Emergency aortic valve replacement and Caesarian section in a primigravida with severe aortic stenosis: a case report

Puneet K Kochhar, V Zutshi, S Shamsunder, S Batra, P Ghosh

Department of Obstetrics and Gynaecology, Lok Nayak Hospital and MAM College, New Delhi, India

Correspondence to
Dr Puneet K Kochhar, F-3/17 Model Town-II, New Delhi 110009, India;
sonia.k20@gmail.com

Accepted 12 July 2011

ABSTRACT

Introduction Congenital bicuspid aortic valve with severe aortic stenosis (AS) is a rare condition (3—6% of patients with congenital heart disease). Pregnancy in these patients carries a high risk of maternal and fetal mortality. With advancing gestational age, these women may develop cardiac failure due to increased cardiorespiratory requirements. When medical therapy proves insufficient, cardiac surgery becomes mandatory to save the patient's life. Balloon valvuloplasty is only palliative treatment, the duration of benefit being only 6 months. Valve replacement is thus recommended. Cardiopulmonary bypass (CPB) surgery with valve replacement has been reported to carry a lower risk of maternal mortality (1.5—13%) but a very high fetal risk (16—40%). This paper reports the case of a 30-year-old primigravida with severe AS with bicuspid aortic valve and pulmonary congestion clinically uncontrolled, in whom CPB surgery and aortic valve replacement was performed as an emergency procedure, along with a lower segment Caesarian section.

Conclusion The outcome of unrelieved severe symptomatic AS in pregnancy is poor. Multidisciplinary management is important to avoid deterioration in cardiac performance in parturients with severe AS. CPB during pregnancy carries a high risk to the fetus. Therefore, open heart surgery during pregnancy should be advised only in extreme emergencies (ie, heart failure refractory to conventional therapy).

A 30-year-old primigravid woman with severe aortic stenosis (AS) presented at 28 weeks of gestation with a 2-week history of palpitations, precordial pain, orthopnoea and dyspnoea on exertion. She had no history of syncopal attacks and had recovered fully from a right-sided hemiplegia following a cerebrovascular accident 3 years earlier. There was no family history of heart disease, hypertension, or dyslipidaemia.

On general physical examination, she was acyanotic and not dyspnoeic at rest. Her height was 1.34 m, weight 40 kg (body mass index 22.3 kg/m^2), her pulse rate was 90/min regular and blood pressure 98/60 mm Hg. She had an ejection systolic murmur (5+/6+) in the aortic region radiating to the carotid arteries. There was equal air entry in both lung fields with no adventitious sounds. On abdominal examination, the uterus was 28 weeks' size, and the fetus was in cephalic presentation with a heart rate of 140 bpm.

Electrocardiography revealed sinus rhythm and hypertrophy of the left chambers. Chest radiography showed cardiomegaly. Echocardiogram showed a bicuspid aortic valve, severe AS, mild left ventricular systolic dysfunction with ejection fraction of 45% and highly calcificied aortic valve (figures 1—4). Haemogram, serum electrolytes, renal and liver function tests were within the normal range. Antistreptolysin-O titre was not raised. Abdominal ultrasound showed a single normal live fetus of 28 weeks' gestation. She was started on oral furesemide 40 mg once a day and cardiology opinion was taken. Intramuscular corticosteroids (betamethasone 12 mg two doses 24 h apart) were given to enhance fetal lung maturation.

At 31 weeks of gestation, the patient developed progressive pulmonary congestion, congestive cardiac failure and hypotension. She required infusions of dopamine and dobutamine to maintain blood pressure at 80/60 mm Hg. Oxygen therapy (fractional inspired oxygen 0.5 at 10—12 l/min) was given by mask. Given that her clinical condition continued to deteriorate, a multidisciplinary team decision was reached to deliver the baby and manage her condition more aggressively including a cardiopulmonary bypass (CPB) surgery with aortic valve replacement.

Endotracheal intubation was performed after establishing an adequate anaesthetic depth and muscle relaxation. Arterial carbon dioxide tension was maintained at less than 40 mm Hg to avoid a possible increase in intracranial pressure associated with hypercapnia. Mean arterial pressure was maintained between 65 and 75 mm Hg. After the induction of general aneasthesia, the fetal heart rate dropped to 80 bpm and a female baby weighing 1500 g was born by Caesarean section with an Apgar scores of 5, 7 and 7 at 1, 5, and 10 min, respectively. The baby was resuscitated and transferred to the neonatal intensive care nursery. The patient's abdomen was kept open to deal with any postpartum haemorrhage during the CPB as heparin was being injected during the valve replacement surgery. Prophylactic B-Lynch uterine compression sutures were placed to prevent uterine atony.

Following closure of the uterus, CPB was started with a pulsatile flow at a rate of approximately 2 l/min. The stenotic aortic valve was replaced with a mechanical valve (25 mm St Jude Medical aortic heart valve St. Jude Medical, St. Paul, MN, USA). CPB time was 145 min and aortic cross clamp time was 90 min.The patient was transfused 6 units of whole blood, 4 units of fresh frozen plasma and 4 units of platelet concentrate perioperatively.

Postoperatively, she was maintained on vasopressors for 48 h, and extubated on the third postoperative day. Warfarin was used for

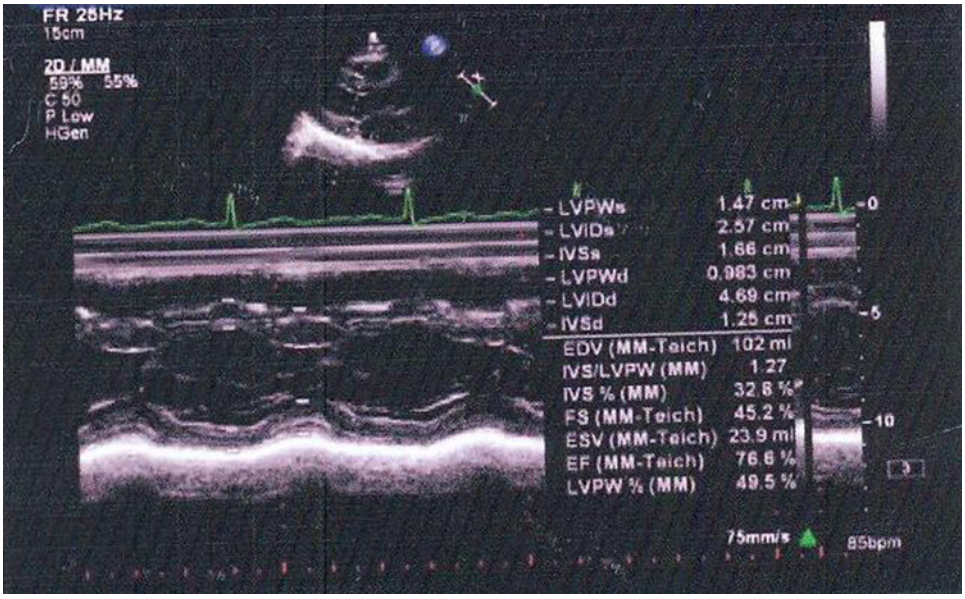

Figure 1 M-mode view of the heart.

anticoagulation, maintaining an international normalised ratio of between 2.5 and 3.5. Abdominal sutures were removed on the tenth day postoperatively. Intravenous antibiotics were continued for 14 days after which she was discharged from the cardiac coronary care unit on warfarin, ecosprin and diuretics. She was transferred to the obstetric ward as the baby was still in the nursery. On the 17th day after surgery, she had an episode of generalised convulsions. Physician referral was taken and tTab. phenytoin 300 mg HS was started. Contrast-enhanced CT (head) showed gliosis in the third ventricle. There was no fresh infarct or haemorrhage. Echocardiography showed no vegetation or clot. All other investigations were within normal limits.

The newborn infant had bronchopneumonia and patent ductus arteriosus but both the mother and baby were discharged home on the 25th postoperative day.

DISCUSSION

Heart disease is the most important cause of non-obstetric mortality in pregnancy. Among women of childbearing age, 2–4% are found to have concomitant heart disease.[1] While congenital heart disease (CHD) is more common in the western world, acquired heart disease (rheumatic valvular heart disease) is more often found to affect women in Asia.[2] Mitral stenosis is the most common lesion seen. Significant AS (acquired or congenital) is uncommon in this age group.

Severe AS with congenital bicuspid aortic valve is found in 3–6% of patients with CHD.[3] The turbulent blood flow results in thickening, fibrosis, calcification and stiffness of the valve leading to clinical manifestations from the third decade of life. Earlier diagnosis of AS has been facilitated with the routine use of echocardiography.[4]

Haemodynamic changes in pregnancy with AS

Pregnancy induces a 20–100% expansion of the blood volume, a 30–40% increase in cardiac output and an increase in heart rate of 10–20 bpm. Increase in cardiac output is limited in patients with severe AS because of obstruction of the left ventricular outflow.[5] This may lead to haemodynamic decompensation (particularly if the valve area is $<1\ cm^2$), which frequently manifests as symptoms and signs of pulmonary congestion, syncope and sudden death. Therefore, these patients

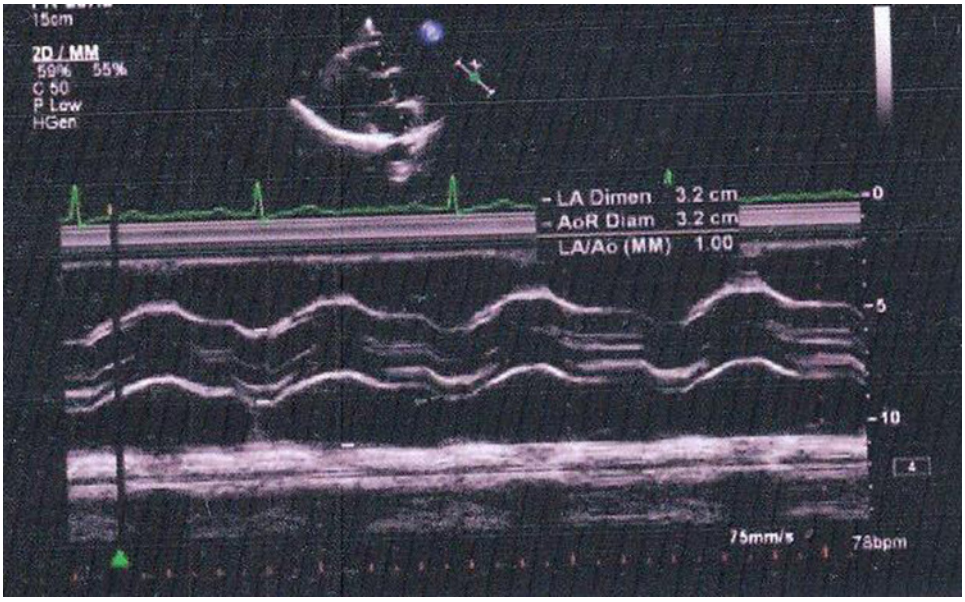

Figure 2 M-mode view of heart at the level of aortic valve.

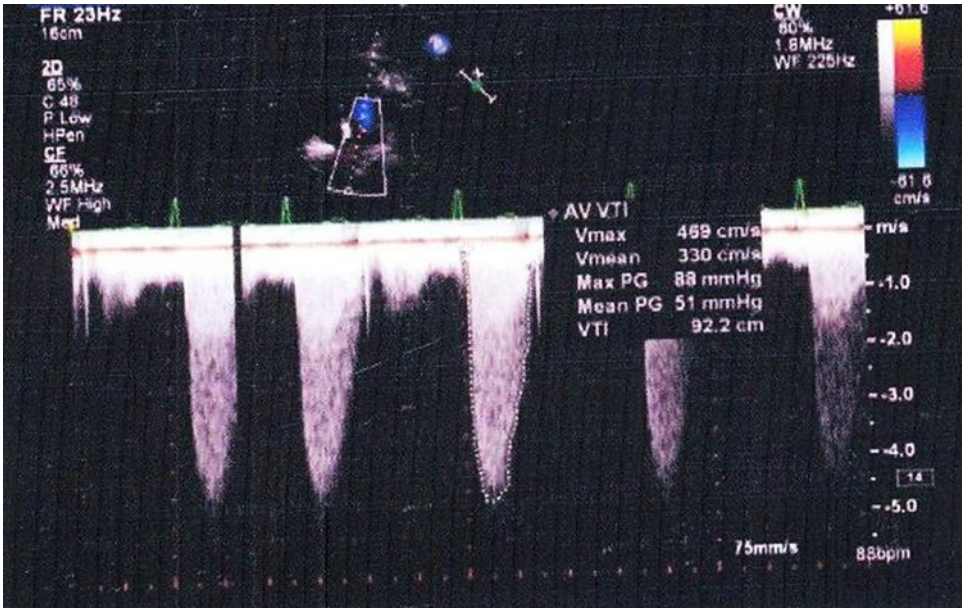

Figure 3 Continuous wave Doppler of aortic valve suggestive of severe aortic stenosis.

are extremely sensitive to small alterations in ventricular volumes, arterial pressure, cardiac frequency and rhythm. The outcome of unrelieved severe symptomatic AS in pregnancy is poor. In late pregnancy, compression of the inferior vena cava by the gravid uterus in the supine position decreases venous return and further reduces cardiac output in these patients. Finally, the hypercoagulable state associated with pregnancy can increase the risk of thrombosis in diseased or prosthetic valves.

Reported outcome

In a recent literature review, significant cardiac complications were seen in 11% of 2491 pregnancies among women with structural CHD.[6] Hypertensive disorders and thromboembolic events were the only obstetric complications found to be more prevalent. Premature delivery rates ranged from 22% to 65%, and the neonates were small for gestational age.

However, pregnancy in patients with severe AS has been reported very infrequently, thus there is no consensus about the ideal management. In 1978, Arias and Pineda,[7] analysing 38 gestations with 23 severe aortic stenoses, reported a maternal mortality rate of 17.4% for untreated patients and a fetal mortality rate of 34%. Attempted therapeutic abortion was associated with 40% maternal mortality. Other reported adverse outcomes include spontaneous abortion, small for dates, prematurity, low Apgar scores, neonatal hypoglycaemia, bradycardia, impaired response to anoxic stress and an increased frequency of CHD in the infant.[8]

An invasive cardiac intervention may be necessary if the clinical condition deteriorates during pregnancy. The development of dyspnoea, syncope, angina and arrhythmias indicate critical left ventricular outflow obstruction. Surgical intervention (balloon valvuloplasty or valve replacement) must be considered in these cases.

Cheitlin[9] recommended surgical balloon valvuloplasty or surgical valve replacement in the presence of symptoms, immediately if evidence of pulmonary congestion exists, and when the valvular area is 0.7 cm^2 or lower, measured on the echocardiogram or by cardiac catheterisation.

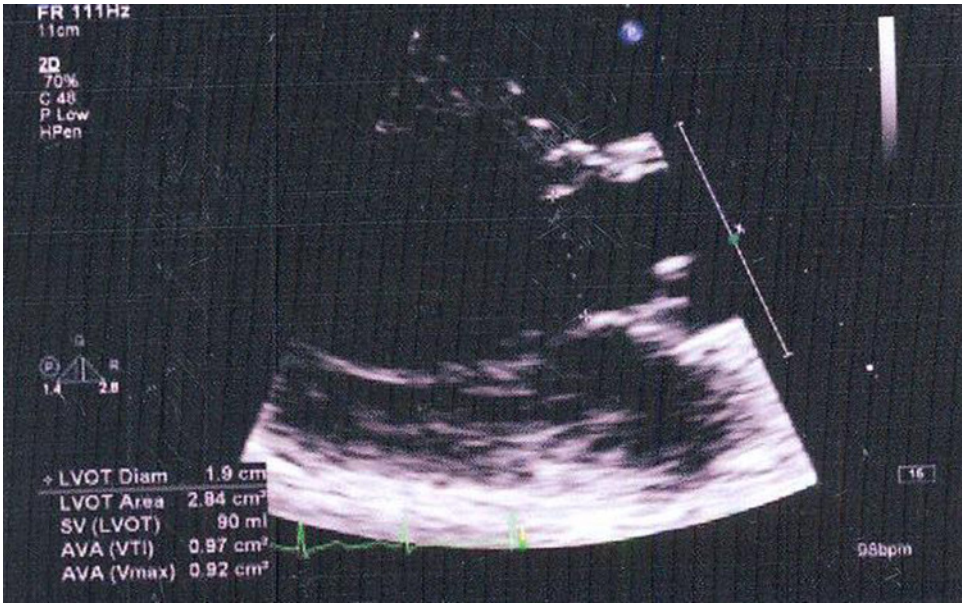

Figure 4 Two-dimensional echo at the level of aortic valve.

However, balloon valvuloplasty has late results, with the duration of benefit lasting approximately 6 months, and is thus a palliative treatment only. The immediate appearance of aortic regurgitation or its progression and the later appearance of restenosis are the major complications of valvuloplasty. Other complications during the procedure include bleeding, arrhythmias, stroke, iliac-femoral arterial complications, injury to mitral valve and death.[3] Exposure to x-rays during the procedure is another potential problem. Therefore, valve replacement is recommended.[10]

CARDIOPULMONARY BYPASS

Effect on the mother

The effects of CPB are relatively well tolerated by the mother. Haemodilution, changes in coagulation, complement activation and hypotension during CPB may have a deleterious effect. However, in two studies on patients undergoing aortic valve replacement, no maternal death was reported, although the overall maternal surgical mortality rate with CPB has been reported to be 1.5.[11 12] With improvements in cardiac surgery and anaesthesia, the maternal risk of surgery is only marginally higher than the risk of CPB in a non-pregnant patient. The functional class of disease is the factor most commonly predicting a higher risk of maternal death.[13] Other risk factors for maternal mortality during cardiac surgery include the use of vasoactive drugs, age, type of surgery and re-operation.[13]

Effect on the fetus

During first two trimesters the incidence of teratogenesis is high. During the third trimester, with improvements in the outcome for premature infants with modern neonatal intensive care, delivery of the child immediately before commencing CPB is a safe option.[14] If this is inappropriate, high-flow, high-pressure, normothermic bypass for as brief a period as possible should be used.

The initial response to CPB is fetal bradycardia, which can be corrected by increasing the perfusion rate. In most cases, the decrease in heart rate reverts to sinus rhythm on restoration of maternal circulation after cessation of CPB.[15–18] This bradycardia is likely to be caused by reduced blood flow to the intervillous spaces resulting in fetal hypoxia and acidosis.[19] The various causes postulated include maternal hypothermia, hypotension, haemodilution, reduced uterine perfusion pressure, fetoplacental dysfunction, uterine contractions and drugs that cross the placental barrier, such as β-adrenergic blockers.[19] When CPB is prolonged, the fetus may develop sustained bradycardia.

Maternal core temperature also affects the fetal response during CPB. More severe hypothermia results in reduced perfusion and oxygen delivery to fetal organs. This induces a decline in fetal and placental function and increases the risk of fetal arrhythmias and cardiac arrest.[15 20]

Alterations in fetal heart rate may be observed even when maternal circulation, acid-base balance and perfusion pressure are stable. These may be related to the narcotic effect of anaesthetic drugs.[19]

The overall fetal surgical mortality has been reported to be 16–33%,[12 21 22] with no correlation to gestational age. Factors affecting fetal mortality are functional class, type of procedure, emergency surgery, re-operation, anoxic time and fetoplacental response to CPB.[13]

Therefore, monitoring of the fetal heart rate is essential as it assists in the early recognition of the potentially deleterious effects of CPB, thus enabling timely intervention to minimise fetal mortality.

Effect on the uterus

Uterine contractions occur in response to bypass, possibly due to a dilutional effect from the stabilising influence of progesterone. Sustained uterine contractions reduce uterine blood flow and intervillous perfusion, resulting in fetoplacental insufficiency and fetal hypoxia. Uterine monitoring is essential to allow the early control of these contractions as they are associated with significant fetal loss.

Various techniques to modify this include the administration of progesterone, β 2-agonists and intravenous alcohol, all with some effect.[14] The use of tocolytic therapy with ritodrine has been reported to prevent early labour, resulting in a favourable fetal outcome.[23 24] However, more recent evidence suggests that there is no benefit.[25] In fact, β-agonists increase myocardial work and oxygen demand in a circulation already compromised by the burden of pregnancy and the initiation of CPB.

Increased sustained uterine contractions are also associated with both the cooling and rewarming phases.[12] This is supported by the fact that there is a notably higher fetal mortality rate with hypothermic than with normothermic CPB.[15]

MANAGEMENT APPROACH

Women with severe AS require close monitoring by their obstetricians and cardiologists, especially during the third trimester, labour and delivery, and the early postpartum period. Good maternal and fetal outcome may be achieved through early preoperative detection of maternal cardiovascular decompensation, use of fetal monitoring, delivery of a viable fetus before CPB and scheduling surgery on an elective basis during the second trimester. Administration of maternal corticosteroids is recommended to initiate endothelial membrane stability and maturation of the fetal lungs, which can substantially improve fetal outcome. In addition, fetal morbidity may be reduced during CPB by optimising maternal oxygen-carrying capacity and uterine blood flow.

Current maternal bypass recommendations include:[26]

1. Maintaining a high pump flow rate greater than 2.5 l/min/m^2 and perfusion pressure greater than 70 mm Hg (to maintain uterine blood flow);
2. Maintaining the haematocrit at greater than 28% (to optimise oxygen-carrying capacity);
3. Using normothermic perfusion when feasible;
4. Using pulsatile flow (may better preserve uterine blood flow); and
5. Using α-stat pH management (for maintenance of carbon dioxide homeostasis as hypocapnia causes uteroplacental vasoconstriction).

The principles for the management of pregnant patients undergoing cardiac surgery with CPB include maintenance of maternal wellbeing, avoidance of teratogenic drugs, avoidance of intrauterine hypoxia and prevention of premature labour.[19] The chief concerns during CPB are the control of temperature, perfusion pressure and nature of the bypass flow. Current evidence favours maintaining normothermic CPB, avoiding the use of vasoconstrictors (which may have a profound effect on the placental unit), and maintaining both high haematocrit and high flow rates.[19]

PERIOPERATIVE MONITORING

Fetal monitoring

If the fetus is viable, intraoperative fetal monitoring is indicated, with a preoperatively agreed plan of action with regard to how a non-reassuring tracing will be managed. Persistent fetal

bradycardia should be treated aggressively by optimising maternal oxygen saturation, correcting any acid-base abnormalities, increasing CPB blood flow, minimising anaesthetic depth, replenishing fetal glycogen stores (avoiding maternal hypoglycaemia) and delivery by Caesarian section.[26]

Uterine monitoring

Monitoring of uterine contractions can also be performed. If uterine contractions are detected, maternal intravascular volume can be increased and tocolytic treatment may be given.

Invasive haemodynamic monitoring

Severe AS is a known indication for pulmonary artery catheterisation in the parturient undergoing cardiac surgery; (other indications are New York Heart Association class 3 or 4 heart disease, cardiac failure, refractory pulmonary oedema and preeclampsia with refractory oliguria or pulmonary oedema).[26]

USE OF ANTICOAGULANT IN CPB IN PREGNANT WOMEN

Oral anticoagulation with warfarin has the lowest incidence of maternal mortality and thromboembolism. However, in the first trimester, warfarin can cause fetal growth retardation, spontaneous abortion and embryopathy. In addition, warfarin is associated with premature birth and fetal and placental haemorrhage in the third trimester.[27] For the purposes of CPB, heparin may be safely used in the usual doses for anticoagulation without risk to the fetus.[28] Post-delivery, warfarin has been found to be safe in breast-feeding women (despite minimal secretion in breast milk), with no reported adverse neonatal effects.

OBSTETRIC MANAGEMENT DURING CPB

When the fetus is not viable, the mother's condition takes priority. If indicated, cardiac surgery should be performed after informing the parents that the fetus may tolerate CPB poorly. In contrast, when the baby is viable, it may be in the best interests of the mother and baby to perform a Caesarean delivery before the cardiac operation, with a neonatologist available for resuscitation, because of the high fetal mortality associated with cardiac surgery. In such situations, delivery by Caesarean section using general anaesthesia is preferred, making sure the mother is adequately hydrated, adequately replacing blood loss and maintaining adequate ventricular filling pressures.

However, a Caesarean section may affect the subsequent cardiac surgery. For example, uterine atony because of smooth muscle relaxation by inhaled anaesthetics can be a major cause of bleeding after heparinisation for CPB. Therefore, if the patient is to be subsequently heparinised, instead of volatile anaesthetics, total intravenous anaesthesia (eg, etomidate and opioids) should be used.[28] Average blood loss for a Caesarean delivery is approximately 1000 ml. This may need to be replaced before subsequent cardiac surgery. Oxytocin, used for uterine atony, can cause systemic hypotension if given rapidly (by exacerbating the hypovolaemia caused by blood loss). Therefore, it should always be given as an infusion and not a bolus.

Even though uterine bleeding may appear to be clinically controlled, the recurrence of bleeding may occur after heparinisation for CPB. The uterus should thus be frequently checked for adequate haemostasis. Uterine compression sutures (such as the B-lynch suture) may help in controlling atonic postpartum haemorrhage.

CONCLUSION

The outcome of unrelieved severe symptomatic AS in pregnancy is poor. Multidisciplinary assessment and careful anaesthetic planning is important to avoid deterioration in cardiac performance in parturients with severe AS. Although the valve lesion can be corrected surgically before delivery at a low risk to the mother, CPB during pregnancy carries a high risk to the fetus. Therefore, open heart surgery during pregnancy should be advised only in extreme emergencies (ie, heart failure refractory to conventional therapy).

Competing interests None to declare.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. **Khandelwal M,** Rasanen J, Ludormirski A, *et al*. Evaluation of fetal and uterine hemodynamics during maternal cardiopulmonary bypass. *Obstet Gynecol* 1996;**88**:667–71.
2. **Abbas AE,** Lester SJ, Connolly H. Pregnancy and the cardiovascular system. *Int J Cardiol* 2005;**98**:179–89.
3. **Tumelero RT,** Duda NT, Tognon AP, *et al*. Percutaneous balloon aortic valvuloplasty in a pregnant adolescent. *Arq Bras Cardiol* 2004;**82**:98–101.
4. **Friedman WF.** Congenital heart disease in infancy and childhood. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. 5th edn. Philadelphia: WB Saunders Co, 1997;**29**:877–962.
5. **Banning AP,** Pearson JF, Hall RJ. Role of balloon dilatation of the aortic valve in pregnant patients with severe aortic stenosis. *Br Heart J* 1993;**70**:544–5.
6. **Drenthen W,** Pieper PG, Roos-Hesselink JW, *et al*. Outcome of pregnancy in women with congenital heart disease: a literature review. *J Am Coll Cardiol* 2007;**49**:2303–11.
7. **Arias F,** Pineda J. Aortic stenosis and pregnancy. *J Reprod Med* 1978;**20**:229–32.
8. **Oakley GD,** McGarry K, Limb DG, *et al*. Management of pregnancy in patients with hypertrophic cardiomyopathy. *Br Med J* 1979;**1**:1749–50.
9. **Cheitlin MD.** The timing of surgery in mitral and aortic valve disease. *Curr Probl Cardiol* 1987;**12**:112–23.
10. **Hall RJC,** Kirk R. Balloon dilatation of heart valves. *Br Med J* 1992;**305**:487–8.
11. **Ben-Ami M,** Battino S, Rosenfeld T, *et al*. Aortic valve replacement during pregnancy. *Acta Obstet Gynecol Scand* 1990;**69**:651–3.
12. **Becker RM.** Intracardiac surgery in pregnant women. *Ann Thorac Surg* 1983;**36**:453–8.
13. **Arnoni RT,** Arnoni AS, Bonini RC, *et al*. Risk factors associated with cardiac surgery during pregnancy. *Ann Thorac Surg* 2003;**76**:1605–8.
14. **Parry AJ,** Westaby S. Cardiopulmonary bypass during pregnancy. *Ann Thorac Surg* 1996;**61**:1865–9.
15. **Pomini F,** Mercogliano D, Cavalletti C, *et al*. Cardiopulmonary bypass in pregnancy. *Ann Thorac Surg* 1996;**61**:259–68.
16. **Koh KS,** Friesen RM, Livingstone RA, *et al*. Fetal monitoring during maternal cardiac surgery with cardiopulmonary bypass. *Can Med Assoc J* 1975;**112**:1102–4.
17. **Pardi G,** Ferrari MM, Iorio F, *et al*. The effect of maternal hypothermic cardiopulmonary bypass on fetal lamb temperature, hemodynamics, oxygenation, and acid-base balance. *J Thorac Cardiovasc Surg* 2004;**127**:1728–34.
18. **Werch A,** Lambert HM, Cooley D, *et al*. Fetal monitoring and maternal open heart surgery. *South Med J* 1977;**70**:1024.
19. **Patel A,** Asopa S, Tang AT, *et al*. Cardiac surgery during pregnancy. *Tex Heart Inst J* 2008;**35**:307–12.
20. **Hess OW,** Davis CD. Electronic evaluation of the fetal and maternal heart rate during hypothermia in a pregnant woman. *Am J Obstet Gynecol* 1964;**89**:801–7.
21. **Chambers CE,** Clark SL. Cardiac surgery during pregnancy. *Clin Obstet Gynecol* 1994;**37**:316–23.
22. **Bernal JM,** Miralles PJ. Cardiac surgery with cardiopulmonary bypass during pregnancy. *Obstet Gynecol Surv* 1986;**41**:1–6.
23. **Lamb MP,** Ross K, Johnstone AM, *et al*. Fetal heart monitoring during open heart surgery. Two case reports. *Br J Obstet Gynaecol* 1981;**88**:669–74.
24. **Mooij PN,** de Jong PA, Bavinck JH, *et al*. Aortic valve replacement in the second trimester of pregnancy: a case report. *Eur J Obstet Gynecol Reprod Biol* 1988;**29**:347–52.
25. **Dodd JM,** Reid K. Tocolysis for assisting delivery at caesarean section. *Cochrane Database Syst Rev* 2006;(4):CD004944.
26. **Chandrasekhar S,** Cook CR, Collard CD. Cardiac Surgery in the Parturient. *Anesth Analg* 2009;**108**:777–85.
27. **Chan WS,** Anand S, Ginsberg JS. Anticoagulation of pregnant women with mechanical heart valves: a systematic review of the literature. *Arch Intern Med* 2000;**160**:191–6.
28. **Van De Velde M,** De Buck F. Anesthesia for non-obstetric surgery in the pregnant patient. *Minerva Anestesiol* 2007;**73**:235–40.