

Low dose combined spinal and epidural anaesthesia in a parturient with severe mitral stenosis and severe pulmonary arterial hypertension for Caesarean section

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ABSTRACT

We describe the anaesthetic management for an elective Caesarean section, of a parturient with severe mitral stenosis and severe pulmonary hypertension, using low dose of hyperbaric bupivacaine and for spinal block, supplemented with epidural lignocaine to achieve an adequate level. This patient was vulnerable to develop complications such as hypotension and tachycardia, should conventional regional anaesthesia be employed. This case reports highlights the haemodynamic stability using carefully titrated combined spinal-epidural anaesthesia in a patient with severe mitral stenosis.

Introduction

The pregnant patient with heart disease presents a difficult challenge to the obstetrician and the obstetric anaesthesiologist. A decision on appropriate anaesthesia modalities requires an understanding of the parturient's pathophysiology as well as pharmacological therapy, and the impact on patient care. The presence of clinically significant maternal heart disease during pregnancy increases the risk of adverse maternal, fetal and neonatal outcomes.¹ Rheumatic heart disease is the most common cardiac disorder in pregnancy in developing countries, with mitral stenosis being the single most prevalent lesion. Anaesthetic care requires preoperative optimisation, constant intraoperative haemodynamic stabilisation, and postoperative pain relief to decrease complications and ensure better maternal and fetal outcomes. We describe the anaesthetic management for an elective Caesarean section in a 26-year-old parturient with severe mitral stenosis and severe pulmonary hypertension, using a low dose of 5 mg hyperbaric 0.5% bupivacaine and 25 µg fentanyl for spinal block, supplemented with epidural lignocaine to achieve an adequate level. The patient had excellent haemodynamic stability and outcome.

Case report

A 26-year-old parturient (gravida 2, para 1, height 155 cm, weight 58 kg), with known chronic rheumatic heart disease was scheduled for elective Caesarean delivery at 36 weeks' gestation. Closed mitral valvotomy had been performed 10 years previously for critical mitral stenosis. She had a normal in-hospital vaginal delivery two years previously. In her second pregnancy, she experienced one episode of dyspnoea and palpitations at 26 weeks' gestation, for which digoxin 0.25 mg and furosemide 20 mg daily, and diltiazem 30 mg 8 hourly were commenced orally. At 36 weeks' gestation she was NYHA class 2 and had no palpitations or chest pain. On examination she was comfortable, her heart rate was 80 beats per minute and regular; blood pressure was 110/64 mm Hg and jugular venous pressure was elevated. There was a mid diastolic murmur and opening snap at the cardiac apex. There was no evidence of pulmonary oedema, hepatomegaly or peripheral oedema.

The electrocardiogram (ECG) showed sinus rhythm, 86/minute, with a right axis deviation, premature atrial complexes and left

posterior fascicular block. Her echocardiogram demonstrated severe mitral stenosis (0.8–1.0 cm²), trivial mitral regurgitation and severe tricuspid regurgitation. The peak pressure gradient across the mitral valve was 39 mmHg (mean of 24 mmHg), the ejection fraction was 56% and left ventricular systolic function was normal. There was severe pulmonary arterial systolic hypertension (51 mmHg). Her chest radiograph showed cardiomegaly with prominent bronchovascular markings. Her haemoglobin was 12 gm%, potassium 4.8 meq/L and renal function was within normal limits.

It was decided that she should undergo elective Caesarean section at 36 weeks' gestation. Pre-operatively she received diazepam 5 mg orally and aspiration prophylaxis (Tab ranitidine 150 mg and Tab metoclopramide 10 mg). In addition, antibiotic prophylaxis was given 30 minutes prior to surgery (Inj ampicillin 2 g and Inj gentamycin 80 mg).

In the operating room noninvasive blood pressure monitoring, 5 lead ECG and pulse oximetry was attached and an 18G intravenous line was secured. Her oxygen saturation on room air was 97%, the blood pressure was 106/64 mmHg and heart rate was 96 beats per minute. Under local anaesthesia the left radial artery and right internal jugular vein were cannulated. The initial central venous pressure was 3 mmHg, prompting the administration of 300 ml of normal saline over 30 minutes, and an increase in CVP to 6 mmHg.

The patient received combined spinal-epidural anaesthesia at the L3–4 level. Loss of resistance was obtained using an 18G Tuohy needle. Spinal anaesthesia was then performed using a needle through needle technique, employing a 26G Quincke spinal needle. The patient received 5 mg of hyperbaric 0.5% bupivacaine and 25 µg preservative-free fentanyl in the subarachnoid space. This resulted in loss of sensation to pinprick to the T10 dermatome, which was supplemented with 3 ml 2% epidural lidocaine. This resulted in a loss of sensation to pinprick to the T6 dermatome. A female infant of 2.5 kg was delivered with Apgar scores of 9 at 1 and 5 minutes, and umbilical arterial pH of 7.28. Blood loss during surgery was approximately 300 ml. Furosemide 10mg and oxytocin 5IU were given slowly IV after delivery. Intraoperative heart rate was 70–80/min and systolic blood pressure was 100–120 mmHg.

An infusion of bupivacaine 0.125% with fentanyl 2.0 µg/ml at 5.0 ml/hr via the epidural catheter was started at the end of surgery for postoperative analgesia, and the patient was transferred to high dependency unit. Her postoperative arterial blood gas analysis, chest radiograph, ECG, and haemoglobin level were within normal limits. On the following day, the epidural catheter was removed and the patient was transferred to the general ward. She was discharged on the fourth postoperative day.

Discussion

The haemodynamic alterations associated with pregnancy pose unique problems for the patient with mitral stenosis. This lesion is the most common valvular defect associated with maternal death in pregnancy.² With severe disease, such women face a pregnancy-related mortality of 5%.³ Labour, delivery and the immediate puerperium appear to be the times of the maximal risk.⁴ Pulmonary arterial hypertension carries a very high risk during pregnancy (30–50% mortality).⁵ A recent five year review of practice in an Australian hospital found that six of seven parturients with NYHA 4 symptoms received regional anaesthesia (three for Caesarean section and three for labour) and 12 out of 17 with NYHA 3 symptoms received regional anaesthesia (six for Caesarean section and six for labour).

There is no evidence to support any particular technique, but cardiovascular stability is the goal.⁵ Our anaesthetic goals were to achieve adequate level of blockade after combined spinal-epidural anaesthesia without producing hypotension and consequent tachycardia. Hypotension in such patients may produce myocardial ischaemia, and tachycardia may increase myocardial oxygen consumption and also decrease left ventricular filling time, which is a critical factor in mitral stenosis. Our goal in fluid management was normovolaemia since hypervolaemia may lead to pulmonary oedema and hypovolaemia would have decreased her preload. Central venous pressure monitoring was used for fluid management. We did not use a pulmonary artery catheter in this patient as her status was NYHA 2 and she had not been in heart failure. Moreover, there are documented cases of pulmonary artery rupture and pulmonary artery thrombosis when floatation catheters were used in patients with severe pulmonary hypertension, and their use has not been shown to improve survival in these patients.⁶⁻⁷

Low dose spinal anaesthesia given as part of a combined spinal-epidural (CSE) technique has been used effectively to provide anaesthesia for Caesarean section.⁸⁻⁹ It is important in clinical settings where hypotension may be poorly tolerated. The incidence of hypotension following spinal anaesthesia is reported to be as high as 94% in uncomplicated pregnancies.⁸ No interventions have been able to prevent hypotension completely but its incidence decreased 31% when intrathecal isobaric bupivacaine 5 mg and fentanyl 25 µg were used.⁸

Various combinations of low dose spinal anaesthesia supplemented by epidural anaesthesia for Caesarean section have been described in the literature. Fan et al concluded that low dose intrathecal hyperbaric bupivacaine (5 mg) in combination with 2% epidural lidocaine can provide effective and rapid anaesthesia for Caesarean section with minimum adverse effects in healthy term parturients.¹⁰

Lim Y et al used ultra low dose spinal bupivacaine (2.5 mg), fentanyl 25 µg and morphine 100 µg, with 3 ml of 1.5% epidural lidocaine for Caesarean delivery in an obese, preeclamptic patient.¹¹

Hamlyn EL et al used a low-dose CSE technique in an obstetric patient with mitral stenosis, employing intrathecal hyperbaric bupivacaine 5 mg with fentanyl 20 µg, followed by 5.0 ml epidural normal saline. During surgery, anaesthesia was supplemented by three, 2.0 ml increments of 0.5 % bupivacaine and one bolus of 25 µg fentanyl.¹²

Turker et al used continuous spinal analgesia (CSA) over-the-catheter technique (reduces risk of postdural puncture headache) in five parturients having moderate to severe mitral stenosis with pulmonary arterial hypertension. Initially, spinal fentanyl 25 µg and thereafter increments of fentanyl 10 µg provided effective analgesia during first stage of labour but during the second stage, 0.5% heavy bupivacaine 2.5 mg with saline 0.5 ml was used to supplement analgesia. This technique allowed maternal cardiovascular stability with analgesia for labour and no significant fetal heart rate abnormalities.

In our parturient, a combination of the reliability of intrathecal blockade with the flexibility of an epidural catheter provided titratable, haemodynamically stable anaesthesia. CSE using low dose intrathecal bupivacaine and fentanyl with epidural lignocaine supplementation was adequate for the performance of an uncomplicated Caesarean section with minimal side effects and good fetal outcome. In the early postoperative period, patients with mitral stenosis are at risk of developing pulmonary oedema, precipitated in part by auto transfusion from the contracting uterus. This may be exacerbated by the cessation of epidural analgesia and the return of sympathetic vascular tone.¹⁴ We commenced an epidural infusion in the postoperative period and the patient had an uneventful recovery.

Declaration

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

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