

Low dose Spinal Anaesthesia for Cesarean Section in Gravida with Rheumatic Heart Disease



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ABSTRACT

Cardiac disease in pregnancy remains an important etiology of maternal and fetal morbidity and mortality.¹ Mitral stenosis is the most commonly acquired valve lesion encountered in pregnant women and is almost invariably caused by Rheumatic heart disease (RHD).¹ Pregnancy and peripartum period represent a physiologic burden that may worsen symptoms in even moderate degrees of cardiac disease.¹ Consequently, many women are first diagnosed with the cardiac disease during pregnancy.¹ In this case report, we describe

the peripartum management of a 38 year old woman with congestive heart failure functional class II, severe mitral stenosis (MS), moderate mitral regurgitation (MR), and moderate tricuspid regurgitation because of RHD. She successfully underwent cesarean section with low-dose spinal anaesthesia using 7 mg hyperbaric bupivacaine intrathecally. This report highlights that low-dose spinal anaesthesia remains a good option in anaesthesia management for cesarean section in gravida with RHD, especially with severe MS.

Keywords: Rheumatic Heart Disease, RHD, Severe Mitral Stenosis, Low dose spinal anaesthesia, Cesarean section.

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INTRODUCTION

Maternal heart disease comprises approximately 1% of all pregnancies, but rheumatic heart disease (RHD) is a major cause of morbidity and mortality in pregnancy with acquired heart disease.² We present a case of the successful management of a parturient with congestive heart failure functional class II, severe mitral stenosis, moderate mitral regurgitation, moderate tricuspid regurgitation because of RHD, whom underwent cesarean section using low-dose spinal anaesthesia.

CASE REPORT

A 38 year old woman (gravida 3; para 2) presented at 37 weeks and 4 days gestational age, labour assessment revealed 8 cm in cervical dilatation, with congestive heart failure class II symptoms (New York Heart Association Functional Classification system), severe mitral stenosis, moderate mitral regurgitation, moderate tricuspid regurgitation caused by RHD. The patient was planned to undergo emergency cesarean section and tubectomy. The patient complained of coughing that gets worse but no complaint of shortness of breath. She started complaining about her coughing at 32 weeks of gestation until present.

Physical examination revealed an arterial blood pressure (BP) of 110/70 mmHg, a regular heart rate

of 100 beats/min, a respiratory rate of 22 breaths/min, and 96% peripheral oxygen saturation in room air. The patient's lungs revealed wet rhonchi in both lungs. Echocardiography showed left ventricle dilatation, 55% ejection fraction, global normokinetic, severe mitral stenosis (MS), moderate mitral regurgitation (MR), moderate tricuspid regurgitation, 185 mL end diastolic volume, 100 mL end systolic volume, and 0.9 cm² mitral valve area (MVA) planimetry. The electrocardiogram showed normal sinus rhythm with premature atrial contraction. The patient then was counselled to the anesthesiologist. She was accepted for anaesthesia in American Society of Anesthesiologist (ASA) class IV, with problems gravida with severe valvular heart disease.

In the operating room, standard monitoring was applied to the patient. Baseline vitals were recorded as BP of 125/80 mmHg, HR 79 beats/min, and SpO₂ 98 %. We made an arrangement of invasive arterial BP monitoring via radial artery cannulation. Peripheral IV lines (18G) were secured and she was premedicated with intravenous Furosemide (40 mg) followed by continuous infusion of 10 mg/hr.

The patient was placed in left lateral position and then, taking all aseptic precautions, a 27G Quincke's spinal needle was introduced via midline approach in L3-L4 interspace and 7 mg (1.4 mL) of 0.5 % hyperbaric bupivacaine was injected in the subarachnoid space. Injection is done within

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30 seconds. Block height and incision time recording can be seen in [table 1](#).

Table 1

During Surgery	
Injection time	21:10
Incision time	21:15
Delivery	21:30
Surgery last for (min)	30
Highest sensory level	Thoracal 6th
Time to achieve highest sensory level (min)	4
Time to achieve Bromage grade III (min)	4
APGAR score min-1 and min-5	7 and 8
Hypotension/ Bradycardia	None
Itching/ Nausea/ Vomiting/ Shivering	None

Table 2

Hemodynamics during surgery	
Systole (mmHg)	99 - 130
Diastole (mmHg)	60 - 85
Heart rate (beats/min)	65 - 80
Respiratory rate (breaths/min)	14 - 16
SpO ₂ (%)	98 - 100

The patient was then placed supine with a small pillow under the head. Oxygen was administered at 2 L/min via the nasal cannula. Urinary catheterization was done to monitor urine output. To prevent any visceral pain, intravenous Fentanyl (50 mcg), Ketamine (5 mg), and midazolam (1.5 mg) were given after birth. During surgery, the patient remained hemodynamically stable ([Table 2](#)).

No additional vasopressor was given. The surgery lasted for 30 minutes with blood loss and urine output of 550 mL and 800 mL, respectively. Intraoperatively, she received 500 mL crystalloid (Ringer lactate) and 200 mL colloid (Gelofusine). Postoperative analgesia was maintained with intravenous Morphine (20 mg), Ketamine (15 mg), Dexamethasone (5 mg), and Diphenhydramine (10 mg) in 50 mL of NaCl 0.9% administered at 2.1 mL/hr via a syringe pump. She was then transferred to the Intensive Care Unit for postoperative monitoring. The patient was discharged from the Intensive Care Unit on postoperative day 4 and from the hospital on postoperative day 7.

DISCUSSION

Normal pregnancy results in dramatic changes to the cardiovascular system. Pregnancy produces a 30-50% increase in blood volume and cardiac output.³ The increase in cardiac output is primarily the result of an increase in stroke volume with a smaller contribution from an increased heart rate.⁴ Cardiac output increases during labour and is 50% higher than before delivery.⁴ Immediately in the postpartum period, cardiac output increases maximally and reaches 80% over the prenatal period and is approximately 100% above the value when the woman is not pregnant, this is because at the time of uterine contractions there is a placental autotransfusion of 300-500 mL, each contraction increases the preload and hence, the cardiac output.⁴ There is an additional increase in venous return as a result of autotransfusion from the contracting uterus as well as from the loss of fetal compression of the inferior vena cava.¹ Central venous pressure increases 4-6 cmH₂O because there is an increase in maternal blood volume.⁴ The increased stroke volume and heart rate maintains the increased cardiac output.⁴

Increased cardiac output is not well tolerated in patients with valvular heart disease (aortic stenosis, MS) or coronary heart disease.⁴ Pregnancy reduces systemic vascular impedance.³ At the time of labour and delivery, pain and anxiety increases catecholamine release and as a result increases in heart rate, arterial blood pressure, and cardiac output.¹

Rheumatic fever (RF) is an autoimmune disease caused by gram-positive *Streptococcus pyogenes* bacteria that are prevalent in untreated throat infections in susceptible children and influenced by environmental factors, such as poor living standard and lack of access to health center.² The pathogenesis of acute rheumatic fever is believed to involve the triad of a genetically susceptible individual, infection with a rheumatogenic strain of group A streptococcus, and an aberrant host immune response.¹ Currently, clinical examination remains the basis of a diagnosis of RF and carditis, and the role of echocardiography should be considered supportive.⁵ However, an echo-Doppler examination should be performed if the facilities are available.⁵ The other invasive and noninvasive diagnostic modalities for RF, such as endomyocardial biopsy and radionuclide imaging, should be considered research tools.⁵

Rheumatic fever is considered as a precursor of RHD.² Rheumatic fever has manifestations on the system of cardiovascular, musculoskeletal, subcutaneous tissue, and central nervous system (such as acute cardiac inflammatory, polyarthritis, erythema marginatum, subcutaneous nodule, and chorea).² Incidence and pathological patterns of the mitral valve in RHD vary with age.² Patients that are <30 years old predominantly have MR, middle aged patients predominantly have MS, and older ages patients dominantly have a combination of MS and MR.²

The normal mitral valve orifice area is 4 to 6 cm². MS is characterized by a mechanical obstruction to left ventricular diastolic filling secondary to a progressive decrease in the size of the mitral valve orifice.⁶ This valvular obstruction produces an increase in left atrial volume and pressure.⁶ With mild MS, left ventricular filling and stroke volume are maintained at rest by an increase in left atrial pressure. However, stroke volume will decrease during stress-induced tachycardia or when effective atrial contraction is lost, as with atrial fibrillation.⁶ Symptoms usually develop when the mitral valve area is less than 1.5 cm².⁶ As the disease progresses, the pulmonary venous pressure is increased in association with the increase in left atrial pressure.⁶ The result is transudation of fluid into the pulmonary interstitial space, decreased pulmonary compliance, and increased work of breathing, which leads to progressive dyspnea on exertion.⁶ Over time changes in the pulmonary vasculature result in pulmonary hypertension, and eventually, right-sided heart failure may occur.⁶ Episodes of pulmonary edema typically occur with atrial fibrillation, sepsis, pain, and pregnancy.⁶

In our patient, she had complained of coughing from 32 weeks of gestation that continually worsened. Physical examination revealed wet rhonchi in both lungs. In addition to having severe MS, she had moderate MR and moderate tricuspid regurgitation. In general, regurgitant lesions are well tolerated during pregnancy because the reduction in systemic vascular resistance reduces the regurgitant flow.⁷ Conversely stenotic lesions have a greater potential to decompensate.⁷ Those with significant MS fail to tolerate the cardiovascular demands of pregnancy (increase volume loads and tachycardia) leading to pulmonary hypertension and an advancing NYHA class.⁷ Anaesthesia goals for pregnant women with MS are focused on the control of the heart rate and left atrial pressure.² Use of invasive monitoring depends on the complexity of the operative procedure and the magnitude of physiologic impairment caused by the MS.⁶ In this case, we applied invasive arterial BP monitoring via

radial artery cannulation, to confirm the adequacy of cardiac function and intravascular fluid volume.

Intravenous furosemide (40 mg) was administered followed by 10 mg/hr continuous infusion was given as a pre-anaesthesia with the aim to decrease the left atrial pressure and prevent fluid overload at the time after delivery.

Oxygen was applied at 2 L/min via nasal cannula to prevent hypoxemia. Hypoxemia can precipitate pulmonary hypertension and right ventricular heart failure due to increased heart rate and pulmonary vascular resistance.

When considering delivery by cesarean section, expert opinion recommends anaesthetic technique should be individualized. It is dependent on understanding the physiology of pregnancy and its interaction with the individual patient's pathophysiology, including lesion severity. The use of neuraxial anaesthesia requires measures to avoid hypotension, maintain adequate preload, and avoid tachycardia. Hyperbaric bupivacaine (0.5%, 7 mg, 1.4 mL) was injected in the L3-L4 interspace to obtain sufficient block height by preventing decreased of systemic vascular resistance. The increased heart rate that occurs as a reflex can lead to a decrease in cardiac output.

Accordingly, in case of hemodynamic instability, the vasopressor choice should be tailored to these hemodynamics goals.¹ Epinephrine should be avoided as it may induce tachycardia.¹ Phenylephrine may restore stable hemodynamics with little or no unwanted effect on uteroplacental perfusion.¹ The use of norepinephrine may provide additional inotropic support without significant increases in heart rate, while vasopressin relatively spares pulmonary vasculature.¹ During surgery, the patient remained hemodynamically stable and therefore, no vasopressor was needed.

To prevent any visceral pain and anxiety, an additional low dose ketamine and sedative after delivery (intravenous fentanyl 50 mcg, ketamine 5 mg, and midazolam 1.5 mg) was administered. Tachycardia resulting from pain further decreased diastolic left ventricular filling time.

Liquid restriction (700 mL) was considered to prevent excessive perioperative fluid that may increase the central blood volume and precipitate the occurrence of congestive heart failure.

Postoperative analgesia was maintained with intravenous morphine (20 mg), ketamine (15 mg), dexamethasone (5 mg), and diphenhydramine (10 mg) in 50 mL of NaCl (0.9%) administered at 2.1 mL/hr via a syringe pump. This prevents postoperative pain that causes an increased heart rate and pulmonary vascular resistance. Morphine has

advantages with respect to its venodilatory effects and for the relief of patient anxiety in addition to its analgesic effects.⁸

Alteration in hemodynamic status continues to occur for the first 24 hours after delivery.⁶ The risk of pulmonary edema and right heart failure can still occur until the postoperative period therefore, adequate cardiovascular monitoring must be maintained.⁶ For the postoperative period, the patient continued her treatment in the intensive care unit.

CONCLUSION

Low dose spinal anaesthesia remains a good option in anaesthesia management for cesarean section in gravida with rheumatic heart disease, especially with severe mitral stenosis.

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