Case Report

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# Anaesthesia for Antenatal Diagnosed Duodenal Atresia with Undiagnosed Persistent Pulmonary Hypertension of Neonate

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## ABSTRACT

Conversion of fetal circulation to adult-type occurs immediately after birth but neonates with problems in the development of pulmonary vasculature are prone to revert back to fetal circulation. This phenomenon is known as flip-flop circulation which may be induced perioperatively and as such anesthesiologist are central to its management. We report a case of term neonate planned for repair of duodenal atresia that despite having no respiratory symptom preoperatively developed severe hypoxemia under anesthesia that was even unresponsive to 100% oxygen. The intraoperative hemodynamics of the neonate was managed along with supportive care successfully. A postoperative echocardiogram confirmed the evidence of persistent pulmonary hypertension of the newborn.

**Keywords:** Duodenal atresia; flip flop circulation; neonates; persistent pulmonary hypertension of newborn

## INTRODUCTION

The incidence of duodenal atresia and PPHN per 10000 live births are 1 to 4 [1] and 1.9 [2] respectively. Duodenal atresiamay be associated with polyhydramnios and congenital anomalies like VACTREL. Surgical repair with duodenoduodenostomy is indicated.

Respiratory events are common causes of perioperative hypoxia in neonates.[3] Cardiac, metabolic, neurologic and infectious disorders are differential diagnoses. Preoperative screening of asymptomatic neonate may not always reflect all underlying cardiopulmonary issues.[4]

Persistent pulmonary hypertension of the newborn (PPHN) results when an abnormally elevated pulmonary vascular resistance (PVR) causes right-to-left shunting and hypoxia.[5] Intraoperative deterioration occurs due to multiple stressors.

## CASE REPORT

A 32-year-old primigravida with polyhydroamnios in the late second trimester, was suspected with fetal duodenal atresia. Rest of her antenatal assessment had been unremarkable. At $37^{+2}$ weeks, 6 hours following the rupture of membrane, a female child weighing 3.5 kg was delivered vaginally. Although the liquor was moderately stained,the APGAR score was 7/10 and 9/10 at 1 and 5 minutes respectively.Thorough examination of the neonate was unremarkable. The baby had aheart rate of 144bpm, blood pressure of 62/38 mm of Hg, respiratory rate of 48 bpm and SpO2 of 98 to 100 %. Her Ultrasonography confirmed duodenal atresia but transthoracic echocardiography showed no structural anomaly. It detected right ventricular systolic pressure of 30mm Hg and right to left shunts across foramen ovale and ductus arteriosus. Rest of the routine investigations were normal.

Laparotomy with diamond duodenoduodenostomy (Kimura operation) was planned to commence early feeding. Following counselling a written informed consent was obtained from the parents. A 24'G' cannula was secured and 10 % dextrose infusion was commenced at 8.5ml /hr. A nasogastric (NG) tube (6 Fr) was placed followed bybladder catheterization with a feeding tube (5 Fr).

The Operation theatre and the mattress were heated appropriately.Warm Inj. RL at 15ml/hr was started

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before surgery. ASA standard Monitors and a precordial stethoscope were attached. NG aspiration was followed by pre-oxygenation with a facemask at 6 lpm for 90 seconds. Balanced anesthetic induction was obtained with Inj. Propofol 10mg, Inj Fentanyl 5mcg and InjSuxamethonium 6mg. Airway was secured with 3.5 mm un-cuffed endo-tracheal tube and fixed after confirmation. Vital signs remained stable at induction and intubation. Sevoflurane at 0.89%-1.30% was commenced. Ventilation was initiated with pressure-controlled mode. A caudal injection was performed with 4.2 ml of 0.25% bupivacaine. Cotton padding was applied.

On commencement of surgery, rapid decrease in saturation to 50-60% occurred. Pulse rate reduced to 80 bpm, mean arterial pressure to 30 mmHg and ETCO2 to15-20 mmHg. At that time, a loud systolic murmur over the left heart border was heard. Sevoflurane was discontinued and manual ventilation was initiated. An assessment of oxygenation and ventilation confirmed them to be adequate. Arterial blood gas showed pH of 7.38, CO2 of 44 mmHg, arterial oxygen of 47 mm Hg and an A-a gradient of 611 mmHg. Her electrocardiogram showed normal sinus rhythm. Subsequently, Phenylephrine 1µg/kg IV and inj NS 10 mL/kg were administered. A rapid increase in SpO2 ensued followed shortly by normalization of heart rate and blood pressure.The episode lasted 3 minutes. Sevoflurane was restarted and tolerated, surgery resumed. A SpO2 of 88% to 92% and stable hemodynamics was achieved throughout the remainder of surgery. The surgery lasted 2 hours and there was blood loss of 1-2 ml. Ongoing fluid loss was replaced with NS and total urine output was 10 to 12ml. Following the surgery, she was transferred to neonatal intensive care unit (NICU)intubated for a step-wise extubation. Echocardiogram then revealed moderate tricuspid regurgitation and right ventricular systolic pressure of 90 mm Hg. Tab Sildenafil 0.5mg/kg tds was commenced. Post-operative arterial blood gas was unremarkable. She was extubated later that day and remaining hospital days were uneventful. Echocardiogram prior to discharge detected trace tricuspid regurgitation and right ventricular systolic pressure of 25 to 30 mmHg. However, it was unremarkable after a month.

## DISCUSSION

Causes of intraoperative hypoxemia includes patient(27%) or procedure(16%) related respiratory causes followed by intubation issues (13%), laryngeal or bronchial spasms (9%), congenital heart diseases (1%) or equipment failures (1%).[6]

PPHN is manifested by increase in lung vascular resistance and severe hypoxemia. Pulmonary vasculature has either developmentally reduced cross sectional area, abnormal muscular thickenings or if normally developed, fails to relax postnatally. Congenital diaphragmatic hernia, post-term delivery, meconium staining or aspiration, pulmonary parenchymal diseases, group B streptococcus infections and perinatal depression may be associated. Typically, these neonates display respiratory distress and labile oxygen saturation. Higher preductal saturations indicate shunting,which is controlled by subtle changes in the balance between PVR and Systemic vascular resistance (SVR) and causes labile hypoxemia. Examination may reveal tachypnoea, chest retractions and grunting. Cardiac examination may have a loud P2, split S2, systolic murmur of tricuspid regurgitation or a diastolic murmur of pulmonary regurgitation. Hepatomegaly due to right heart failure may be present.[7] ECG may reveal right ventricular hypertrophy and CXR may detect meconium aspiration, pneumonia and CDH. Enlarged right ventricle and pulmonary arterial shadow and attenuated pulmonary vascular markings may also be seen. Transthoracic echocardiography confirms the presence of pulmonary hypertension and provides information about the etiology.

Management of PPHN includes symptom relief and correction of the etiology. Initial management includes decreasing PVR, supporting cardiac output, and removing various stressors. Avoidance of pain, acidosis, hypercarbia and maintenance of SVR, myocardial contractility, preload and sinus rhythms are vital. Other stressors include low FiO2, ventilation mode, blood products and cardiopulmonary bypass. Monitoring of pre and post ductal oxygen saturation is beneficial.

Inhaled Nitric oxide, a pulmonary vasodilator, is the agent of choice for initial management. It has rapid onset, is easy to administer intraoperatively and could be continued postoperatively.[8] Other agents include phosphodiesterase inhibitors (sildenafil, milrinone) and prostacyclin analogues (epoprostenol).

Isoflurane and sevoflurane produce pulmonary vasodilation[9] while halothane, enflurane and nitrous oxide have no effects. Fentanyl attenuates the pulmonary vascular response to noxious stimuli[10] whereas benzodiazepines, propofol and etomidate have no clinically significant effects. Propofol may increase right-to-left shunt by decreasing SVR. Ketamine may produce adverse pulmonary responses when baseline PVR and PAP are very high[9] but when used in combination with pulmonary vasodilating drug or enriched FiO2, it may preserve coronary blood flow and limit right-to-left-shunt by maintenance of SVR.

Thus, no single anesthetic agent has been advocated as ideal and therefore balanced anesthesia is preferred.[9] Moderate hyperventilation with 100% oxygen, treatment of acidosis, and attenuation of precipitating stimuli and regional anesthesia, should be considered.Above all, preparedness is vital.

## CONCLUSIONS

Although, undiagnosed PPHN could produce rapid and severe perioperative hypoxemia and deterioration, preparedness and supportive care tailored to the degree of physiologic instability of the neonate will result in improved outcome.

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