Epidural anesthesia: A safe option for cesarean section in parturient with severe pulmonary hypertension

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Abstract

Rheumatic heart disease is the most common cardiac disease complicating pregnancy in developing countries. Heart disease accounts for 15% pregnancy-related mortality. In the presence of maternal heart disease, the circulatory changes of pregnancy may result in exacerbation of the hemodynamic perturbations due to complex cardiac valvular lesions leading to decompensation or death of mother or fetus. Determining the ideal anesthetic technique for cesarean section in the presence of complex cardiac conditions remains a much debated topic. General anesthesia is associated with a further increase in pulmonary pressure in response to laryngoscopy and intubation along with myocardial depression by anesthetic agents. Neuraxial blockade may lead to decrease in systemic vascular resistance and cardiac output. We report the successful anesthetic management of a parturient suffering from rheumatic heart disease with multivalvular lesions resulting in severe pulmonary hypertension under epidural anesthesia with good maternal and neonatal outcome. Successful management requires vigilant perioperative monitoring and thorough knowledge of the hemodynamics of complex cardiac valvular disease.

Key words: Cesarean section, epidural anesthesia, multiple valvular lesions, rheumatic heart disease, severe pulmonary hypertension

INTRODUCTION

Prevalence of heart disease in pregnancy varies from 0.3% to 3.5%. In the presence of maternal heart disease, the circulatory changes of pregnancy may result in decompensation or death of mother or fetus. Severe pulmonary hypertension is poorly tolerated in pregnancy. We shall discuss the anesthetic management for elective cesarean section of a young parturient suffering from rheumatic heart disease with multivalvular involvement and severe pulmonary hypertension in a Tertiary Care Teaching Hospital.

CASE REPORT

A 20-year-old primigravida with history of rheumatic heart disease with multivalvular lesions and severe pulmonary hypertension was posted for elective cesarean section at 36 weeks of gestation. Preoperative examination revealed history of exertional breathlessness and chest discomfort since 12 weeks of gestation which gradually occurred on less than ordinary activities with progression of pregnancy. There were no symptoms suggestive of paroxysmal nocturnal dyspnea, syncope, or congestive heart failure. Echocardiography revealed severe mitral regurgitation, moderate mitral stenosis (MS) (mitral valve area [MVA] 1.2 cm², Pressure gradient 20 mmHg), moderate aortic regurgitation, and mild tricuspid regurgitation with severe pulmonary hypertension (pulmonary artery systolic pressure [PASP] 75 mmHg, left ventricular ejection fraction [LVEF] 52% with mild LV systolic dysfunction). Electrocardiogram (ECG) showed sinus rhythm with bifid P waves [Figure 1]. Hematological and biochemical parameters were within normal limit. She was prescribed tablet metoprolol 25 mg twice daily, tablet frusemide 20 mg twice daily, pentids 400 mg twice daily, salt restriction, bed rest, and was considered to be at a high cardiac risk for pregnancy.

In the operating room, standard American Society of Anesthesiologists monitors were applied. Clinical examination revealed bilateral basal crepitations, tachypnea, noninvasive arterial blood pressure (BP) 113/60 mmHg, mean arterial pressure (MAP) 73 mmHg, heart rate (HR) 96/min, regular in rhythm, and SpO₂ 100% in room air. Central venous cannulation was performed through the right internal jugular vein for perioperative central venous pressure (CVP) monitoring. Baseline CVP was 7 cm of H₂O.
A graded epidural anesthesia was planned to maintain hemodynamic stability and to maintain optimum systolic BP, diastolic BP, HR, CVP, and prevent further rise in pulmonary vascular resistance (PVR). Epidural catheter was inserted through L1-L2 interspace with the patient seated and was placed 4 cm into the epidural space. Position was confirmed following administration of a test dose of 3 ml of 2% lignocaine with adrenaline. The patient was placed in the supine position with a left tilt of 15° to prevent aortocaval compression and a sensory block to T6 dermatome was achieved by 12 ml of 0.5% bupivacaine in fractionated doses of 3 ml over a period of 20 min with 50 μg fentanyl. Oxygen was administered by a face mask at 6 l/min throughout the intraoperative period. IV fluid infusion was guided by continuous monitoring of CVP. Arterial cannula for invasive BP monitoring was not instituted due to logistic constraints. It should be preferably used, if possible.

A male baby of 2.2 kg with Apgar score 9 and 10 at 1 min and 5 min, respectively, was delivered. Following delivery of the baby, 5 units of oxytocin was administered intramuscularly followed by 5 units in 500 ml of lactated ringer solution (RL) infused over 1 h. Hypotension (BP 116/43 mmHg, MAP 70 mmHg) occurred after oxytocin infusion was corrected by intermittent bolus of 50 μg of phenylephrine to a total of 300 mcg [Figure 2]. Total 1 L of RL was infused maintaining a CVP of 5-7 cm of H₂O [Figure 3].

Duration of surgery was 60 min. Following surgery, BP 112/58 mmHg, MAP 73 mmHg, PR 110/min, SpO₂ 100% in room air, and CVP 5 cm H₂O were recorded. There was slight improvement of the symptoms with mild decrease in the basal crepitations. The patient was transferred to the critical care unit for observation for 48 h. Analgesia was maintained with 0.125% bupivacaine at 5 ml/h infusion and paracetamol infusion (1 g in 100 ml) for 6 hourly. The patient was shifted to the ward after 48 h. Postoperative period was uneventful. Prescribed cardiac drugs were continued throughout the perioperative period. The patient was discharged from the hospital on 7th postoperative day after obtaining cardiological consultation for further management of underlying cardiac disease.

**DISCUSSION**

Pure or predominant MS occurs in approximately 40% of patients with rheumatic heart disease. The increased blood volume and cardiac output (CO) by 30-40% during pregnancy and a further increase in the CO to 80-100% of prelabor values following delivery are poorly tolerated by parturient with valvular heart disease such as mitral and aortic stenosis. A severe decompensation in myocardial function can develop during third trimester, labor, and immediately after delivery. Cardiac output, heart rate, and stroke volume decrease to prelabor values by 24-72 h postpartum and return to nonpregnant levels within 6-8 weeks after delivery.[1]

Severe pulmonary hypertension (PASP >75 mmHg), MS with New York Heart Association (NYHA) Class II–IV, mitral regurgitation (MR), and AR with NYHA Class III-IV predispose to a high maternal and fetal morbidity and mortality.[2]

Cardiac disease was diagnosed incidentally in this patient at a time when therapeutic termination was not feasible. Cesarean delivery was planned at 36 weeks gestation to maximize fetal lung maturation and to avoid deterioration in maternal cardiac status. Anticoagulants were not prescribed as there was no evidence of thrombi in the left atrial (LA) and ECG showed sinus rhythm.

The choice of anesthetic technique in this case was a challenge as it involved a rare combination of multiple valvular lesions along with severe pulmonary hypertension. Few cases of complex valvular heart disease in the obstetric patient population have been reported. Besides, the hemodynamic perturbations due to complex lesions were further exacerbated during pregnancy. Anesthetic management was individualized keeping in view the hemodynamic goals relevant to the underlying complex pathophysiology.

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**Figure 1**: Electrocardiogram depicting bifid P wave in lead II
Echocardiography findings suggested a normal sized LV with mild global hypokinesia and mild systolic dysfunction (LVEF 52%) and a dilated LA (LAID 4.7 cm) with MVA 1.2 cm² along with a bifid P wave on ECG. This indicates that MS was the predominant lesion with coexisting MR and AR which had led to an enlarged LA with pulmonary hypertension. Coexistent MR and AR causes severe volume overload of LV. MS and AR generate opposite loading conditions; hence, hyperdynamic contractility and LV enlargement may be less evident. MS restricts left ventricular filling and thus diminishing the impact of the aortic regurgitation on left ventricular volume.[3]

In this patient, symptoms were predominantly due to longstanding increase in LA volume and pulmonary hypertension along with a mild decrease in LV systolic function.

Neuraxial blockade in the form of graded epidural anesthesia that allows a gradual onset of block with avoidance of hypotension by intermittent fluid bolus and judicious use of vasopressors has been used successfully in the past.[4-8] Subarachnoid block was avoided to prevent the sudden hemodynamic perturbations. Some authors have described the use of general anesthesia with good maternal outcome[9] whereas others have reported increased pulmonary arterial pressure during laryngoscopy and tracheal intubation. Adverse effects of positive-pressure ventilation on venous return may lead to cardiac failure. Avoidance of tachycardia is of utmost importance which may be achieved by the use of opioids such as fentanyl before or during the induction of general anesthesia with neonatal respiratory depression as an adverse outcome.

Weeks and Smith[10] had concluded that although epidural anesthesia has been used with success, in preexisting RHF, large decrease in systemic vascular resistance (SVR) may cause further decrease in CO. On the other hand, GA may cause increase in PVR in response to laryngoscopy, intubation, and negative inotropic effects of anesthetic agents. According to Gomar and Errando,[11] with the exception of tetralogy of fallot, primary pulmonary hypertension, idiopathic hypertrophic subaortic stenosis, and anticoagulation, neuraxial techniques with low segmental blockade of dermatomes offer an alternative to general anesthesia in parturient with cardiac disease during cesarean section. Adequate cardiovascular invasive monitoring is essential and should be administered and maintained in the postpartum period with the same criteria that reduce morbidity and mortality in cardiac patients undergoing general surgery.

Keeping the underlying pathophysiology in view, we aimed to maintain hemodynamic stability by maintaining an optimum SVR, preload, heart rate, sinus rhythm, and avoiding myocardial depression and increase in pulmonary vascular resistance. This was achieved by epidural anesthesia in a graded manner using small fractionated doses of local anesthetic to ensure a gradual onset of block and minimize hemodynamic changes resulting from sympathetic autonomic blockade. We chose to avoid general anesthesia in our patient to prevent the rise in pulmonary vascular resistance and worsening of pulmonary hypertension resulting from sympathetic stimulation during laryngoscopy, intubation, and nitrous oxide inhalation and to prevent myocardial depression in response to anesthetics. We administered oxytocin by intramuscular route and by slow intravenous infusion to avoid tachycardia and diastolic hypotension.[12] Agents that increase PVR such as methylergometrine, prostaglandin F2 alpha, hypothermia, hypoxia, and inadequate analgesia were strictly avoided.[13]

SVR and CO increase after delivery which is a critical period for patients with pulmonary HT and thereby require
vigilant postoperative monitoring. Keeping this in view, we had monitored the patient closely for 48 h in the Intensive Care Unit.

Epidural anesthesia provides a safer alternative to general anesthesia in parturient with complex valvular lesions. However, it is easier said than done. Successful management necessitates strict vigilance and an extremely cautious approach to maintain the hemodynamic stability throughout the peripartum period.

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Conflicts of interest
There are no conflicts of interest.

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