DOI: 10.7860/JCDR/2026/84218.23557

Case Report

Anaesthesia Section

Airway Challenge in a Neonate with Pulmonary Atresia and Patent Ductus Arteriosus: Anaesthetic Implications During Planned Stenting

MEENAL RAJPUT[1], KARUNA TAKSANDE[2], KATTULA LEELA KRISHNA[3], PAYODHI MODI[4]

ABSTRACT

Neonates with duct dependent pulmonary circulation and complex conotruncal anomalies pose significant anaesthetic challenges, particularly during interventions such as Patent Ductus Arteriosus (PDA) stenting where even minor physiological or airway disturbances may precipitate rapid decompensation. We present the case of a seven-day-old male neonate weighing 2.7 kg, diagnosed with complex congenital cardiopulmonary disease in the form of pulmonary atresia, large misaligned Ventricular Septal Defect (VSD), and PDA arising between the Left Common Carotid Artery (LCCA) and the Left Subclavian Artery (LSCA), who was planned for PDA stenting. The infant had a baseline Oxygen Saturation (SpO2) of 86% on room air and a normal systemic examination, with stable haematological and biochemical investigations, except for mildly deranged coagulation parameters and neonatal hyperbilirubinaemia. Echocardiography revealed confluent branch Pulmonary Arteries (PA) supplied exclusively through a 3 mm PDA. After adequate fasting and optimisation, general anaesthesia was induced with fentanyl and ketamine, and muscle relaxation with rocuronium was administered after confirmed mask ventilation, and gentle ventilation was maintained with oxygen and sevoflurane. Despite repeated attempts, tracheal intubation was not possible, suspecting an associated airway anomaly. The procedure was abandoned to prevent hypoxia and haemodynamic instability in this duct-dependent circulation, and neuromuscular blockade was immediately reversed with sugammadex, allowing rapid return of spontaneous ventilation and recovery. The child was then referred for high-resolution Computed Tomography (CT) of the airway and great vessels to define the cause of intubation failure, which could not be done locally due to the unavailability of advanced imaging facilities. This case highlights the importance of anticipating airway anomalies in neonates with conotruncal defects and complex ductal anatomy, the utility of sugammadex as a rescue agent in neonatal airway emergencies, and the necessity of multidisciplinary referral to specialised centers for the safe completion of PDA stenting.

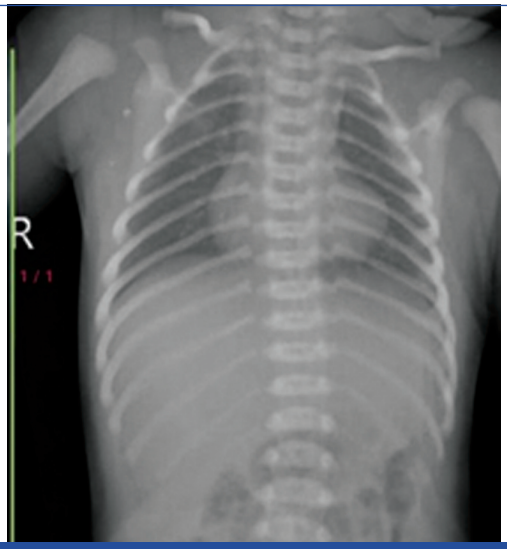
Keywords: Cardiovascular abnormalities, Haemodynamics, Newborn infants, Tracheal stenosis, Vascular malformations

CASE REPORT

A seven-day-old male neonate, weighing 2.7 kg and measuring 52 cm in length (Body Mass Index: 9.98 kg/m^2), American Society of Anesthesiologists (ASA) grade III, was admitted for PDA stenting. The diagnosis of a complex congenital cardiac anomaly had been made on an antenatal anomaly scan, and the baby was referred to our hospital for further management. The neonate was stable on presentation, with baseline SpO_2 of 86% on room air. The systemic examination was normal, with no dysmorphic features, a normal respiratory pattern, and no organomegaly. Cardiovascular and respiratory system examinations were unremarkable apart from the known congenital lesion. The neonate's haematological, coagulation, renal, and biochemical parameters were monitored. Mild abnormalities in coagulation, creatinine, sodium, and bilirubin were noted, consistent with neonatal physiology [Table/Fig-1]. Chest X-ray showed enlarged cardiac silhouette and reduced pulmonary vascular markings [Table/Fig-2].

Parameter	Value	Reference/Remark
Haemoglobin	16.1 g/dL	Normal for age
Total leukocyte count	10,300/µL	Normal
Platelet count	2.54 lac/µL	Adequate
Prothrombin time	15.5 sec	Mildly prolonged
International normalised ratio	1.32	Mildly elevated
Activated partial thromboplastin time	54.9 sec	Prolonged
Blood urea nitrogen	29 mg/dL	Normal
Serum creatinine	1.1 mg/dL	Mildly elevated for age
Sodium	149 mmol/L	Mild hypernatraemia
Potassium	4.8 mmol/L	Normal
Total bilirubin	7.3 mg/dL	Neonatal jaundice
Conjugated bilirubin	0.3 mg/dL	Normal

[Table/Fig-1]: Laboratory investigations of the patient.



[Table/Fig-2]: Chest X-ray showing enlarged cardiac silhouette, reduced pulmonary vascular markings.

Echocardiography revealed large segmental pulmonary atresia with absent forward pulmonary flow, a 3 mm PDA shunting from the aorta to the pulmonary artery, a large misaligned sub aortic VSD with bidirectional shunt, PDA arising between the LCCA and the LSCA, and confluent branch PA.

Anaesthetic management was tailored to the patient's duct-dependent pulmonary circulation. The goals were to maintain Systemic Vascular Resistance (SVR) to preserve ductal perfusion into the PA, avoid hyperventilation and hypocarbia {which could lower Pulmonary Vascular Resistance (PVR) and worsen right-to-left shunting}, and prevent hypoxia, hypotension, acidosis, and hypothermia. The baby was kept nil per os for an adequate fasting period, and intravenous (i.v.) glucose-containing maintenance fluids were optimised to prevent hypoglycaemia and maintain preload.

In the Operating Room (OR), ASA standard monitoring was utilised, including Electrocardiography (ECG), Non-Invasive Blood Pressure (NIBP), pulse oximetry, and temperature monitoring. Resuscitation medication and equipment were prepared, and vascular access was established. Premedication was with glycopyrrolate 0.004 mg/kg i.v. to minimise secretions and bradycardia. Induction was with fentanyl 2 µg/kg i.v. for blunting the sympathetic response, and ketamine 1 mg/kg i.v. to maintain systemic pressures while preserving SVR. Rocuronium 0.6 mg/kg i.v. was administered after ensuring adequate mask ventilation. Manual mask ventilation was continued with 100% oxygen and sevoflurane for three minutes, with gentle ventilation to prevent intrathoracic pressure and PVR changes.

Laryngoscopy revealed a Cormack-Lehane grading of III. Despite three attempts by a senior anaesthesiologist, endotracheal intubation was not possible. Due to repeated failure, the procedure was abandoned. Neuromuscular blockade was reversed with sugammadex 16 mg/kg i.v. The neonate recovered spontaneous ventilation with stable SpO_2 and was allowed to recover fully before being transferred from the OR. Due to the failed intubation and the risk of associated airway anomaly, a CT scan of the airway and great vessels was planned. Since high-slice CT was unavailable in our institute, the neonate was referred to a higher centre for further imaging and definitive treatment.

DISCUSSION

The PDA stenting in neonates with pulmonary atresia and a large misaligned VSD is an intricate process, and it necessitates a thorough anaesthetic plan. The entire physiology is dependent on the duct, with pulmonary blood flow originating from the ductus. Any drop in SVR or a sudden drop in PVR can drastically change the ratio of systemic to pulmonary blood flow. The anaesthetic objective is to maintain SVR, avoid significant drops in PVR, prevent hypoxia, acidosis, and hypothermia, and also maintain the stability of intravascular volume. In this situation, it was crucial that the patient's breathing be kept under control at all times, as over-ventilation or high oxygen concentration would lower PVR and cause that is, diversion of pulmonary blood flow into the systemic circulation. In contrast, under-ventilation or lack of oxygen would increase PVR and intensify cyanosis. Other cases, such as those reported by Agarwal A et al., Mohanty PK et al., and Gaál V et al., have also described this phenomenon, where even slight changes in ventilation led to rapid desaturation and serious haemodynamic deterioration, thereby demonstrating the difficulty of maintaining stable ventilation in neonates with limited physiological reserve [1-5].

Anatomical features also significantly affected the anaesthesia of this patient. The PDA was located vertically between the LCCA and the LSCA, a morphology typically associated with ductal tortuosity and angulation, which makes the intervention more challenging and increases the risk of haemodynamic instability. This type of ductal anatomy can prolong the operation time and cause intermittent reductions in pulmonary blood flow. Similar anatomical problems were mentioned in the case of congenital tracheal stenosis described by Agarwal A et al., airway narrowing due to the ectopic thymus reported by Mohanty PK et al., and the complex airway anatomy in tracheal agenesis described by Gaál V et al. These cases emphasise that neonates with cardiac defects usually have airway or vascular anatomical variances that not only complicate airway management but also haemodynamic stability. Some of the management strategies include the use of invasive monitoring, being prepared for rapid circulatory support, and early imaging to anticipate technical difficulties [1,3-6].

The most important issue to which the anaesthesiologist's attention should be drawn was airway management in this patient. Neonates with conotruncal anomalies and arch variants are susceptible to developing signs of airway problems, such as tracheal compression, tracheobronchomalacia, or intrinsic narrowing, before intubation, which can be difficult to recognise. In this situation, the lack of success in repeated intubation attempts performed under optimal technique indicated that the structural abnormality was the cause, rather than procedural difficulty. Similar airway problems have been successfully demonstrated in the above-mentioned case studies, which include congenital tracheal stenosis and tracheal agenesis in Agarwal A et al., airway obstruction due to ectopic thymus in Mohanty PK et al., and Floyd Type II tracheal agenesis, as reported in Gaál V et al., the difficulties of anaesthesia in these cases include a sudden, difficult airway, failure to pass the endotracheal tube beyond the cords, inadequate ventilation, rapid desaturation, and the absence of effective rescue airway options. The management recommendations based on these cases are no more than one or two attempts of intubation, maintenance of spontaneous ventilation if possible, early use of alternative airway devices, senior anaesthesiologists and ENT surgeons being available at any moment, and pre-procedure imaging like CT or bronchoscopy for identifying the location and cause of the obstruction [3-5,7,8].

Terminating the operation as a result of the unsuccessful intubation was the right move. In duct-dependent physiology, repeated laryngoscopy or prolonged apnea may induce severe hypoxemia, acidosis, and circulatory collapse. The sugammadex-induced reversal of neuromuscular blockade facilitated the patient's quick recovery of spontaneous breathing, and they became stable. Even though it is off-label in neonates, sugammadex is an excellent and very rapid reversal option in cannot-intubate situations when rapid reversal is paramount to patient safety. In contrast, fatalities were reported in the cases by Mohanty PK et al., and in the study by Gaál V et al., likely due to the absence of rapid rescue options. Equally important is post-event planning. Referring for high-resolution imaging of the airway and great vessels was crucial in this case, which is similar to the approach suggested by Agarwal A et al., where CT and bronchoscopy provided both diagnosis and guidance for the future anaesthetic strategy [3-5,9-11].

A comparison of management strategies in similar cases is seen in [Table/Fig-3] [3-5].

Author	Case Description	Management	Key Takeaways
Present Case	7-day-old male neonate, 2.7 kg, pulmonary atresia, large malaligned VSD, vertical PDA between Left Common Carotid Artery (LCCA) and Left Subclavian Artery (LSCA), baseline SpO_2 86%. Normal systemic examination, mild coagulopathy, neonatal jaundice. Failed intubation suspected due to an airway anomaly.	Fentanyl and ketamine induction, rocuronium administered after confirming mask ventilation, maintained on oxygen and sevoflurane. Intubation failed despite multiple attempts, and the procedure was abandoned. Neuromuscular blockade was reversed with sugammadex 16 mg/kg. A referral for high-resolution CT of the airway and great vessels followed stable recovery.	Anticipation of airway anomalies in conotruncal and arch defects is critical. Repeated intubation attempts should be avoided in duct-dependent physiology. Sugammadex can be a valuable rescue agent in a neonatal airway crisis. Advanced airway and vascular imaging is essential before further intervention.

Agarwal A et al., [3]	Case 1: Preterm male, 1100 g, 30+4 weeks, failed intubation attempts due to inability to pass tube beyond cords, later diagnosed as congenital tracheal stenosis on CT. Case 2: Term female, 1800 g, cyanotic at birth with repeated failed intubations, later identified as tracheal agenesis with carinoesophageal fistula and multiple anomalies consistent with VACTERL association.	Case 1: Stabilised with suboptimal ET position and conventional ventilation, later underwent slide tracheoplasty successfully on day 78, and was discharged on day 136. Case 2: Managed with a mask and attempted oesophageal ventilation but remained unstable; due to poor prognosis and severe anomalies, ventilator support was withdrawn.	Failure to pass the ET tube beyond the vocal cords should raise suspicion of congenital tracheal malformations. Bronchoscopy and CT are essential for diagnosis. Slide tracheoplasty is the preferred treatment for congenital tracheal stenosis with good outcomes. Tracheal agenesis carries a poor prognosis; oesophageal ventilation may serve as a temporary measure. Early suspicion and referral to tertiary centres are critical.
Mohanty PK et al., [4]	Baby boy, 960 g, born at 36 weeks to a primigravida by emergency caesarean for fetal bradycardia and severe oligohydramnios. Antenatal scan showed Intrauterine Growth Restriction (IUGR), single umbilical artery, placental insufficiency and hypoxia. At birth, the baby was limp and apnoeic with Heart Rate (HR) 70/ min. Bag and mask ventilation failed, multiple failed intubation attempts as the tube could not pass beyond the glottis, suspected subglottic obstruction. Emergency tracheotomy planned, but the baby died. Autopsy revealed bilateral renal agenesis, tracheoesophageal fistula (type C), periglottic ectopic thymus compressing trachea, and congenital talipes equinovarus.	Immediate resuscitation with bag-mask ventilation and chest compressions failed. Multiple intubation attempts were unsuccessful. Baby succumbed during resuscitation. Autopsy confirmed airway compression from ectopic thymic masses along with various anomalies.	Ectopic thymus is a rare cause of neonatal airway obstruction and difficult intubation. Antenatal suspicion is difficult without a visible neck mass, but an MRI can confirm the diagnosis. EXIT procedure or planned surgical excision may be lifesaving when airway obstruction is anticipated. Association with bilateral renal agenesis and tracheoesophageal fistula indicates a possible syndromic or VACTERL association. Delivery in tertiary centres with multidisciplinary preparedness is essential.
Gaál V et al., [5]	Female infant, 34 weeks, 1630 g, delivered by emergency caesarean for intrauterine bradycardia. Prenatal ultrasound showed polyhydramnios and right upper limb abnormality (absent ulna and thumb, shortened radius). At birth, severe respiratory distress with copious foamy secretions. Intubation failed as the tube could not be advanced beyond the subglottis. Bag-mask ventilation and Nasal Continuous Positive Airway Pressure (NCPAP) temporarily improved oxygenation, but subsequent attempts at intubation and tracheostomy failed. Autopsy revealed cardiac anomalies (ASD, VSD), upper limb anomaly, and Floyd Type II tracheal agenesis with tracheo-oesophageal fistula	Initial resuscitation with bag-mask ventilation and NCPAP provided transient stabilisation. Despite multiple intubation attempts by neonatologists, anaesthesiologists, and ENT surgeons, the airway could not be secured. Tracheostomy failed as no trachea was palpable below the cricoid. Baby deteriorated rapidly, and resuscitation was withdrawn due to a lethal diagnosis.	Tracheal agenesis is an extremely rare but lethal anomaly. Prenatal diagnosis is challenging, especially when a tracheo-oesophageal fistula is present. Postnatal indicators include severe respiratory distress, absent cry, failed intubation, and absence of palpable trachea. Floyd's classification guides understanding; Floyd Type II is the most common. Bag-mask ventilation or oesophageal ventilation may provide transient stabilisation. Definitive therapy, including oesophageal interposition, allogenic grafts, and 3D-printed tracheal replacements, remains experimental. Delivery at tertiary centres with readiness for EXIT or advanced airway interventions is crucial

[Table/Fig-3]: Anaesthetic management in similar cases [3-5].

CONCLUSION(S)

The PDA stenting in neonates with pulmonary atresia and complex arch anatomy demands a delicate balance between stable haemodynamics and safe airway management. Although the primary anaesthetic concern is maintaining SVR, avoiding sudden drops in PVR, and preventing acidosis or hypothermia, the airway can be a crucial factor in procedural success, as in this case. The inability to intubate despite repeated attempts emphasised the possibility of concomitant airway or vascular anomalies, necessitating advanced imaging before further intervention. Rapid reversal of neuromuscular blockade with sugammadex enabled a rapid return of spontaneous breathing and a safe recovery, emphasising its role as a rescue agent in neonatal airway emergencies. Definitive treatment requires a multidisciplinary approach, referral to a higher-level center with advanced imaging and airway expertise, and coordination of cardiac and airway factors to enable a safe and effective reattempt of PDA stenting.

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PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India.
2. Professor, Department of Anaesthesia, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India.
3. Junior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India.
4. Junior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Meenal Rajput,
Junior Resident, Department of Anaesthesia, JNMC, Sawangi Meghe, Wardha, Maharashtra, India.
E-mail: drminals10@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 07, 2025
- Manual Googling: Jan 30, 2026
- iThenticate Software: Feb 02, 2026 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Sep 21, 2025**
Date of Peer Review: **Nov 28, 2025**
Date of Acceptance: **Feb 05, 2026**
Date of Publishing: **Jun 01, 2026**