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Current Anesthesia Management of Congenital Diaphragmatic Hernia: A Narrative Literature Review

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A B S T R A C T

Congenital diaphragmatic hernia (CDH) is a congenital disorder characterized by a defect in the diaphragm, which causes the contents of the stomach to protrude into the chest cavity. Increased survival of CDH patients related to initial management in the form of a ventilator strategy, management of pulmonary hypertension, improvement of surgical and anesthetic techniques, and use of extracorporeal membrane oxygenation (ECMO) when indicated. Early stabilization of the patient is a priority before performing a surgical intervention for the hernia organ. This narrative literature review aimed to explain the current management, especially in the field of anesthesia in a congenital diaphragmatic hernia. In conclusion, the current management aims to improve survival rates and reduce the morphology of CDH patients.

1. Introduction

Congenital diaphragmatic hernia (CDH) is a defect in the diaphragm that causes the protrusion of abdominal contents into the chest cavity. This condition can appear as a stand-alone lesion or as part of a syndrome. The incidence of CDH ranges from 0.8 – 5/10,000 births and varies. The incidence of CDH is more prevalent in males, and African – American races have a lower risk of developing CDH.[1]

In general, there are three basic types of CDH, namely Bochdalek hernia (posterolateral), Morgagni hernia (retrosternal or anterior), and hiatal hernia. Bochdalek hernia occurs due to failure of closure of the left pleuroperitoneal membrane, while the Morgagni hernia occurs due to failure of the union of the rib and sternal muscles. hiatal hernia is the entry of the abdominal esophagus and gastric cardia into the chest cavity through the widening of the esophageal hiatus. The most common congenital diaphragmatic hernia is a hernia in Bochdalek, with an incidence of 1 in 2000-4000 live births.[2]

The use of ultrasonography (USG) and magnetic resonance imaging (MRI) in prenatal care is an excellent predictor and is used as a prognostic factor for CDH, especially in patients with congenital heart

defects and pulmonary hypertension. Echocardiography in CDH patients on the first day of life helps estimate the degree of pulmonary hypertension, along with blood gas analysis and oxygenation index (IO) examinations. The presence of supra-systemic pulmonary hypertension is considered a risk factor for death in CDH patients.[3]

CDH, with hypoplasia and pulmonary hypertension, is a major determinant of morbidity and mortality. Compression of the lung parenchyma will cause embryological disturbances early in fetal development, causing babies to be born with underdeveloped or hypoplastic lungs. The development of fetal lungs in the womb of the fetus can develop well because of the normal circulation of the placenta and gas exchange. The hypoplastic lungs of CDH patients are unable to meet the circulation needs of the adult type. This causes hypoxia, which further worsens pulmonary vascular resistance and causes progressive respiratory failure. If this condition is not treated with mechanical ventilation and other interventions, it can be a cause of death in newborns.[4,5]

Increased survival of CDH patients related to initial management in the form of a ventilator strategy, management of pulmonary hypertension, improvement of surgical and anesthetic techniques, and use of extracorporeal membrane oxygenation (ECMO) when indicated.[6] In recent decades, there has been an increase due to advances in cardiopulmonary resuscitation in the intensive care unit. ECMO is an excellent treatment modality for neonates with reversible circulatory or respiratory failure, with a significant impact on survival.[7,8]

Research by Kai et al. (2020) showed that infants with CDH undergoing ECMO are associated with increased survival. This study found that early initiation of ECMO (within 72 hours of cannulation) was associated with increased survival, minimal bleeding, shorter ECMO duration, and fewer circuit changes. In addition, perioperative administration of anticoagulants such as aminocaproic acid or tranexamic acid, as well as close perioperative monitoring of coagulation parameters, are associated with a reduced risk of bleeding with improved ECMO.[9]

Early stabilization of patients is a priority before performing a surgical intervention for herniated organs, so the management of perinatal pulmonary hypertension is an important focus in the current treatment of CDH. Bartlett et al. (1975) reported the first successful use of ECMO in a newborn with cardiopulmonary failure. This technique was immediately applied to CDH neonates with severe respiratory distress to provide preoperative stabilization. The use of ECMO in CDH neonates is still being debated. A Cochrane review of its use for severe respiratory failure in infants showed no significant survival benefit for neonates with CDH. Although many surgeons in the United States and Europe consider ECMO as a treatment option, its use in Asia for CDH neonates has been rarely reported.[10] This narrative literature review aimed to understand the latest management, especially in the field of anesthesia in patients with congenital diaphragmatic hernia.

Congenital diaphragmatic hernia

Congenital diaphragmatic hernia (CDH) is a defect in the diaphragm, which occurs more frequently on the left side and posterolaterally, allowing herniation of the abdominal contents into the thorax. Hypoplastic lungs and abnormal blood vessels lead to persistent respiratory insufficiency and pulmonary hypertension with high mortality. Despite improved medical and surgical management, survival remains at 60-70%.[11,12] Hernias can arise from the failure of normal diaphragm growth or arise from areas that are prone to pressure, namely the foramen Bochdalek, foramen Morgagni, and esophageal hiatus.[2]

Pulmonary hypoplasia may be a major causative factor in the pathophysiology of diaphragmatic hernia. The main pathophysiology underlying CDH is a combination of pulmonary immaturity and leading to hypoplasia. Persistent pulmonary hypertension of the newborn (PPHN).[1] Pulmonary hypoplasia occurs on the ipsilateral side of the herniation, with the contralateral side being affected to varying degrees (Figure 1). The most recent hypothesis explaining lung injury in CDH is that the initial compromise occurs during the organogenesis stage resulting in bilateral hypoplasia,

followed by ipsilateral lung compression secondary to herniated abdominal viscera. The interference results in decreased branching of the bronchioles and pulmonary vessels, causing acinar hypoplasia. Terminal bronchioles become smaller with the thickening of the alveolar septa. The lungs are relatively immature and cause pulmonary vascular hypoplasia PPHN.[5]

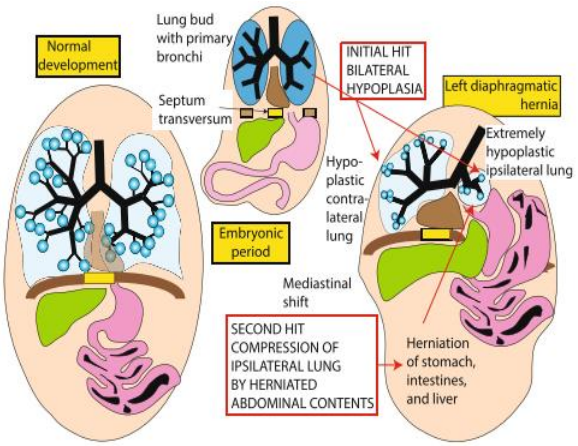

Figure 1. CDH hypothesis.[5]

In CDH, the total pulmonary vascularity decreases with a decrease in the number of vessels per lung unit. In addition, remodeling of the pulmonary vessels with medial hyperplasia and peripheral extension of the muscle layer into small arterioles. The lack of pulmonary vasculature and vascular remodeling contributes to this PPHN being fixed or irreversible in CDH. After birth, the combination of pulmonary arterial hypertension, hypertrophy with right ventricular failure, and left ventricular hypoplasia with pulmonary venous hypertension results in PPHN unresponsive to conventional management.[14]

Ventricular dysfunction is observed in some patients with severe PPHN because of CDH. During fetal life, the ductus arteriosus functions as a pop-off value and limits right ventricular stretch. After birth, remodeling Pulmonary vessels in CDH cause pulmonary hypertension and cause right ventricular dysfunction. Left ventricular abnormalities have also been reported in infants with CDH. When compared with other neonates, infants with left-sided CDH have significantly lower left ventricular mass. Decreased left ventricular output has been seen in the congenital left and right-sided diaphragms. Reduced left ventricular mass contributes to functional left ventricular hypoplasia and can lead to increased left atrial pressure and pulmonary venous hypertension (Figure 2).[5]

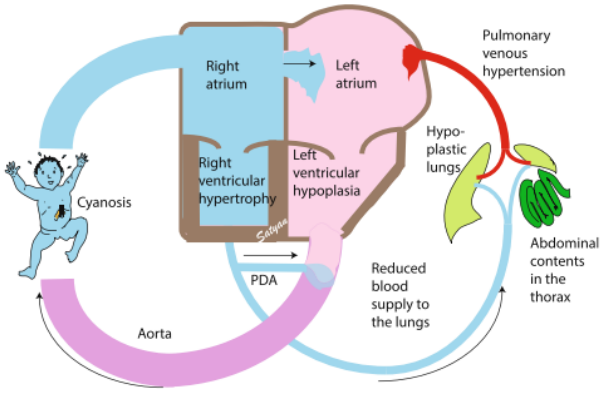

Figure 2. Cardiovascular effects of CDH.[5]

Prenatal diagnosis by ultrasound detects more than 50% of cases of CDH at a mean gestational age of 24 weeks. Three-dimensional ultrasound, fetal echocardiography, and fetal MRI are other prenatal diagnostic modalities used in assessing the severity and prognosis of CDH. High-resolution ultrasound should be used to assess severity to assist in parental counseling. This usually includes measuring lung volumes and liver position and looking for significant comorbidities.[8] If not diagnosed antenatally, the early signs of CDH will usually occur soon after birth with respiratory distress, as well as possibly a scaphoid abdomen. A chest radiograph will usually show the abdominal organs within the chest cavity.[8]

Lung-to-head ratio (LHR) is often used to predict the outcome of CDH. Obstetrical ultrasound techniques are used in measuring LHR where calculating LHR at 22-23 weeks or 32-33 weeks of gestation can be used as an early assessment of fetal lung development and predicts short-term survival or morbidity. LHR < 1 is associated with increased mortality, need for ECMO, and incidence of chronic lung disease. LHR <1.35 is associated with a poor outcome, compared to a ratio >1.35, which has a better outcome. However, LHR measurements varied greatly by gestational age and were inconsistent across the centers studied observed to expected lung-to–head ratio (O/E LHR) independent of gestational age. Currently, O/E LHR examined by ultrasound is the most accepted method of evaluating lung size.[16]

Postnatal management includes the administration of surfactant, preferably immediately when the baby takes its first breath, and surgical treatment after birth needs to consider three things, namely the benefits of surgery, the optimal time for surgery, and the best surgical approach (Figure 10).[15] Davis et al. stated that pre-prepared surgery followed by ECMO therapy gave better results. The exact time to perform surgery is not known with certainty, some experts recommend that surgery be performed 24 hours after the baby is stable, but a delay of up to 7-10 days can also be tolerated.[15]

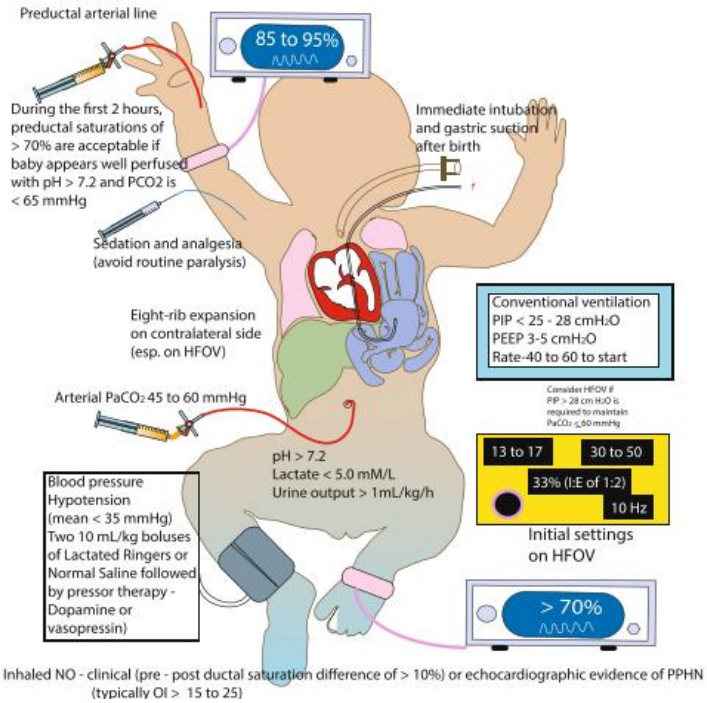

Figure 3. Treatment of CDH.[17]

Current anesthesia management of diaphragmatic hernia

The delivery process and neonatal intensive care unit

Babies should be born in health centers with the ability to manage CDH babies and related complications that have adequate pediatric surgery and perinatology facilities. In general, the facilities needed are endotracheal intubation and the use of a mechanical ventilator according to the severity of the abdominal organ herniation. The use of manual bag ventilation should be avoided because the stomach and intestinal organs will be distended by air which results in further pressure on the lungs and intrathoracic organs. Installation of a nasogastric tube for decompression, as well as avoiding the use of high inspiratory pressures.[2] Ventilation using a T-piece resuscitation is preferred to avoid high airway pressures. Peak inspiratory pressure (PIP) should be below 25 cm H_2O to avoid damage to hypoplastic/immature lungs. A pre-ductal oximeter is placed in the right upper extremity as soon as possible, where a preductal saturation > 70% is acceptable for the first 1-2 hours if arterial pH and carbon dioxide for $PaCO_2$ are within normal limits.[1]

Preoperative stabilization

In CDH babies, there are hypoplastic lungs, no atelectasis of abnormal arteriolar vascularization, and pulmonary hypertension, so that surgery is considered postponed or prepared in advance. The average lifespan for surgery is about 72 hours.[10] Central or peripheral venous access is obtained for the administration of fluids and medications. Placement of a preductal artery line to monitor blood pressure and collect blood gases is necessary, preferably in the right radial or ulnar artery. Umbilical artery values reflect postductal arterial oxygen tension (PaO_2) and cause an increase in inspired oxygen fraction (FIO_2). Systemic blood pressure is maintained at normal values for gestational age. Pre-ductal saturation is maintained between 85-95%. A chest X-ray is obtained to assess the initial condition of the lungs and the contents of the hernia.[1]

Conventional mechanical ventilation

The provision of mechanical ventilation must consider factors that increase pulmonary vascular resistance (hypoxia, acidosis, hypotension, and hypercarbia). Low-pressure inspiratory ventilation was chosen because it reduces the possibility of a contralateral pneumothorax which can increase cardiorespiratory system instability and decompensation. If conventional mechanical ventilation fails, another ventilation strategy is used, namely high-frequency oscillatory ventilation (HFOV), Gentle ventilation dan intratracheal pulmonary ventilation (ITPV). In addition to the ventilation strategy, supporting therapy is also needed to support the success of the surgery and improve the prognosis.[11]

Optimal ventilation for CDH infants and hypoplastic lungs is unknown. Many centers start Conventional Mechanical ventilation (CMV) for respiratory support and optimize ventilation by adjusting PIP and respiratory rate. VICI trial (Ventilation in infants with a congenital diaphragmatic hernia) recently compared CMV and HFOV or high-frequency oscillatory ventilation as the initial mode of ventilation in CDH. The results obtained showed no statistically significant difference in the outcome of death or bronchopulmonary dysplasia (BPD). The study was conducted on 91 patients receiving CMV and 80 patients getting HFOV. A total of 41 patients (45.1%) with CMV died or had BPD compared with 43 patients (53.8%) in the HFOV group, with an odds ratio of 0.62 (P = 0.31) for death/BPD for CMV vs. HFOV. Patients initially ventilated by CMV had fewer ventilation days (P = 0.03) and also required less ECMO support (P = 0.007), inhaled nitric oxide (iNO, P = 0.045), sildenafil (P = 0.004), had a shorter duration than vasoactive drugs (P = 0.02), and fewer medication failures (P = 0.01) compared with infants initially ventilated by HFOV. It is important to note that the guidelines for the initial management of CMV in this study included low positive end-expiratory pressure (PEEP) (3 – 5 cm H_2O) and PIP (20-25 cm H_2O).

These findings suggest that early efforts to CMV are more rational for patients with CDH. An autopsy review of 68 infants with CDH showed significant lung

injury (alveolar damage, hyaline membrane formation, pneumothorax in 2/3 autopsies) secondary to mechanical ventilation in which 53 infants were switched to HFOV within an average of 15 hours from birth. To prevent volutrauma and barotrauma, a gentler ventilation approach is preferred in infants with CDH. Mode CMV with PIP is usually under 25cm H_2O and PEEP = 5 cm H_2O by targeting >85% preductal saturation, >70% post-ductal saturation, and $PaCO_2$ 45-60 mm Hg is used to initiate ventilation. Many centers turn to HFOV or jet ventilation as an alternative therapy if ventilation targets cannot be achieved on CMV. Adjustment on HFOV by maintaining the mean airway pressure (MAP) is usually adjusted to maintain adequate inflation of the lung contralateral to the 8th rib in the range of 13-17 cm H_2O.[1]

Extracorporeal membrane oxygenation (ECMO)

ECMO is used in CDH infants who experience a worsening clinical status, often secondary to a pulmonary hypertensive crisis. Indications for the use of ECMO are not uniform. Some relative indications for ECMO include increased oxygenation index (OI), persistently low oxygen saturation despite maximal ventilator support, hypercarbia, and increased alveolar-arterial oxygen gradient (A-aDO2).[10]

Possible indications for ECMO if the following conditions are met at two consecutive time points for at least 3 hours; inability to maintain a preductal oxygen saturation above 85% (52 mmHg or 7 kPa) or a postductal oxygen saturation above 70% (40 mmHg or 5.3 kPa); increased $PaCO_2$ > 65 mmHg or 8.5 kPa despite optimized ventilation management; Peak inspiratory ventilator pressure (PIP) > 28 cm H_2O; average airway pressure (MAP) > 17 cm H_2O; inadequate oxygen delivery with metabolic acidosis defined as lactate ≥5 mmol/L and pH <7.20; 6) Hypotension resistant to fluid therapy and inotropic support resulting in urine output <0.5 ml/kg/hour; 7) oxygenation index ≥ 40.[14] ECMO contraindications are significant congenital anomalies (e.g., severe cardiac lesions) and lethal chromosomal abnormalities irreversible brain damage, uncontrolled bleeding, and intraventricular hemorrhage of degree III or greater. Other relative contraindications include less than 2 kg body weight, less than 32-34 weeks of gestation, and a high probability of poor prognosis.[4]

Hemodynamic monitoring and management

Invasive hemodynamic monitoring is preferable to non-invasive monitoring. In addition, monitoring of saturation before and after surgery should be continuously monitored. The goal of hemodynamic monitoring in infants with CDH is to achieve optimal end-organ perfusion. Signs of adequate perfusion include normal heart rate range for gestational age, normal capillary refill, urine output >1.0 ml/kg/hour, arterial pH >7.2, and lactate level <3-5 mmol/L. If there are signs indicating poor perfusion, volume resuscitation, and vasopressor therapy should be considered. Assessment of cardiac function is assessed by echocardiogram and volume requirements; in cases of hypovolemia, a bolus of isotonic solutions, such as 0.9% normal saline or Ringer's lactate solution, 10 ml/kg intravenously, can be given. Volume resuscitation is usually followed by vasopressor/inotropic therapy.[13]

Vasopressor and inotropic therapy

Dopamine is the most commonly used cardiovascular drug in the NICU and is administered as an infusion for the purpose of maintaining systemic blood pressure appropriate for gestational age. Dobutamine is better in infants with poor myocardial contractility. Norepinephrine and epinephrine can be used as first-line agents in some institutions, secondary to potent vasoconstrictors. Incorrect epinephrine infusions may increase lactate levels and may interfere with management. Low-dose hydrocortisone is useful in vasopressor-resistant hypotension in the immediate postpartum period. Vasopressin was reported to be effective in stabilizing systemic hemodynamics in a retrospective chart review with reduced pulmonary/systemic pressure ratio in patients with CDH.[10]

Treatment of pulmonary hypertension

PPHN in infants CDH is a secondary complication due to hypoplastic lungs and experiencing blood vessel

remodeling. Pulmonary arterial hypertension, together with left ventricular hypoplasia and right ventricular hypertrophy and/or failure complicated by pulmonary venous hypertension, lead to severe PPHN unresponsive to conventional therapy. Because of the right-to-left shunt, differences in pre and post-ductal saturation can be observed. In some patients with the immediate postnatal phase of CDH, there is a brief period of better oxygenation referred to as the honeymoon period. However, a progressive decrease in oxygenation is usually observed with worsening PPHN. An echocardiogram is the best non-invasive test to assess cardiac function and pulmonary pressure in infants with CDH and is usually performed within the first 24 hours and followed up as needed.[9]

If preductal saturation falls below 85%, adjustment of ventilation and hemodynamic management should precede the initiation of any therapy. Measures to increase systemic blood pressure can minimize right-to-left shunt. However, there is no need to increase blood pressure if the preductal saturation remains above 80%. Catecholamines, especially dopamine, in addition to increasing systemic vascular resistance, also increase pulmonary vascular resistance. The consortium recommends maintaining arterial blood pressure at normal levels for gestational age if preductal saturation is between 80 and 95%.

Treatment of pulmonary hypertension with inhalation of nitric oxide (iNO), which is the agent of first choice in infants> 34 weeks of gestation. It is a selective pulmonary vasodilator and relaxes the smooth muscle cells of the pulmonary vessels. The criteria for initiating iNO were based on the severity of PPHN as assessed by the oxygenation index (OI). The oxygen saturation index (OSI) is a non-invasive way of estimating oxygenation status and can be used in the absence of arterial blood gases, but it requires further validation. Previous studies have reported an average OI of 25 ± 9 as cut off for iNO initiation. Currently, in neonates with PPHN not due to CDH, it is acceptable to initiate iNO with OI = 20 and evidence of right-to-left shunt by clinical examination (pre-post-ductal saturation difference = 10%) and/or echocardiographic evidence of extrapulmonary right-to-left shunt. The complete response to iNO is considered to be an increase in the ratio of arterial oxygen tension (PaO_2) to the fraction of inspired oxygen (FiO_2) = 20 mmHg after iNO therapy.[1]

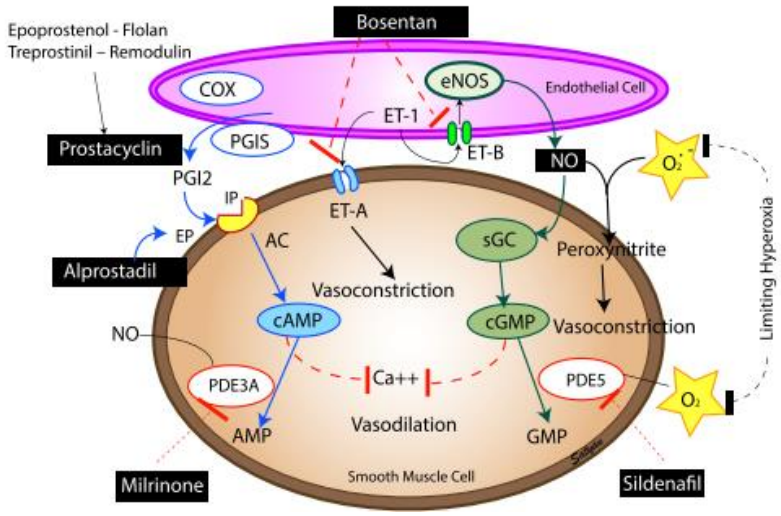

Figure 4. Management of pulmonary hypertension in CDH.

In contrast to PPHN from conditions other than CDH, iNO did not reduce ECMO requirements or mortality in a prospective randomized trial of CDH infants. Despite these negative studies, iNO continues to be used in US tertiary centers in the management of infants with CDH with no change in ECMO utilization or mortality. If there is no response to iNO after optimizing ventilation and hemodynamic status, iNO is gradually weaned off. Some patients decompensate and become hypoxemic with the

discontinuation of iNO. In this case, iNO was weaned to a low dose for several hours and then discontinued.[11]

iNO with high concentrations of oxygen has the detrimental side effect of forming peroxynitrite, which is a vasoconstrictor and is toxic. Thus, continuing iNO therapy in the absence of a response is controversial, and side effects must be considered.[15] Infants with corrected CDH are at risk of developing advanced pulmonary hypertension, but inhalation of nitric oxide may play an important role in treating exacerbations of pulmonary hypertension in these patients.[1]

Intravenous prostaglandins (PGE1) have been used primarily in the setting of right heart failure to maintain ductal patency. PGE1 trials to reopen the ducts can reduce the load on the right ventricle. PGE1 infusion was started when the duration of the right-to-left shunt through the ductus arteriosus was longer than the left-to-right shunt. Inhaled PGE1 is also used as an alternative agent in treating PPHN, but this therapy is not FDA-approved.[1]

Prostacyclin (PGI), commonly used in adults, may be useful in the management of advanced pulmonary hypertension in infants after CDH repair. There is no evidence to support this therapy in infants, but some centers use it as a second-line pulmonary vasodilator. Prostacyclin can be used as an inhalation agent or an intravenous agent. Three forms of prostacyclin are used in the management of pulmonary hypertension. Epoprostenol (Flolan), Treprostinil (Remodulin), and inhaled Iloprost (Ventavis-analogue of inhaled prostacyclin) are approved for adults with pulmonary arterial hypertension.[1]

Sildenafil is a phosphodiesterase (PDE)5 inhibitor that inhibits degradationguanosine monophosphate (cGMP), which causes vasodilation. Oral sildenafil increases oxygenation and reduces mortality in PPHN in health facilities where iNO and ECMO are not available, and sildenafil has been shown to be effective in increasing oxygenation in patients with PPHN with and without prior exposure to iNO. There are no trials to support its use in infants with CDH. According to the FDA, high mortality is associated with the use of sildenafil in chronic CDH pediatric patients (age 1-17 years) with pulmonary arterial hypertension. Parents should be informed about the benefits and side effects of sildenafil before starting use in chronic CDH patients.[1]

Milrinone is an inhibitor of PDE 3, which increases the concentration of cyclic adenosine monophosphate (cAMP) in smooth muscle and myocardium, and has lusitropic and inotropic properties. In the PPHN fetal sheep model, milrinone relaxes the pulmonary arteries and reduces pulmonary artery pressure. This therapy has been used in the management of iNO-resistant PPHN in CDH infants with hypotensive side effects. The initial dose (50 g/kg over 30-60 minutes) was followed by a maintenance dose (0.33 g/kg per minute and escalated to 0.66 and then to 1 g/kg per minute based on response). This initial dose increases the risk of hypotension but may achieve stable plasma levels more rapidly. Usually, give a volume bolus before the initial dose to avoid systemic hypotension.[1]

Bosentan is an endothelin receptor blocker and is sometimes used as an oral agent in the management of chronic pulmonary hypertension in CDH. There is limited experience with its use in neonates. Liver function tests must be followed closely during their use. Extracorporeal membranous oxygenation (ECMO) is considered a last resort for saving the life of infants at 34 weeks gestation or weighing >2 kg with CDH and no severe anomaly after the failure of conventional medical management.[1]

Sedation and analgesia

Neonates with severe pulmonary hypertension will require sedation and analgesia to facilitate optimal ventilation. Pain score assessments need to be recorded at least every four hours. Optimized administration of analgesia and sedation with narcotic infusions is usually initiated in the ventilated neonate. Clinical evidence of ongoing pain and/or distress (pain score) should be managed with additional boluses, increased infusion rate, and consideration of other drugs or additional sedation. In addition, muscle relaxation by bolus or infusion should be considered in any neonate who is difficult to stabilize. Midazolam infusion may also be considered in neonates requiring muscle relaxation or increased doses of morphine.[18]

Giving fluids and nutrition intake

Some recommendations for providing fluids and nutrition to babies with CDH are:[18] restrictive fluid management in the first 24 hours of 40 mL/kg/day of fluids; the concentration of glucose in the intravenous fluids may need to be increased to ensure an adequate glucose infusion rate is provided; fluid and caloric intake should be increased based on the clinical condition; diuretics should be considered if the fluid balance is too positive, especially if the urine output is low, provided the cardiovascular status is stable; early parenteral nutrition is recommended; enteral feeding should be started postoperatively in combination with anti-reflux drugs; colostrum can be started from birth at trophic levels (no more than 1 mL in 4 hours) through a gastric tube. Patency and continuous free drainage of the gastric tube must be maintained to allow adequate ventilation.

2. Conclusion

The mortality of diaphragmatic congenital hernia is highly dependent on the state of development and function of the lungs and can be predicted at the time of intrauterine using ultrasound or at the postnatal time using oxygenation parameters. Early stabilization of the patient is a priority before performing a surgical intervention for the hernia organ.

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