Case Study: Anaesthetic management of a parturient with severe pulmonary stenosis undergoing Caesarean section

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Abstract
We report the successful management of a parturient with severe pulmonary stenosis undergoing Caesarean section. Anaesthesia was managed with combined spinal and epidural anaesthetic technique. During the intraoperative period, haemodynamic parameters were well maintained. There were no episodes of haemodynamic fluctuations or oxygen desaturation. The patient delivered a full-term, normal foetus.

Introduction
The presence of valvular heart disease due to both congenital and acquired aetiologies in pregnant patients continues to pose a challenge to clinicians. The choice of the various anaesthetic techniques used for Caesarean section has different impacts on the underlying cardiac status of these patients. Only limited data, however, are available in literature to guide the anaesthetic management of parturient patients with severe pulmonary stenosis (PS).\(^1,^2\) This report describes the successful anaesthetic management of pregnant patients with severe PS undergoing Caesarean section.

Case report
A 22-year-old patient, gravida 2 para 1, presented to the gynaecological emergency department at 26 weeks of gestation with shortness of breath and easy fatigability (New York Heart Association [NYHA] class III). She had received no prior antenatal care and had no previous medical history of note. Her obstetric history included having delivered a live infant from an unsupervised, uneventful pregnancy 18 months previously.

Examination revealed a Grade 4/6 ejection systolic murmur best heard in the second left intercostal space with a delayed pulmonic component of the second heart sound. The patient’s heart rate was 80 beats per minute. The echocardiography showed severe valvular PS with a peak systolic gradient of 85 mmHg, a mild mitral valve prolapse, an ejection fraction of 60% and normal systolic function. The patient’s biochemical profile revealed elevated thyroid-stimulating hormone with low free thyroxine and free triiodothyronine.

The patient was therefore started on 50 μg of oral thyroxine together with 10 mg of amiloride and 40 mg of furosemide. The patient’s symptoms improved on medical therapy and the subsequent antenatal period was uneventful. She was readmitted for observation at 36 weeks, when the decision was made to perform an elective Caesarean section at 37 weeks.

The preoperative anaesthetic examination revealed an NYHA class II patient with a heart rate of 90 beats per minute and blood pressure of 100/70 mmHg. Jugular venous pressure was not raised, airway examination was unremarkable and the patient’s haematological profile was normal. The electrocardiogram (ECG) showed sinus rhythm with right-axis deviation. 50 mg of oral ranitidine and 10 mg of oral metoclopramide were given, together with 50 μg of oral thyroxine, on the morning of surgery.

In the operating room, non-invasive blood pressure monitoring, a 5-lead ECG with ST segment analysis and pulse oximetry were applied. The patient’s
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Oxygen saturation, breathing room air, was 98%. Intravenous access was secured with an 18G cannula. The patient's blood pressure was 130/80 mm Hg and heart rate was 88 beats per minute. Invasive blood pressure monitoring was established via left radial artery cannulation under local anaesthesia. Central venous cannulation of the right internal jugular vein was performed. Co-loading with 200 ml of lactated Ringers was done to maintain a baseline central venous pressure of 12 mm Hg. An 18G epidural catheter was inserted at the L3-L4 level in the left lateral position. The subarachnoid space was located using a 26G Quincke needle and 2.5 mg (0.5 ml of 0.5%) of hyperbaric bupivacaine and 25 μg of fentanyl were administered intrathecally. A prophylactic phenylephrine infusion was started at 20 μg/min.

The patient was subsequently placed in the supine position with a left lateral tilt and slightly head-up position. Oxygen supplementation (FiO₂ = 0.5) was administered via a venturi mask. A T10 sensory block to pinprick was achieved; this required augmentation. An amount of 3 ml of lignocaine 2%, with adrenaline 1:200 000, was given, followed by a 20 mg (4 ml of 0.5%) bolus of isobaric bupivacaine administered epidurally five minutes later. This resulted in a loss of sensation to pinprick up to T4. There were no episodes of hypotension or desaturation. Haemodynamic parameters were well maintained throughout the intraoperative period.

A healthy infant weighing 2.5 kg was delivered with Apgar scores of eight and nine at one and five minutes, respectively. Intravenous infusion of 3.0 IU/hour of oxytocin was then started. The remainder of the intraoperative course was uneventful. Postoperative analgesia was maintained for the following 24 hours with intermittent boluses of 10 mg (8 ml of 0.125%) of isobaric bupivacaine.

Discussion

Isolated PS during pregnancy is most commonly due to congenital obstruction at valvular level.³ Despite limited case reports of pregnant patients with PS, available evidence nevertheless seems to suggest that, in contrast to other stenotic lesions, the severity of PS does not significantly impact on maternal or foetal outcome.⁴ Few investigators, however, have attempted to classify the various cardiac lesions in pregnancy based on the risk of maternal death or severe morbidity. They nevertheless considered PS as a low-risk factor (with mortality of 0.1% to 1.0%) and suggested that progression to severe PS increases the risk of maternal death to between 1% and 5% (an intermediate risk).⁵ Morbidity and mortality in a pregnant patient with underlying cardiac disease, however, correlate more strongly with functional status; NYHA classes III and IV are considered as high-risk factors for maternal mortality (5% to 30%).⁵ ⁶

The anaesthetic management of these women at delivery requires an understanding of the pathophysiology of the valvular defect. The impact of the normal cardiovascular changes of pregnancy and the anaesthetic technique can then be anticipated.

Pulmonary artery stenosis increases intraventricular pressure and the work of the right ventricle; the right ventricle adapts readily to the situation and maintains output until late in the course of the disease. Pregnancy-induced increase in intravascular volume and heart rate, however, can precipitate right ventricle failure. As the gradient of stenosis increases to more than 80 mmHg, left ventricular preload and thus cardiac output decrease. Systemic vascular resistance (SVR) usually increases to maintain blood pressure. Pregnancy-related decrease in SVR may counteract this compensatory mechanism. Preload may also be reduced during pregnancy as a result of aortocaval compression and at delivery due to vasodilatation associated with neuraxial sympathetic block.

When considering these patients for anaesthesia, it is important to maintain right ventricle filling pressures for effective ventricular contraction in response to increased pulmonary valve stenosis. Excessive preload can furthermore precipitate right heart failure and atrial arrhythmias.⁷ ⁸ Right ventricle output also depends on heart rate, as stroke volume is relatively fixed. A decrease in SVR can also decrease blood pressure and cardiac output in patients with PS. Resulting hypotension can then lead to foetal asphyxia and acidosis through a decrease in uterine blood flow. The goal is therefore to maintain baseline haemodynamic status.⁹ A good anaesthetic technique should thus maintain normal heart rate while avoiding marked decreases in SVR and myocardial depression. With the above goal in mind, a combined spinal epidural (CSE) technique was selected.

Our patient was NYHA class II, having had a previous normal vaginal delivery. We chose to undertake a sequential CSE technique because of the advantage of spinal anaesthesia, with a rapid-onset, low-level block with the flexibility of a further extension of the block with an epidural catheter. Even though Bray, Fernando, Patel and Columb found no benefit in...
terms of the cardiovascular stability of sequential-to-standard CSE for elective Caesarean delivery in healthy pregnant patients, women in high-risk populations (pre-eclampsia, cardiac failure, fixed cardiac output states) in whom the use of fluid or vasopressors may be restricted may still benefit from a sequential CSE technique.10 This technique has also been shown to produce better analgesia and muscle relaxation and is associated with decreased total drug usage and less hypotension when compared to epidural anaesthesia for Caesarean section. We furthermore used a very small dose of local anaesthetic (LA) intrathecally to minimise hypotension.

After careful induction of spinal anaesthesia, we administered Ringer’s lactate and started a prophylactic infusion of phenylephrine. We used 7 ml of LA sequentially via the epidural catheter to achieve a sensory level of T4. Increased sensitivity or enhanced diffusion of LA to membrane receptor sites,11–13 with reduced size of epidural space due to increased epidural venous engorgement, may be the cause of lesser dose requirement in pregnancy.14 Regional anaesthesia also avoids hazards related to the management of a difficult airway and to haemodynamic responses associated with laryngoscopy. Invasive arterial pressure monitoring was performed to facilitate early recognition of blood pressure changes. Central venous catheterisation was done to guide fluid therapy and maintain right-ventricle filling pressures while avoiding excessive preload.

Our review of literature revealed only three case reports of the anaesthetic management of parturient patients with severe PS.1,2,15 Epidural anaesthesia with the use of incremental boluses of 0.5% of bupivacaine was used successfully for Caesarean section in a woman with Watson’s syndrome who had PS.1 Analgesia for labour and delivery was provided with an intrathecal sufentanil infusion, followed by a 15 mg bolus of 1% of lignocaine for vacuum extraction, in another parturient patient with isolated severe PS.2 Campbell, Rosaeg and Chan described the anaesthetic management of a parturient patient with PS and aortic incompetence with the use of epidural anaesthesia:15 a total of 15 ml of lignocaine 2%, with epinephrine 1:200 000, resulted in loss of sensation to pinprick to T10 and a further 5 ml of solution resulted in profound hypotension and junctional arrhythmias. To the best of our knowledge, there is no description of the use of a CSE technique in the management of Caesarean section in a parturient patient with isolated PS.

In conclusion, severe PS in pregnancy places patients in the intermediate-risk category of cardiac complications.6 These patients, presenting for Caesarean section, can be managed successfully with sequential CSE. This may have the advantage of avoiding haemodynamic alterations associated with a single-shot anaesthetic technique.

References

1. Conway JB, Posner M. Anaesthesia for Caesarean section in a patient with Watson’s syndrome. Can J Anaesth. 1994; (11):1113–6.
2. Ransom DM, Leicht CH. Analgesia with sufentanil for labor and delivery in a parturient with severe pulmonary stenosis. Anesth Analg. 1995;80(2):418–21.
3. Warnes CA, Elkayam U. Congenital heart disease in pregnancy. In: Elkayam U, Gleicher N, editors. Cardiac problems in pregnancy. New York, NY: Wiley-Liss, 1998; p. 39–55.
4. Hameed AB, Goodwin TM, Elkayam U. Effect of pulmonary stenosis on pregnancy outcomes – A case-control study. Am Heart J. 2007;154(5):852–4.
5. Dob DP, Yentis SM. Practical management of the parturient with congenital heart disease. Int J Obstet Anesth. 2006; 15(2):137–44.
6. Thilen U, Olsson SB. Pregnancy and heart disease: a review. Eur J Obstet Gynecol Reprod Biol. 1997;75(1):43–50.
7. Siu SC, Colman JM. Heart disease and pregnancy. Heart. 2001;85(6):710–5.
8. Siu SC, Sermer M, Harrison D. Risk and predictors for pregnancy-related complications in women with heart disease. Circulation. 1997;96(9):2789–94.
9. Kee WD, Khaw KS, Ng FF. Comparison of phenylephrine infusion regimens for maintaining maternal blood pressure during spinal anaesthesia for caesarean section. Br J Anaesth. 2004;92(4):469–74.
10. Bray JK, Fernando R, Patel NP, Columb MO. Suprasternal Doppler estimation of cardiac output: standard versus sequential combined spinal epidural anesthesia for cesarean delivery. Anesth Analg. 2006;103(4):959–64.
11. Bromage PR. Continuous lumbar epidural analgesia for obstetrics. Can Anaesth Soc J. 1961;85(21):1136–40.
12. Datta S, Lambert DH, Gregus S, Gregus J, Gissen AJ, Covino BG. Differential sensitivities of mammalian nerve fibers during pregnancy. Anesth Analg. 1983;62(12):1070–2.
13. Butterworth JF, Walker FO, Lysak SK. Pregnancy increases median nerve susceptibility to lidocaine. Anesthesiology. 1990;72(6):962–5.
14. Marx GF, Oka Y, Orkin LR. Cerebrospinal fluid pressures during labor. Am J Obstet Gynecol. 1962;84:213–9.
15. Campbell N, Rosaeg OP, Chan KL. Anaesthetic management of a parturient with pulmonary stenosis and aortic incompetence for caesarean section. BJA. 2003;90(2):241–3.