PREOPERATIVE STABILIZATION OF INFANTS BORN WITH CONGENITAL DIAPHRAGMATIC HERNIA : A CASE REPORT

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ABSTRAK

Hernia diafragma kongenital adalah kelainan bawaan langka, di mana organ perut masuk ke rongga toraks akibat defek pada diafragma. Pada praktik kedokteran saat ini, penundaan operasi hingga stabilisasi kardiorespiratori tercapai terlebih dahulu mulai dipopulerkan. Pasien merupakan neonatus aterm (37–38 minggu) dengan distress napas 10 menit setelah lahir. Skor Apgar 7 dan 6 pada menit ke-1 dan ke-5, tangisan tidak teratur, tubuh kemerahan, dan ekstremitas sianosis. Bunyi peristaltik terdengar di hemitoraks kiri, dan foto toraks menunjukkan hernia diafragma kongenital kiri dengan pergeseran mediastinum. Pasien mendapat ventilasi dengan mode *volume control/assist control* (VC/AC): fraksi oksigen (FiO_2) 35%, laju napas 40, *positive end-expiratory pressure* (PEEP) 6, mempertahankan saturasi oksigen perifer (SpO_2) 91–99%. Setelah pasien stabil, dilakukan laparotomi dengan anestesi umum. Premedikasi berupa atropin dan fentanil, induksi dan pemeliharaan menggunakan oksigen, sevofluran, dan udara terkompresi. Analgesia pascaoperasi diberikan fentanil dan parasetamol. Evaluasi pascaoperasi menunjukkan suara napas kiri membaik dan bunyi peristaltik menghilang. Pasien dirawat di NICU selama tiga hari, kemudian lima hari di ruang rawat biasa sebelum pulang. Penundaan operasi memberikan waktu adaptasi terhadap hipoplasia paru dan kontrol hipertensi pulmonal. Mekanisme proteksi paru yang meliputi volume tidal rendah dan hiperkapnia yang ditoleransi penting untuk mengurangi komplikasi. Kasus ini menegaskan pentingnya penundaan operasi hingga setelah stabilisasi pada hernia diafragma kongenital.

Kata kunci : hernia diafragma kongenital, neonatus, stabilisasi preoperatif

ABSTRACT

Congenital diaphragmatic hernia (CDH) is a rare birth defect where abdominal organs herniate into the thoracic cavity due to a diaphragmatic defect. While previously managed as a surgical emergency, current practice emphasizes delayed surgery following cardiorespiratory stabilization. A term neonate (37–38 weeks) developed respiratory distress 10 minutes post-delivery. Physical examination revealed peristaltic sounds in the left hemithorax. Apgar scores were 7 and 6 at 1 and 5 minutes, respectively. The infant had irregular crying, pink body, and cyanotic extremities. Chest radiograph confirmed a left CDH with mediastinal shift. Ventilatory support was initiated with volume control/assist control (VC/AC) mode: oxygen fraction (FiO_2) 35%, RR 40, and positive end-expiratory pressure (PEEP) 6, maintaining peripheral oxygen saturation (SpO_2) of 91–99%. After stabilization, laparotomy was performed under general anesthesia with orotracheal intubation. Premedication included atropine and fentanyl. Anesthesia was induced and maintained with oxygen, sevoflurane, and compressed air. Postoperative analgesia included fentanyl and paracetamol. Post-surgery, breath sounds improved on the left side, and peristaltic sounds resolved. The patient was monitored in the neonatal intensive care unit (NICU) for three days, followed by five days in the regular ward before discharge. Stabilization before surgery reduces pulmonary complications by allowing adaptation to lung hypoplasia and managing pulmonary hypertension. Lung-protective strategies, including low tidal volume and permissive hypercapnia, are critical.

Keywords: *congenital diaphragmatic hernia, neonatus, preoperative stabilization*

INTRODUCTION

Congenital diaphragmatic hernia (CDH) is considered a rare congenital defect that poses a surgical emergency condition in neonates requiring comprehensive management. CDH affects approximately 1 in 3600 live births and is considered a life-threatening condition (Quinney & Wellesley, 2018). To date, the mortality and morbidity rates of this case are still high. The majority of neonates born with CDH only have an estimated survival rate of 67% (Haroon & Chamberlain, 2013). The exact aetiology of this congenital defect is still unknown. However, some patients are associated with several genetic and chromosomal abnormalities. Neonates with CDH usually present with one or more structural abnormalities, with the most common being found in the cardiovascular system (Quinney & Wellesley, 2018).

The congenital malformation in CDH causes an incomplete closure in the neonate's diaphragm. This condition enables the herniation of abdominal organs into the thoracic cavity. The herniation could substantially increase the pressure in the affected hemithorax and potentially disturb the neonate's pulmonary development. Pulmonary hypertension is a particular condition that requires special attention, as it could aggravate CDH and is strongly associated with cardiopulmonary dysfunction. Despite the herniated mass, cardiopulmonary dysfunction in CDH is predominantly associated with hypoplasia and pulmonary hypertension (Pranata & Kurniyanta, 2021). Neonates with more severe hypoplasia could exhibit symptoms earlier, ranging from a few minutes to hours immediately after birth (George & Joshi, 2013).

Respiratory distress poses a challenge in CDH cases and is associated with lung hypoplasia and pulmonary hypertension. Other contributing factors included hypoxemia, hypercarbia, metabolic acidosis, and shunting from right to left. Several potential problems arising from the neonates that could induce further complications and require special attention are hypothermia, hypoglycemia, and bradycardia (Pranata & Kurniyanta, 2021). Several decades ago, CDH was considered a neonatal emergency requiring urgent surgery. However, the current approach includes prioritizing the stabilization of cardiorespiratory status via gentle ventilation, close hemodynamic monitoring, and pulmonary hypertension treatment prior to surgery initiation. Current advancements focus on the significance of protective ventilation, judicious scheduling of surgical intervention, and the consideration of extracorporeal membrane oxygenation (ECMO) (Chandrasekharan et al., 2017). In this case, we reported the preoperative stabilization of neonate with congenital diaphragmatic hernia.

CASE ILLUSTRATION

A neonate was referred to our hospital with a chief complaint of shortness of breath, which was observed 10 minutes immediately after birth. The patient was born term at 37-38 weeks of gestational age through Caesarean section with clear amniotic fluid, an Apgar score of 6/7, and cried spontaneously with vigorous movements at birth before experiencing worsening respiratory distress 10 minutes later. Antenatal history revealed that the neonate experienced intrauterine growth restriction (IUGR), and a pathological non-stress test (NST) was observed, which served as the indication for the Caesarean section procedure. Upon birth, the neonate's weight was measured at 2900 grams with a total length of 48 cm, and a body temperature of 36.7 $^{\circ}$C. Head circumference and chest circumference were 35 cm and 32 cm, respectively. Pain assessment using the Face, Legs, Activity, Cry, and Consolability (FLACC) scale was 0/10.

The neonate was under sedation at physical examination with 70/32 mmHg blood pressure, 160-170 bpm heart rate, and normal cardiac auscultation findings. Respiratory evaluation showed a respiratory rate of 43 times per minute, symmetrical chest movement, and vesicular breath sounds predominantly in the right lung without any wheezing or rales. The

patient had a 91-99% peripheral oxygen saturation on ventilator mode volume control/assist control (VC/AC) with oxygen fraction (FiO2) 35%, respiratory rate 40, and positive end-expiratory pressure (PEEP) 6 cmH_2O. Peristaltic sounds were observed in the left hemithorax. Abdominal examination revealed a soft abdomen without any tenderness, distention, or muscular defense. Peristaltic bowel sounds were normal at 3 to 4 times per minute. Urogenital examination showed no abnormalities. The patient's extremities were warm without any edema, and capillary refill time was below 2 seconds. The Mallampati score was difficult to evaluate in the patient. Additional laboratory findings are summarized in Table 1.

Table 1. List of Laboratory Results

Blood Count	
White blood cells (10^3 µL)	16.44
Hemoglobin (g/dL)	16.00
Hematocrit (%)	49.20
Platelet (10^3 µL)	318.00
Mean corpuscular volume (fL)	106.00
Mean corpuscular hemoglobin (pg)	34.50
Mean corpuscular hemoglobin concentration (g/dL)	32.50
Coagulation Profile	
Partial thromboplastin time (s)	16.4*
Activated partial thromboplastin time (s)	35.5
International normalized ratio	1.20*
Liver Function	
AST/SGOT (U/L)	75.00*
ALP/SGPT (U/L)	16.00
Kidney Function	
Urea (mg/dL)	10.5
Creatinine (mg/dL)	0.67
Electrolytes	
Natrium (mmol/L)	141
Kalium (mmol/L)	4.61
Chloride (mmol/L)	107.5
Random blood glucose (mg/dL)	133

*high

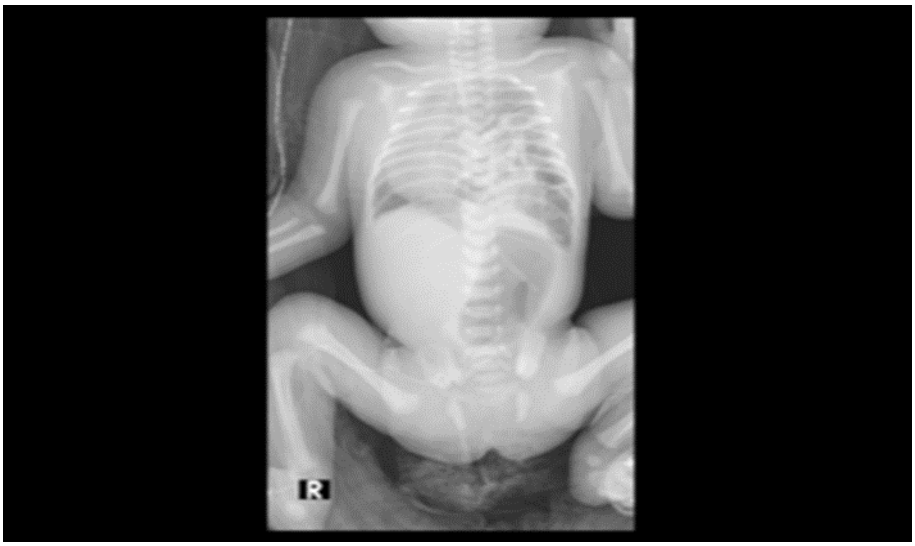

Figure 1. Babygram

A babygram radiograph was taken before referral, and radiological findings revealed a left diaphragmatic hernia that pushed the trachea, heart, and mediastinal structures to the right side.

No visible signs of ileus or pneumoperitoneum were observed. An endotracheal tube was seen with the distal tip projecting at the right side of the 4th thoracic vertebrae. A gastric tube was also observed with the distal tip projecting at the left side of the 3rd lumbar vertebrae. The patient has several potential problems, including bronchospasm, laryngospasm, desaturation, hypoglycemia, bradycardia, hypothermia, and vagal reflex. Initial stabilization efforts were oxygen supplementation through nasal canulla and orogastric tube (OGT) insertion for decompression. The abdomen was soft upon palpation after decompression. The patient was also given Dextrose 10% through infusion as a management for hypoglycemia as well as crystalloids at 60 mL per kilogram body weight as enteral route was not feasible to be accessed. Urgent laparotomy to repair the hernia was then planned with a pediatric surgeon.

Preoperative preparation included fasting the patient for 6 to 8 hours prior to surgery. The anesthetic procedure was performed with general anesthesia with orotracheal intubation (GA-OTT). The patient was administered 0.1 mg of intravenous sulfas atropine as premedication. The analgesic used was 5 mcg intravenous fentanyl. Induction was achieved using oxygen and sevoflurane at concentrations of 6-8%. Endotracheal tube placement was confirmed by symmetrical bilateral chest auscultation and appropriate packing. Maintenance of anesthesia was carried out using oxygen, compressed air, and sevoflurane. Additional medication administered intraoperatively included paracetamol 40 mg IV. Postoperative management included the administration of 100 mcg fentanyl via 10 ml NaCl 0.9% at an infusion rate of 0.6 ml/h and 40 mg paracetamol every 6 hours. The patient was transported to the NICU with a ventilator after surgery. Recovery was monitored strictly in the NICU for three days with ventilator on pressure control-assist control (PC-AC) mode, FiO2 35%, PEEP 6, PIP 18, and respiratory rate 40 breaths per minute. Transfusion of packed red cells, platelet concentrate, and albumin was also given to provide postoperative hemodynamic stability and optimize recovery. Heart rate was measured at 142 beats per minute and axillary temperature at 36.9^{o}C.

The patient was put on fasting for an additional three days after procedure and was given liquid diet afterwards. The patient tolerated the intake well with one episode of vomiting and was transferred to the regular inpatient ward for five days before discharge. Upon discharge, hemodynamic was stable with heart rate measured at 148 beats per minute, respiratory rate at 40 times per minute, peripheral oxygen saturation at 98% on room air, and axillary temperature at 36.9^{o}C. Nutritional intake and bowel movements were sufficient for care to be continued at home.

DISCUSSION

CDH is a congenital defect in which an opening in the diaphragm permits abdominal organs to migrate into the chest cavity, potentially impairing lung development in newborns. It is a rare condition, occurring in approximately 1 out of every 3,600 live births. However, it is associated with a high mortality and morbidity rate, and is considered a neonatal emergency requiring comprehensive and multimodal management. The exact aetiology of CDH is still unknown and is assumed to be multifactorial. CDH may present as an isolated lesion, but is more commonly associated with other abnormalities, especially congenital heart diseases. CDH predominantly occurs on the left diaphragm via the left posterolateral *Bochdalek* foramen. It could also affect the right posterolateral *Bochdalek* foramen and the anterior Morgagni foramen with a significantly lower prevalence (Greer, 2013). The spectrum of defects in the diaphragm ranges from a focal defect in the posterior muscle rim to complete agenesis of the diaphragm (Chandrasekharan et al., 2017).

CDH's embryological origin is debated. It may result from failed diaphragm fusion[7] or from lung bud disruption affecting diaphragm development via the post-hepatic mesenchymal plate (PHMP).[5] Diagnosis relies on history, physical exam (e.g., cyanosis, dyspnea,

dextrocardia), and imaging showing bowel in the chest and lung underdevelopment (Chandrasekharan et al., 2017). In CDH, the severity of pulmonary hypoplasia and pulmonary hypertension is widely recognized as the most critical determinants of postnatal morbidity and mortality, making them key predictive indicators for patient outcomes (Lakshminrusimha & Vali, 2020). Underdeveloped lungs characterize pulmonary hypoplasia due to the compression in the thoracic cavity, thus resulting in fewer and abnormally structured airways and alveoli (Dumpa & Chandrasekharan, 2023). Most neonates with severe CDH also develop pulmonary hypertension, which arises from aberrant pulmonary vascular remodeling. It is characterized by reduced numbers of pulmonary arterioles and thickened vascular walls, thus preventing the normal fall in pulmonary vascular resistance after birth (Dumpa & Chandrasekharan, 2023). This persistent elevation in pulmonary vascular pressure leads to significant right-to-left shunting of blood, impaired systemic oxygenation, and right ventricular dysfunction (Singh & Lakshminrusimha, 2021).

Several decades ago, CDH was initially considered a neonatal emergency requiring urgent surgical repair to reduce mediastinal mass effect. However, the current approach prioritizes postponing surgical intervention until cardiorespiratory stability is achieved under medical management within the first 24 to 48 hours of life (Baschat et al., 2024). The three main challenges that are prioritized in CDH preoperative care are pulmonary hypoplasia, pulmonary hypertension, and biventricular cardiac dysfunction (Lakshminrusimha et al., 2022). In sequence, preoperative management and stabilization should involve early intubation, proper ventilator management, and correcting pulmonary hypertension before proceeding to surgical repair (Guidry et al., 2012). Our case followed this sequence closely, emphasizing thorough preparation and multidisciplinary coordination. The patient was kept nil per os (NPO) for 6–8 hours, aligning with guidelines to reduce aspiration risk. Early involvement of anesthesiology ensured readiness, including emergency and anesthetic drugs (e.g., Sulfas Atropine), proper IV access, warming devices (IV warmer, mattress warmer), temperature monitoring, and a range of uncuffed ETTs (sizes 3.0, 3.5, and 4.0) to accommodate airway needs and avoid barotrauma. Use of NICU ventilator support was pre-planned, consistent with the goal of lung-protective ventilation prior to repair.

Our anesthetic plan also reflects current recommendations. General anesthesia with oral tracheal intubation (GA–OTT) was chosen to secure the airway definitively. Premedication with Sulfas Atropine (0.1 mg IV) served to reduce secretions and vagal responses. Analgesia was achieved using fentanyl (5 mcg IV), a short-acting opioid with minimal hemodynamic impact. Induction using 6–8% sevoflurane with oxygen ensured smooth transition without compromising pulmonary function. ETT placement was confirmed via symmetrical auscultation, minimizing the risk of one-lung ventilation, a critical consideration given the underlying pulmonary hypoplasia. Maintenance with sevoflurane, oxygen, and compressed air provided stable anesthesia with minimal cardiovascular depression. Adjunctive medication like paracetamol (40 mg IV) was used to provide multimodal analgesia, reducing opioid requirements postoperatively.

Proper ventilator management includes optimizing ventilation and V/Q matching to decrease postnatal pulmonary vascular resistance (PVR). This is a crucial step to prevent the incidence of acidosis, high PVR, and worsening cardiac dysfunction, which increase the need for extracorporeal life support (ECLS) (Baschat et al., 2024; DeKoninck et al., 2021). Due to the presence of asymmetric lungs in CDH neonates, providers should provide balanced ventilation between atelectasis of the larger lung and hyperinflation of the smaller lung. Hyperinflation could be prevented by applying lower mean airway pressures. Hyperinflation could compress the alveolar vessels and increase PVR if not adequately managed. Ventilation is usually considered adequate when an expansion of eight to nine ribs is observed on chest radiograph (Baschat et al., 2024).

In our case, while the detailed ventilator settings were not explicitly listed, the preparation of NICU ventilator access indicates that proper respiratory support planning was made in anticipation of these ventilation strategies. Furthermore, the emphasis on ETT size selection (3.0, 3.5, 4.0 uncuffed) reflects an intention to maintain a secure, low-pressure airway suitable for pressure-controlled ventilation, in line with gentle ventilation principles. However, no data were available on the use of specific ventilator settings such as FiO_2 levels, PIP, or PEEP, which makes it difficult to fully confirm adherence to the permissive hypercapnia or lung-protective ventilation strategy in detail. In earlier practices, neonates with CDH were often subjected to aggressive hyperventilation via high fraction of inspired oxygen (FiO_2) and high peak inspiratory pressures (PIP) with the intention of inducing hyperoxia and respiratory alkalosis to pharmacologically vasodilate the pulmonary vasculature and mitigate pulmonary hypertension. However, this strategy often led to severe ventilator-induced lung injury in the already hypoplastic and fragile lungs, resulting in significant morbidity and mortality. The principles of the current approach in the preoperative stabilization of CDH neonates are the adoption of a lung-protective, or gentle ventilation strategy to prevent barotrauma and volutrauma to the susceptible pulmonary parenchyma (Traynor, 2024).

In our case, there is no indication that inhaled vasodilators or ECMO were required, suggesting that the patient remained stable under initial ventilatory support and anesthetic management. This may indicate successful early stabilization or a less severe degree of pulmonary hypoplasia and hypertension. However, the absence of echocardiographic or blood gas data limits further evaluation of pulmonary vascular status and left heart function. Early intubation in CDH neonates immediately after birth is recommended as standard care. It is recommended for the PIP to be adjusted to < 25 cm H_2O and initial FiO_2 to be started at < 1.0 in settings of pressure-controlled ventilation. A lower FiO_2 could prevent the adverse effects of oxygen toxicity. Ventilation is also recommended to be initially set to provide 3-5 cm H_2O PEEP (Snoek et al., 2016). PEEP will then only be adjusted to correct overinflation of atelectasis (Traynor, 2024). Another crucial component of this gentle ventilation is the acceptance of permissive hypercapnia, which allows for higher partial arterial pressures of carbon dioxide ($PaCO_2$) above the normal range as long as it does not increase right-to-left shunting (Guidry et al., 2012). The resulting hypercapnic acidosis from permissive hypercapnia may reduce the physical compression of alveolar vessels, resulting in a decreased PVR (Traynor, 2024).

The permissive hypercapnia strategy allows $PaCO_2$ levels to be within the 45 to 60 mmHg range with a 7.25-7.40 pH. In the delivery room, preductal SpO_2 is generally maintained above 85% but lower than 95% if any supplemental oxygen is used (Traynor, 2024). A preductal SpO_2 level as low as 70% during the first two hours of life is allowed, provided that gradual improvements are observed, organ perfusion is adequate (pH > 7.2), and ventilation is maintained at $PaCO_2$ < 65 mmHg. Afterwards, preductal SpO_2 levels are recommended between 85-95% (Snoek et al., 2016). In certain conditions, a low preductal SpO_2 level down to 80% can still be tolerated provided that organ perfusion is adequate (pH > 7.2), lactate level < 5 mmol/L, and urinary output > 1 ml/kg/h. Postductal SpO_2 should be maintained above 70% (Snoek et al., 2016). Concurrently, comprehensive stabilization efforts are undertaken to correct any underlying metabolic acidosis, reduce detrimental right-to-left shunting, enhance pulmonary perfusion, and judiciously administer crystalloid and blood products to ensure adequate intravascular volume and red blood cell mass (McCann et al., 2018). In our case, while capnograph monitoring was included in the preparation checklist—indicating a plan to monitor $PaCO_2$ or end-tidal CO_2—no specific blood gas or SpO_2 targets were reported. The use of mattress warmer, IV warmers, blood products, and crystalloid access supports efforts to maintain hemodynamic stability and adequate perfusion, consistent with theory recommendations.

In conditions where ventilation alone is not adequate in managing pulmonary hypertension, inhaled pulmonary vasodilators could be considered. Drug delivery may be commenced only if optimal ventilation has been achieved (Baschat et al., 2024). However, the use of pulmonary vasodilators should be considered carefully, as efforts to increase pulmonary blood flow could put a significant burden on the underdeveloped left heart (Moore et al., 2024). Despite numerous advanced efforts, 25-30% of CDH neonates require ECLS, particularly ECMO (Sferra et al., 2023). The cannulation options in ECLS include both veno-venous (VV) support via a dual-lumen cannula and vena-arterial (VA) support via two separate cannulas. The VV-ECLS cannulation is preferred in patients presenting with respiratory failure with or without slight cardiac failure, but it is not possible in low birthweight neonates due to the small jugular vein. VA-ECLS cannulation is more commonly opted for and can be an option when cardiac failure worsens (Guner et al., 2018).

The clinical course for neonates with CDH can be unpredictable, with some infants initially showing a favorable response to stabilizing maneuvers such as gentle ventilation and hemodynamic support. However, this early period of apparent stability may represent a deceptive "honeymoon period", often followed by a sudden, and frequently unexplained, clinical deterioration (Traynor, 2024). This rapid decline is typically characterized by a return to, or exacerbation of, persistent pulmonary hypertension, coupled with severe physiological derangements including acidosis, escalating hypoxemia, and marked hypercapnia. Furthermore, this deterioration is often compounded by increased right-to-left shunting of blood through persistent fetal pathways, notably the foramen ovale and patent ductus arteriosus, exacerbating systemic hypoxia and contributing to profound cardiorespiratory instability (Davis & Cladis, 2021). In the discussed case, the patient had worsening respiratory distress upon admission. However, blood gas analysis was not performed and acidosis could not be evaluated. Early stabilization and prompt decision making in handling the case made further condition worsening with clear clinical manifestation preventable.

Our case did not experience sudden clinical deterioration in the perioperative or immediate postoperative period, as evidenced by the controlled anesthetic management and stable transition to NICU ventilator post-surgery. The continuation of ventilator support postoperatively, combined with analgesic and thermoregulation strategies, implies that the team successfully maintained cardiorespiratory stability beyond the typical “honeymoon period.” This constant ventilator support diminished the need for ECMO and provided maintenance of preductal $SpO_2 > 85\%$ and postductal $SpO_2 > 70\%$. Fluid therapy also resulted in sufficient measured urine output at 5 mililiters in 2 hours, which was more than 0.5 ml/kg/h. There were reported cases of persistent pulmonary hypertension, which clinicians commonly initiate trials of nitric oxide (NO) and high-frequency oscillatory ventilation (HFOV), while simultaneously evaluating the need for ECMO (Davis & Cladis, 2021). In cases where NO does not provide the desired response, intravenous prostacyclin, phosphodiesterase type 5 inhibitor or sildenafil, and other drugs involving the endothelin pathway could be considered as alternatives. However, the use of these drugs should prompt a repeated cardiac evaluation as they can lead to insufficient filling of the heart and poor systemic perfusion (Snoek et al., 2016).

A study by Dillon et al. investigated the relationship between pulmonary artery pressure (PAP) and survival in 47 full-term neonates with CDH. These infants were managed with a strategy of delayed surgery and gentle ventilatory approaches, high-frequency oscillatory ventilation (HFOV), permissive hypercapnia, nitric oxide (NO), or extracorporeal membrane oxygenation (ECMO). The study found a strong correlation between estimated PAP/pulmonary artery pressure (measured via echocardiogram within the first 3 weeks of life) and patient outcomes. All 23 infants (49% of the cohort) who maintained normal PAP (defined as less than 50% of systemic pressure) survived. Conversely, none of the eight patients who exhibited

persistent systemic or higher PAP survived. Among the remaining 16 patients whose PAP fell between these two extremes, 12 ultimately survived (Dillon et al., 2004). PAP was not measured in this case. However, ventilation settings used both in surgery and postoperative monitoring allow proper oxygenation control to ensure proper lung expansion.

Surgical repair can be performed as soon as the transition to extrauterine circulation is complete and the PVR has been stabilized (Traynor, 2024). Intraoperative ventilation and oxygen delivery strategies for infants undergoing CDH repair closely mirror those employed in the preoperative period, prioritizing the prevention of lung injury and stabilization of the neonate. To avoid volutrauma and shear stress-induced lung injury, the approach involves delivering a small tidal volume and maintaining minimal PEEP, sufficient to prevent atelectasis. The goal for oxygenation is to achieve adequate saturation (SpO_2 90% to 95%) without inducing hyperoxia. Furthermore, permissive hypercapnia is also maintained, allowing for higher $PaCO_2$ while ensuring the arterial pH remains above 7.25 (Guidry et al., 2012). Specifically, the European CDH EURO Consortium Consensus recommends that infants be ventilated with a PIP not exceeding 25 cm H_2O and a PEEP set between 3 to 5 cm H_2O. The respiratory rate is then adjusted to achieve a target arterial carbon dioxide tension ($PaCO_2$) of 45 to 60 mmHg (Snoek et al., 2016).

Intraoperatively, our case used: (1) general anesthesia with sevoflurane (6–8%) and compressed air + oxygen; (2) ETT confirmation via symmetrical auscultation; (3) capnograph monitoring; and (4) fentanyl and paracetamol for multimodal analgesia. These steps demonstrate an anesthetic and ventilation approach likely aligned with gentle ventilation goals. The use of a capnograph implies that $PaCO_2$ was being monitored, which is crucial in maintaining permissive hypercapnia. However, we did not document specific intraoperative ventilator settings (PIP, PEEP, tidal volume), SpO_2, or ABG results, limiting direct comparison with the consortium's targets. Nevertheless, the absence of intraoperative instability or escalation of support suggests that lung-protective strategies were likely effective. Additionally, the use of appropriate ETT sizing, thermoregulation tools, and NICU ventilator planning shows thoughtful integration of the core principles from the European CDH management guidelines.

CONCLUSION

CDH is a rare, life-threatening congenital defect where incomplete diaphragm closure leads to abdominal organ herniation into the thoracic cavity. Pulmonary hypoplasia and pulmonary hypertension are two critical predictors of morbidity and mortality in CDH cases. Historically managed as a surgical emergency, current practice prioritizes stabilizing the neonate's cardiorespiratory status before surgery. Proper stabilization technique allows prevention of lung hypoplasia and pulmonary hypertension, as well as optimizing cardiac function and ensuring better surgical outcomes. A multidisciplinary approach is necessary to ensure optimal stabilization efforts.

ACKNOWLEDGEMENTS

The author wishes to express his gratitude to the professors of the Anesthesiology and Intensive Care Department, Faculty of Medicine, Udayana University / Prof. dr. I.G.N.G Ngoerah General Hospital for their valuable guidance and support in the preparation and publication of this case report. The author also extends appreciation to the department for providing continuous encouragement and meaningful learning opportunities.

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