



Perioperative Hemodynamic Management in Pregnancy with Aortic Coarctation Undergoing Cesarean Delivery under General Anesthesia: A Case Report

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ABSTRACT

Aortic coarctation represents a complex challenge during pregnancy due to altered cardiovascular physiology. We describe the successful perioperative management of a 24-year-old primigravida at 32 weeks of gestation with unrepaired coarctation of the aorta undergoing elective cesarean section. The patient presented with significant hypertension (upper limb 140-150/100 mmHg versus lower limb 90-100/70 mmHg) and dyspnea. Intraoperative management included dual invasive arterial line placement for continuous hemodynamic assessment, general anesthesia with careful titration of intravenous nitroglycerin (1 mL/hour), and thoughtful fluid administration resulting in a positive balance of 500 mL. Postoperative blood gas analysis demonstrated normal acid-base parameters. This case underscores the necessity for multidisciplinary coordination, invasive hemodynamic monitoring, and meticulous pharmacological intervention in high-risk cardiac pregnancies.

Introduction

Aortic coarctation, a congenital stenosis of the descending thoracic aorta occurring in approximately 3-5 per 10,000 live births, creates significant obstacles when encountered during pregnancy [1]. The normal physiological modifications during gestation—including 40-50% expansion of blood volume, 30-50% increase in cardiac output, and 20-30% reduction in systemic vascular resistance—become problematic in patients with this fixed stenotic lesion [2]. These adaptations lead to exaggerated proximal hypertension, progressive left ventricular strain, and compromised placental perfusion distal to the narrowing, endangering both mother and fetus. The maternal risks in uncorrected coarctation include acute hypertensive crises, left ventricular decompensation, aortic rupture, and aortic dissection, with reported fetal loss rates

approaching 20%. Anesthetic management demands comprehensive preoperative assessment, invasive hemodynamic monitoring, and careful pharmacological control. While regional techniques have been described, general anesthesia with invasive monitoring remains the preferred approach for high-risk cardiac patients undergoing cesarean delivery. This report documents our management strategy and perioperative outcomes.

Case Report

A 24-year-old primigravida at 32 weeks and 4 days of gestation (BMI 19.48 kg/m²) presented for elective cesarean section with a confirmed diagnosis of unrepaired coarctation of the aorta. She had been diagnosed with chronic hypertension one year prior and initiated on antihypertensive therapy during the third gestational month. Medications included Tab. Labetalol 100 mg three times daily and Tab. Nifedipine-R 10 mg

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twice daily, which were continued throughout pregnancy. Five weeks before this cesarean delivery, the patient had undergone incision and drainage of a vulval cyst under local anesthesia without complications. At that time, she first sought a cardiology consultation after experiencing breathlessness. Echocardiography at that visit displayed narrowing of the descending thoracic aorta with turbulent flow and mild concentric left ventricular hypertrophy. Under the supervision of cardiology, her medical optimization proceeded despite this cardiac history, with plans for an elective cesarean delivery to reduce hemodynamic stress.

Preoperative evaluation

During pre-anesthesia checkup, the patient had NYHA Class II dyspnea. Auscultation revealed a systolic murmur at the left midscapular region. Breath sounds heard were clear and bilaterally equal. Physical examination documented a substantial blood pressure differential between the upper and lower body compartments. Specifically, the right upper limb measured 150/100 mmHg, the left upper limb 140/100 mmHg, the right lower limb 90/70 mmHg, and the left lower limb 100/70 mmHg, establishing a systolic gradient of 40-50 mmHg. Heart rate of 98 beats per minute with respiratory rate of 20 breaths per minute was recorded.

ECG showed sinus arrhythmia. Transthoracic echocardiography revealed a preserved ejection fraction of 60% with concentric left ventricular hypertrophy and trivial mitral and tricuspid regurgitation. The study also showed narrowing of the descending aorta, suggestive of coarctation. Chest radiography showed left ventricular enlargement attributable to chronic hypertensive burden.

Laboratory assessment revealed the following findings. Hemoglobin was 11.0 g/dL (lower than the normal range), total leucocyte count was 9,600 cells/mm³ (within reference), and platelet count was 2.01 lakhs/mm³ (acceptable). Prothrombin time was 13.60 seconds and INR was 1.12. Renal function showed serum urea of 18 mg/dL and creatinine of 0.44 mg/dL, both preserved. Electrolytes were marginally abnormal: sodium 134 mEq/L, potassium 3.69 mEq/L. Hepatic parameters were essentially normal, with total bilirubin 0.35 mg/dL, direct bilirubin 0.16 mg/dL, SGOT 10 IU/L, SGPT 10 IU/L, and ALP 135 IU/L. Thyroid function tests were within normal range.

Given the presence of uncorrected coarctation with left ventricular hypertrophy, dyspnea, and chronic hypertension, the patient was assigned an ASA Physical Status Grade III classification. ICU beds and mechanical ventilators were reserved preoperatively. Inj. ranitidine 50 mg and inj. metoclopramide 10 mg were given intravenously as prophylaxis for aspiration.

Intraoperative Management

Anesthetic Technique

Standard monitoring was initiated followed by placement of two arterial line catheters one in the left radial artery and other in the left femoral artery enabling real time simultaneous assessment of systolic and diastolic pressures in the upper and lower body compartments. Compared to non-invasive techniques, this method provided better hemodynamic data by enabling continuous beat to beat variability of the pressure gradient across the coarctation.

After Preoxygenating with 100% oxygen for 3 minutes, rapid sequence induction was done using Inj. Propofol 140 mg intravenously followed by Inj. Succinylcholine 100 mg intravenously. Endotracheal intubation was achieved using a 7.0 mm cuffed endotracheal tube under direct laryngoscopy. Anesthesia was maintained with a combination of oxygen, air, and sevoflurane at 0.8-1.2% end-tidal concentration. Neuromuscular relaxation was achieved with Inj. Vecuronium 5 mg intravenously (Figure 1).



Figure 1- Perioperative setup and invasive monitoring.

Perioperative Hemodynamic monitoring and management

After the fetus was delivered, the patient's proximal blood pressures continued to rise, indicating a sustained hypertensive response. A conservative dose of about 0.8 µg/kg/min of intravenous nitroglycerin was started in order to lessen this reaction while maintaining organ perfusion. In order to reduce afterload on the hypertrophied left ventricle and minimize excessive vasodilation, this dosing strategy was selected. Because it reduces systemic and pulmonary vascular resistance without substantially impairing myocardial contractility or coronary perfusion pressure, nitroglycerin was chosen

for pregnant patients with coarctation due to its favorable hemodynamic profile.

Intravenous fentanyl 50 micrograms was given as supplementary analgesia. Inj. Oxytocin 20 IU infused slowly to increase myometrial contractility and prevent postpartum hemorrhage. Invasive hemodynamic monitoring showed a persistent systolic blood pressure difference of 40-50 mmHg between upper and lower limbs and persistent tachycardia with a heart rate of 120 beats/min. These hemodynamic changes persisted even after administration of nitroglycerin infusion, intravenous injection of 1000 mg of paracetamol, and ongoing general anesthesia, suggesting the presence of fixed stenotic nature of the aortic lesion.

Fluid Management and Operative Losses

During the operative period, 1,400 mL of Ringer's lactate was administered intravenously. Quantifiable blood loss totaled 600 mL, and measured urine output was 300 mL. Accounting for these volumes, the total measured output was 900 mL, resulting in a calculated positive fluid balance of 500 mL. A mildly positive balance was maintained intentionally to ensure that adequate perfusion of organs take place given the peripheral vasodilation induced by nitroglycerin therapy and the absolute requirement to maintain venous return during the operative procedure.

Emergence and Reversal

At procedure completion, neuromuscular blockade was reversed using Inj. Sugammadex 200 mg intravenously. Recognizing elevated blood pressure at emergence, an additional dose of Inj.

Labetalol 25 mg intravenously was administered to attenuate the hypertensive response accompanying tracheal extubation. After restoration of protective airway reflexes patient was extubated successfully and immediate postoperative period was un eventful with stable hemodynamics.

Postoperative Course

Arterial blood gas analysis obtained during immediate postoperative period showed a pH of 7.41, indicating appropriate acid-base balance without acidemia or alkalemia. Partial pressure of carbon dioxide was 35 mmHg, confirming adequate ventilation. Partial pressure of oxygen was 81 mmHg, demonstrating satisfactory oxygenation. Serum bicarbonate concentration was 23.4 mEq/L, and base excess was -2 mEq/L, both consistent with normal physiology. These values collectively indicated that the anesthetic technique successfully maintained acid-base homeostasis without development of respiratory or metabolic derangement.

Postoperative electrolyte assessment revealed minimal decrease of sodium to 133 mEq/L, (compared to preoperative 134 mEq/L), probably due to dilutional

effects from crystalloid administration. Potassium remained at 4.0 mEq/L and glucose was 101 mg/dL (elevated mildly). Serum lactate was 1.3 mmol/L, within the normal range. The postoperative total hemoglobin was 11.2 g/dL, which was nearly the same as the preoperative value. However, this was probably due to the combined effects of hemodilution from intravenous fluid administration and operative blood loss.

Postoperative Monitoring

The patient was shifted to the Surgical Intensive Care Unit for close and continuous hemodynamic monitoring. During the immediate postoperative period invasive arterial pressure monitoring was continued. Over first 24 hours, the patient remained hemodynamically stable with no episodes of hypotension, arrhythmias, or evidence of myocardial ischemia.

Neonatal Outcome

A male neonate was delivered with a weak cry and required intubation in the operation theater. He was immediately transferred to the Neonatal Intensive Care Unit for supportive care. The weak cry and requirement for assisted ventilation was mostly due to multiple contributing factors like premature delivery at 32 weeks of gestation, and possible intrauterine growth restriction secondary to reduced placental perfusion resulting from the maternal aortic coarctation, transplacental passage of anesthetic medications.

Discussion

Pathophysiological Considerations

Coarctation of the aorta equates a fixed obstruction of left ventricular ejection, creating a substantial afterload burden. During normal pregnancy, physiological compensation mechanisms include expansion of circulating blood volume by 40-50%, elevation of cardiac output by 30-50%, and reduction of systemic vascular resistance by 20-30% [3]. However, in patients with coarctation, these protective responses become maladaptive. Simultaneously, the reduced perfusion pressure distal to the coarctation compromises placental blood flow, endangering the fetal well-being [4].

This patient is a typical example of these pathophysiological features with a systolic pressure difference of 40-50 mmHg between upper and lower limbs, mild concentric left ventricular hypertrophy, and dyspnea at reduced exertion. The ejection fraction of 60% suggested a preserved systolic function despite chronic hemodynamic burden. However, the structural cardiac changes and functional limitations necessitated a specialized anesthetic approach.

Anesthetic Considerations

Cesarean delivery is recommended for pregnant women with hemodynamically significant coarctation to tide over the hemodynamic stress imposed by spontaneous labor contractions and delivery efforts, which can precipitate acute hypertensive crisis, acute left ventricular failure, or even aortic rupture. The selection of general anesthesia with invasive hemodynamic monitoring instead of regional anaesthesia can be argued as it provides superior control and permits emergency airway management if complications arise.

Dual invasive arterial catheterization in the radial and femoral positions was instrumental in this case, enabling continuous beat to beat variability, documentation of the pressure gradient and facilitating rational pharmacological intervention [5]. The persistent 40-50 mmHg pressure gradient despite of nitroglycerin therapy confirmed the fixed stenotic nature of the lesion, as vasodilators act predominantly on non-stenotic vascular segments rather than the stenotic site itself.

Role of Nitroglycerin

Intravenous nitroglycerin functions as a direct-acting vasodilator, reducing both arterial and venous smooth muscle vascular tension primarily through nitric oxide-mediated mechanism. The resulting hemodynamic effects include decreased systemic vascular resistance, reduced left ventricular afterload, and mild preload reduction [6]. These properties render nitroglycerin particularly suitable for pregnant women with coarctation, where management goals encompass prevention of further systolic blood pressure elevation, reduction of left ventricular wall stress, and maintenance of organ perfusion.

The conservative dosing strategy (0.8 µg/kg/minute) was chosen to minimize hypotension risk, which could compromise coronary and cerebral perfusion in the setting of significant aortic stenosis. This approach achieved hemodynamic stabilization without adverse effects, demonstrating the effectiveness of low-dose vasodilator therapy in this context.

Assessment and Management of Fluid Balance

The positive fluid balance of 500 mL was intentional and represents overall fluid management strategy. Excessive fluid restriction in cardiac patients risks organ hypoperfusion and acute kidney injury, while liberal fluid administration promotes pulmonary edema and postoperative morbidity [7]. Our moderate positive balance maintained adequate cardiac preload during peripheral vasodilation while avoiding fluid accumulation sufficient to precipitate cardiopulmonary decompensation.

Multidisciplinary Care

Effective management requires close coordination among anesthesiology, cardiology, obstetrics and critical care teams. Preoperative cardiology assessment established the hemodynamic significance of the lesion and ejection fraction, guiding intraoperative monitoring and pharmacological strategy. Obstetric input determined delivery timing at 32weeks + 4 days to balance fetal maturity against maternal hemodynamic tolerance. Invasive hemodynamic monitoring and careful titration of vasoactive agents provided by anaesthesiology team. ICU availability ensured postoperative close hemodynamic monitoring. This collaborative approach is considered appropriate for managing pregnant women with complex cardiac disease [2-3].

Conclusion

This case demonstrates successful perioperative management of an unrepaired aortic coarctation during cesarean delivery through multidisciplinary coordination, invasive hemodynamic monitoring with dual arterial catheters, general anesthesia, and judicious low-dose nitroglycerin therapy. The patient maintained hemodynamic stability throughout the operative period without maternal complications. While neonatal complications reflected the challenges of prematurity and intrauterine exposure to cardiac disease, the maternal anesthetic management achieved its objectives. This case report reinforces the critical importance of comprehensive preoperative assessment, invasive monitoring, and pharmacological optimization in high-risk cardiac pregnancies undergoing major surgery.

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