

CASE REPORT

Anesthesia for an adult patient with patent ductus arteriosus for interval tubectomy

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ABSTRACT

Patent ductus arteriosus (PDA) is a congenital defect where the ductus arteriosus remains patent after birth. The incidence is 1 in 2500 live full-term births, accounting for approximately 10% of all congenital heart defects.

A 24 year old female patient a known case of PDA (left to right shunt) came for bilateral abdominal tubectomy she had no signs and symptoms and the PDA was discovered incidentally. She was not on any treatment. During perioperative period, goals of anesthesia were to decrease systemic vascular resistance, right ventricular preload, and shunt flow and to maintain pulmonary vascular resistance. The course of the anesthesia was uneventful. This case highlights important points in the management of such cases.

Key words: Patent ductus arteriosus; Tubectomy; Anesthesia; Systemic vascular resistance

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INTRODUCTION

Patent ductus arteriosus (PDA) is a congenital defect where the ductus arteriosus remains patent after birth, and it may be associated with other cardiac anomalies. Its estimated incidence in US children born at term is between 0.02% and 0.006% of live births, but is more common in premature babies. Sometimes it may present in grown up adults. It usually results in left to right shift. In this case report we discuss the anesthetic management of a young lady with PDA.

CASE REPORT

A 24 year old female patient presented to us for bilateral abdominal tubectomy. She was a known case of PDA (left to right shunt) with no complaints of headache, giddiness, vomiting, epistaxis, or blurring of vision. She had been married for four years and had two children. During the second pregnancy, PDA was incidentally diagnosed and she was not on any treatment. On general physical examination, there was no pallor, icterus, cyanosis, clubbing, edema or lymphadenopathy. Her pulse

rate was 76 beats/min with normal rhythm and volume, blood pressure of 112/68 mmHg and respiratory rate of 14 breaths/min. During CVS examination, a continuous machinery murmur was heard along with S1 and S2. Normal vesicular breath sounds were audible over bilateral lung fields. Abdomen was soft and non-tender. No focal neurological deficit was seen. Her laboratory investigations showed a Hb 11.9 gm/dl, blood group A positive, platelets 3.35 lakh/mm³, WBC 5500 cells/mm³, blood sugar 92 mg/dl, blood urea 14 mg/dl, serum creatinine 0.6 mg/dl, Na 135 meq/l, K 4.0 meq/l, and liver function tests within normal limits. Her chest x-ray and ECG were normal. Her 2-D Echo revealed a PDA of 4 mm in size, with peak pressure gradient 94 mmHg, normal biventricular function and normal pericardium. There were no signs of pulmonary artery hypertension.

Cardiological opinion was sought, and the patient was stratified as mild risk. No infective endocarditis prophylaxis was given being not advisable.

On the day of surgery, patient was shifted to the operating room and an intravenous line was secured

with 18G cannula into dorsum of the right hand and infusion of Ringers lactate was started. Basic monitoring was instituted. Her HR was 86 beats/min, BP 120/80 mmHg and SPO₂ 99%. She received inj. glycopyrrolate 0.2mg, inj. midazolam 1mg, and fentanyl 100 µg. After pre oxygenation with 100% oxygen for 3 min, anesthesia was induced with inj. propofol 100 mg and succinyl choline 50 mg IV. A classic laryngeal mask airway (LMA) size 3 was inserted, connected to Bain's circuit and anesthesia was maintained with N₂O:O₂, 5:3 ratio with spontaneous ventilation.

Intraoperatively vitals were monitored and were stable. She received 350 ml of intravenous fluids intraoperatively. Intraoperatively care was taken to decrease systemic vascular resistance (SVR) gradually to prevent reversal of shunt and to avoid hypoxia, hypercarbia and acidosis so that pulmonary vascular resistance (PVR) was maintained. LMA was chosen to prevent sympathetic stimulation due to laryngoscopy during intubation and extubation. Immediate short term goals were to monitor for any hemodynamic changes. LMA was removed after adequate oral suctioning. Duration of surgery was 20 min.

Postoperatively patient was conscious and oriented, obeying oral commands. Her vital signs were as follows; HR 88beats /pm, BP 118/78 mm of Hg and SPO₂ of 99% on room air. She was kept in the ward for five days to monitor hemodynamic parameters and to treat any complications. She was followed up by cardiology for further management of PDA.

DISCUSSION

The number of adult patients with congenital heart disease (CHD) is rapidly increasing, and these patients will present for non-cardiac surgery with greater frequency. The cardiovascular anatomy and physiology of CHD is complex and requires specific knowledge of the defect and its anesthetic implications.¹ in adults with congenital heart disease who require non-cardiac surgery, perioperative risks can be reduced, often appreciably, when problems inherent to this patient population are anticipated.²

Ductus arteriosus is a vital foetal vascular structure originating from the left sixth aortic arch during embryonic development and connects the main pulmonary artery to the descending aorta. The ductus diverts blood away from the high-resistance, unexpanded foetal arterial circulation into the descending aorta.³ It normally closes spontaneously

within 24 to 48 hours after birth due to the contraction of medial smooth muscle in the vessel wall, due to the increased oxygen tension and reduced prostaglandin E₂ and I₂ levels. Endothelial adhesion followed by replacement of the muscle fibres with connective tissue results in the remaining ligamentum arteriosum within two to three weeks. If this does not happen, the patent ductus arteriosus persists. PDA is the third most common congenital cardiovascular anomaly, comprising approximately 10% of congenital anomalies or about 1 or 2 in 3000 live births. Although most cases of PDA would seem to occur sporadically, multifactorial inheritance is believed to underlie in many cases and these people are thought to possess a genetic predisposition⁴ Presentation in adult life is rare since the lesion is usually diagnosed and closed surgically in infancy or early childhood.^{5,6}

Adults with CHD may develop pulmonary hypertension for a variety of reasons. Potential aetiology includes pulmonary venous hypertension secondary to elevated ventricular end diastolic pressure, elevated pulmonary venous atrial pressure, or pulmonary vein stenosis. Many of these patients also continue to have decreased oxygen saturation secondary to residual shunts, poor lung function, and persistent decreased pulmonary flow.¹ Our patient was not symptomatic and the lesion was accidentally discovered.

Clinical manifestations of PDA may vary among people and are dependent on size of the ductus, the age of the patient, the pressure differential across the ductus, and the presence or absence of pulmonary hypertension. Some patients with an underlying PDA may be highly symptomatic, presenting with congestive heart failure, pulmonary hypertension, signs of volume overload, atrial fibrillation, recurrent pneumonia, or other complications known to be associated with PDA. Others have no signs or symptoms; and are called "silent".⁷ PDA may be discovered only incidentally on an echocardiogram. But even among asymptomatic PDAs who tolerate it for many years without clinical signs or symptoms, patients may become clinically significant with unrepaired PDAs with acquired conditions such as recurrent pneumonia, the development of chronic obstructive pulmonary disease, or the manifestations of valvular or ischemic heart disease are superimposed.⁸ Surgical treatment is the standard and recommended method for treating these patients.⁹ Treatment of choice must be decided on case to case basis considering size of ductus, and the presence or absence of pulmonary

hypertension, but trans-catheter occlusion can be the clinician's priority when possible. Patients with any shunt lesions are at risk of systemic air embolization. Thus, care should be taken to remove all air bubbles from all IV tubing.

Sympathetic stimulation, hypothermia and vasoconstrictors will lead to increase in SVR, these factors should be avoided. Reducing SVR will lead to decrease in right ventricular preload, which is favourable in shunt lesions. Mild hypoxemia can result in greater pulmonary vascular resistance and reversal of shunt flow. It is also important to avoid hypercarbia and acidosis, which may increase pulmonary vascular resistance severe fall in SVR, can cause an asymptomatic left-to-right shunt to become a right-to-left shunt with hypoxemia. Epidural anesthesia is the preferred technique for patients with PDA as it reduces SVR to decrease the shunt flow. General anesthesia can be used but one should avoid changes in SVR and pulmonary vascular resistance.¹⁰

We chose general anesthesia over regional anesthesia because the anticipated duration of surgery was short. Combination of midazolam

and propofol was used for smoother insertion of LMA and to decrease the hemodynamic response to intubation. Throughout the procedure care was taken to maintain SVR and our patient did not suffer any untoward event. Postoperatively adequate analgesia was provided with intravenous inj. diclofenac 75 mg.

CONCLUSION

Persistent PDA is a congenital heart defect which may be found in patients of any age from premature infants to older adults. The anesthetic implications vary depending on the anatomy of the ductus arteriosus and the underlying cardiovascular status of the patient. Even asymptomatic patient must be treated meticulously and peri-operative care should be provided with the knowledge of the presenting lesion.

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Author contribution:

SIS & LRR: Contributed to the intellectual content, concept and design of this work, the analysis and interpretation of the data (when applicable), as well as manuscript writing

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