaxation by inhaled anaesthetics, can be a major cause of uterine bleeding after heparinisation for CPB surgery.2 It is suggested that whenever possible, if the foetus is more than 28 weeks’ gestation, strong consideration should be given to delivering the baby prior to valve replacement surgery, as it is a reasonable and safe procedure.2,4 However, this mainly applies to developed countries or highly resourced institutes with very low maternal and neonatal mortalities related to prematurity. Ultimately, the decision was made to give the foetus a chance to mature further and to operate on the mother, who had the potential to decompensate severely during labour and post partum if surgery was not done soon.
Surgical procedures reported in the literature include closed mitral commissurotomy, open mitral valvotomy, percutaneous balloon mitral valvotomy (PBVM) and MVR.12,14 Although PBVM is preferred for MS in pregnancy, only MVR is performed at our institute. Exact indications for PBVM during pregnancy are not clear cut.14 Some case series have shown that combined Caesarean delivery and PBVM when indicated during pregnancy can be performed with low risk.15 MVR is indicated in cases of mixed mitral valvular disease of rheumatic origin,1 if the valves are calcified and nonpliable and if there is severe pulmonary arterial hypertension.2 To the best of our knowledge, the anaesthetic management of parturient women with viable foetuses of over 28 weeks’ gestation who undergo CPB surgery is uncommon in our setting.

Anaesthesia and surgery during pregnancy occurs in 1.5–2.0% of all pregnancies.10 The overall goal when managing a pregnant patient who undergoes surgery is to maintain foetal and maternal oxygenation and perfusion.11 The basic principles of obstetric management were applied: avoiding aortocaval compression by placing a wedge under the right hip, maintaining high maternal inspired oxygen concentration, maintaining intravascular volume, checking arterial blood gases frequently, monitoring the parturient and foetus and providing acid-aspiration prophylaxis.2,5,8,10 Since the dominant lesion was MS, the anaesthetic management was tailored to avoid tachycardia and pulmonary vasoconstriction, and maintain LV preload without exacerbation of pulmonary vascular congestion. Drugs that cause tachycardia should be avoided. It is important to point out that oxytocin should be administered as a slow infusion in patients with severe MS who require Caesarean section. Because significant LV dysfunction is present in many MR patients, an induction and maintenance agent that avoids further depressing of LV function is often selected.12 For this reason, large doses of narcotics have been popular in the past.13 General anaesthesia provides a very stable haemodynamic course during cardiac surgery and is safe for both the mother and foetus.

**Conclusion**

This case study illustrates that even though CPB surgery is associated with high foetal mortality for parturient women with severe MS, successful outcomes are possible, even in less resourced institutes. Our case also highlights the importance of careful multidisciplinary perioperative planning before embarking on an anaesthetic when a parturient presents with significant cardiac pathophysiology. Balanced anaesthesia using a volatile agent and an opioid provides a stable haemodynamic course during cardiac surgery and is safe for both the mother and foetus.

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**Declarations**

The authors declared no conflict of interest.

**References**

1. Bath WH Jr. Cardiac surgery in pregnancy. Clin Obstet Gynecol. 2009;52(4):630–646.
2. Chandrasekhar S, Cook CR, Collard CD. Cardiac surgery in parturient. Anesth Analg. 2009;108(3):777–785.
3. Amoni RT, Amoni AS, Bonini RC, et al. Risk factors associated with cardiac surgery during pregnancy. Ann Thorac Surg. 2003;76:1065–1068.
4. Soma-Pillay P, Macdonald AP, Mathvha TM, et al. Cardiac surgery in pregnancy: a 4-year audit at Pretoria Academic Hospital. SAMJ. 2008;98(7):553–556.
5. Patel A, Asopa S, Tang ATM, et al. Cardiac surgery during pregnancy. Tex Heart Inst J. 2008;35(3):307–312.
6. Talwar S, Kale SC, Kumar L, et al. Open heart surgery during pregnancy. JTCVS. 2003;19:184–185.
7. Agarwal RC, Bhattacharya PK, Bhattacharya L, Jain RK. Pregnancy and cardiopulmonary bypass. Indian J Anaesth. 2004; 48(4): 259-263.
8. Kuczkowski KM. Anaesthesia for parturient with cardiovascular disease. SAJAA. 2003;9(2):18–25.
9. Avila WS, Gouveria AMM, Pomeranzteff P, et al. Maternal-foetal outcome and prognosis of cardiac surgery during pregnancy. Arq Bras Cardiol. 2009;93(1):8–13.
10. Kuczkowski KM. Nonobstetric surgery during pregnancy: what are the risks of anesthesia? Obstet Gynecol Survey. 2003;58(1):52–56.
11. Cheek T, Baird E. Anaesthesia for nonobstetric surgery: maternal and foetal considerations. Clin Obstet Gynecol. 2009;52(4):535–545.
12. Carabello BA. Modern mitral stenosis. Circulation 2005;112:432–437.
13. Cook DJ, Housmans PR, Rehfeldt KH. Valvular heart disease: replacement and repair. In: Kaplan JA. Essentials of cardiac anaesthesia. 1st edition. Philadelphia: Saunders Elsevier, 2008; p. 327–357.
14. Elkayam U, Bitar F. Valvular disease and pregnancy: native valves. J Am Coll Cardiol. 2005;46(2):223–230.
15. Birincioglu CL, Seref A, Kucuker SA, et al. Perinatal mitral valve interventions: a report of 10 cases. Ann Thorac Surg. 1999;67:1312–1314.
16. Stoebling RK, Hiller SC. Pharmacology and physiology in anesthetic practice. 4th edition. Philadelphia: Lippincott Williams Wilkins; 2006.