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Hemodynamic Phenotypes and Intensive Care Support in Neonates with Congenital Diaphragmatic Hernia: Single-Center Experience without ECMO

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Abstract

Introduction: Congenital diaphragmatic hernia (CDH) is a condition characterized by a defect in the diaphragm that causes the protrusion of abdominal contents into the chest cavity, associated with pulmonary hypoplasia, persistent pulmonary hypertension and cardiovascular dysfunction. In centers without extracorporeal membrane (ECMO) availability, management depends on individualized ventilatory and hemodynamic support strategies.

Objective: To describe hemodynamic phenotypes, cardiorespiratory support strategies and clinical outcomes of newborns with CDH treated in a tertiary care hospital without ECMO.

Methods: An observational, descriptive and retrospective study that included 10 newborns with a confirmed diagnosis of CDH admitted to the neonatology service in a tertiary care hospital between January 2025 and May 2026. Demographic characteristics, type of anatomical defect, ventilatory requirements, vasoactive support and clinical outcomes were analyzed.

Results: The mean gestational age was 37 weeks; the mean birth weight was 2864g. The left location was the most frequent (90%). Reaching a survival rate of 80%. Patients were stratified into three groups according to the intensity of cardiorespiratory support: conventional support, advanced support without milrinone and advanced combined support. Patients who required advanced combined support represented the group with the highest cardiorespiratory compromise, concentrating all the events with the highest mortality and ventilatory support requirements. The two deaths occurred in patients with anatomical factors of very high risk: a type D defect and an intrathoracic hepatic herniation.

Conclusions: The intensity of cardiorespiratory support allowed us to identify different hemodynamic phenotypes associated with different clinical severity and outcome. The need for advanced combined support seems to be a marker of greater pathophysiological severity in newborns with CDH. These findings may contribute to optimizing clinical stratification and therapeutic decision-making in centers without ECMO availability.

Keywords: Congenital diaphragmatic hernia; Pulmonary hypertension; Milrinone; Norepinephrine; High-frequency oscillatory ventilation; Phenotypes

Introduction

Congenital diaphragmatic hernia (CDH) is a condition characterized by a defect in the diaphragm that causes the protrusion of the abdominal contents into the chest cavity, which interferes with the normal development of the lungs, conditioning different degrees of pulmonary hypoplasia and alterations in pulmonary vascular development. These alterations determine the appearance of persistent pulmonary hypertension in the newborn and in many cases ventricular dysfunction, configuring a highly serious clinical picture. This involvement can present as an isolated lesion or as part of a syndrome [1]. This pathology

usually manifests itself with respiratory distress in the first hours of life, it can be mild, moderate, severe; The current approach has evolved towards protective pulmonary ventilation strategies, with the aim of minimizing ventilator-induced lung damage, as well as towards individualized hemodynamic management based on the predominant pathophysiology of each patient [2]. This includes the selective use of inotropes and vasopressors such as dobutamine, norepinephrine, and milrinone. In this scenario, the analysis of clinical series acquires special relevance, since it allows characterizing the real behavior of the disease in a specific care context. The objective of this study is to describe the hemodynamic phenotypes, cardiorespiratory support strategies and clinical outcomes of newborns with congenital diaphragmatic hernia treated in a tertiary care hospital without ECMO availability.

Epidemiology

Congenital diaphragmatic hernia is a rare, serious defect that occurs with an incidence of 2.3 per 10,000 live births. Overall survival rates for patients with congenital diaphragmatic hernia range from 65 to 80% and are influenced by a variety of factors, and further improvement can be achieved through evidence-based management strategies and standardized protocols. However, diaphragmatic hernia covers a wide spectrum of severity and the most complex and high-risk cases, so it still has a survival rate close to 50% [1]. Bochdlaek's hernias are the most common type, accounting for 70-75%, occurring mostly on the left side and less frequently on the right side with 13% or bilaterally with 2%2. The other types are anterior defects or Morgagni hernias, which represent 23-28%, and central hernias 2-7% [2]. The Diaphragmatic Hernia Study Group agreed on a standardized classification scheme for the size of the defect observed during hernia repair. Defects are classified as defects: type A when completely surrounded by diaphragmatic muscle; type B if less than 50% of the chest wall lacks a diaphragm; type C if more than 50% of the chest wall lacks a diaphragm and type D diaphragmatic agenesis. Diaphragm size is the main determinant of respiratory, gastrointestinal and neurological morbidity and, together with a coexisting major cardiac anomaly, is the general determinant of survival, the larger the defect and an adverse outcome is due to the greater magnitude of herniation of the intrathoracic organs at the beginning of management, which ultimately results in more severe pulmonary hypoplasia at birth [3].

Diagnosis

Prenatal diagnosis in up to 60% of newborns is described by routine prenatal ultrasound: between 18- and 22-weeks' gestation, polyhydramnios, or hydrops fetalis, is usually found. However, the presence of abdominal organs in the chest characterizes the diagnosis of diaphragmatic hernia. In the postnatal stage, chest x-ray shows herniation of the abdominal organs, usually intestines characterized by structures containing air or fluid, towards the right or left hemithorax, with minimal visible pulmonary aeration on the affected side, cardiac displacement to counteract the effect of mass on the contralateral lung, and reduction of abdominal circumference. It is also evident that the feeding tube remains inside the chest cavity or the left mediastinal displacement in the right diaphragmatic hernia. Occasionally, hepatic hernia is the only sign that suggests a right diaphragmatic hernia in such cases a large solid mass in the right chest [4].

Persistent pulmonary hypertension in congenital diaphragmatic hernia

In HDC, the total pulmonary vascular bed is reduced, there is a decrease in the number of vessels in the lung. In addition, pulmonary vascular remodeling is evidenced with hyperplasia of the muscle layer, pulmonary vasculature deficiency, and vascular remodeling; together they contribute to the development of HPPRN. It is also important to mention that altered vasoreactivity is possibly due to an imbalance of autonomic innervation with increased sympathetic innervation and decreased parasympathetic innervation, and altered relaxation of the endothelium of the pulmonary arteries leading to an imbalance between vasoconstrictor and vasodilator mediators. PVR can also increase due to circumstances such as hyperinflation, atelectasis, acidosis, hypothermia, inadequate sedation, so active management focuses on allowing the fetal-neonatal transition to occur as smoothly as possible avoiding the iatrogenic factors that increase it. Maintaining the main focus of gentle ventilation will allow PVR to decrease gradually and naturally until pulmonary vascular pressures have decreased sufficient to receive the entire output of the right ventricle, prostaglandin E1 can be used to keep the ductus arteriosus open and divert the passage of deoxygenated blood to the descending aorta; it is essential to accept low posductal PO2 or low oxygen saturations as they are necessary consequences of using the ductus arteriosus as an escape route for the right ventricle, so saturations, PO2, Aa gradients, and lactate levels used to assess progress and adequate tissue perfusion should come from preductal sources [4]. When ventilation is not enough to reduce pulmonary hypertension, the use of inhaled pulmonary vasodilators such as nitric oxide can be considered for greater vasodilation or if you do not have this medication make use of other vasodilators and although it is not specific to the lungs, the use of milrinone is increasingly accepted. However, it is also vital to be careful of the adverse effects of pulmonary vasodilators as efforts to increase pulmonary blood flow can overload the underdeveloped left heart. Signs of this pathology include pulmonary edema and exacerbated cardiac dysfunction

that led to the requirement for the use of inotropes and/or diuretics to prevent these side [3,10]

After birth a combination of pulmonary arterial hypertension, right heart failure, left ventricular hypoplasia results in severe PPHN that does not respond to conventional treatment. This condition causes dysfunction in the pulmonary circulation, alteration of gas exchange and exacerbation of cardiac dysfunction. In this pathology, pulmonary hypertension is due to abnormal prenatal development of the pulmonary vasculature characterized by hypertrophic smooth muscle cells, vascular thickening, and reduced angiogenesis [14]. These changes result in elevated pressures in the right heart, circulatory bypass, poor ventilation, and decreased oxygenation after birth. The results of a study have shown that 80% of newborns with this pathology will develop pulmonary hypertension and of these 38% require extracorporeal support3. This underscores the importance of pulmonary hypertension as a risk factor and its critical role in the management and prognosis of congenital diaphragmatic hernia.

Fetal predictors of outcomes

The main determinants of the results in CDH are the presence of associated abnormalities, especially heart disease, the degree of pulmonary hypoplasia and the position of the liver. The prognosis of isolated CDH is usually better than that of complicated CDH with multiple abnormalities. Liver hernia is associated with a worse prognosis, studies have revealed a higher survival rate of HDC without a liver in the chest of 74% compared to liver herniation, which is 45%, in addition to the fact that liver hernia is highly predictive of ECMO (80% liver in the chest vs 25% liver down) and survival (93% liver down and 45% liver in the chest).

Treatment

Neonatal care for congenital diaphragmatic hernia focuses on three domains: pulmonary hypoplasia, pulmonary hypertension, and biventricular heart dysfunction.

Delivery room

The resuscitation of a CHD is based on the neonatal resuscitation manual (NRP) which indicates that endotracheal intubation should be immediate for neonates with a known diagnosis of CHD, avoiding ventilation with a bag-valve-mask due to the high risk of hollow visceral insufflation [15]. A T-piece should be used in the bag-valve mask to rigorously avoid a maximum inspiratory pressure (PIP) greater than 25cmH20 from the first breaths in all newborns with HDC15. Place the oxygen saturator in the preductal extremity, it is important to mention that the first 2 hours of life the preductal saturation can be more than 70% is acceptable as long as it is well perfused with pH >7.2 and PCO2 < 65mmHg3. It is essential to place an orogastric tube to decompress the intestine, as well as to obtain a central venous access to administer fluids or medications, an arterial line is required to obtain blood gases, although an umbilical arterial line has traditionally been placed, it may be preferable to obtain a preductal arterial line in the right radial or ulnar artery since the values of the umbilical arterial line reflect the postductal oxygen blood pressure.

Ventilation Mode

Protective pulmonary ventilation is the fundamental pillar in the respiratory management of congenital diaphragmatic hernia, aimed at minimizing ventilator-induced lung damage in the context of structurally hypoplastic lungs that are highly susceptible to volutrauma and barotrauma. This approach prioritizes the use of low tidal volumes (3-5ml/kg), limited inspiratory pressure, and acceptance of permissive hypercapnia (PaCO2 up to 60-65mmHg) in order to avoid alveolar overdistension and preserve the integrity of the remaining lung parenchyma; in addition, using a conservative oxygenation strategy, accepting preductal saturations between 85-95%, thus avoiding hyperoxia, which further aggravates pulmonary vasculopathy. Conventional mechanical ventilation (CMV) should be the initial ventilation method for all infants with CHD, high-frequency oscillatory ventilation or high-frequency jet ventilation should be used as rescue therapy when the pressure required to control hypercapnia by CMV exceeds 25 cmh20 or if a partial pressure of carbon dioxide between < 65 mmHg and a pH between 7.25 and 7.403 is not achieved. There are multiple studies in which the rates of mortality and bronchopulmonary dysplasia between the groups initially treated with conventional mechanical ventilation and high-frequency oscillatory ventilation were similar. It has not been possible to demonstrate the difference in survival, duration of mechanical ventilation or oxygen requirement at discharge between these two groups of patients. Despite this, low positive end-expiratory pressures are essential to maintain the residual functional capacity required by hypoplastic lungs. A volume-based inspiratory strategy allows for instantaneous titration of pressure to dynamic postnatal compliance. The role of surfactant in a retrospective analysis fails to support any beneficial effect of surfactant replacement therapy in term newborns with HDC, and its uses in preterm infants are also associated with a lower survival rate; however, prospective studies are needed to evaluate the benefits of surfactant.

Hemodynamic management

Cardiac dysfunction in this pathology is bilateral and consists of an overloaded right ventricle and an underdeveloped left ventricle. Medical treatment consists of relieving unnecessary after loading on the right side and unnecessary preloading on the left side with the help of prostaglandin E1 that allows fetal circulation to continue. Optimal perfusion of the target organs is

the goal of hemodynamic monitoring of these patients, signs of adequate perfusion include a normal heart rate range for gestational age, normal capillary filling, diuresis of more than 1mlkgh pH > 7.2 and lactate levels < 3.5 mmol/L2. It is essential that treatment of the hemodynamic instability of a CDH on early inotropic support with the prudent use of fluids is essential to prevent pulmonary edema as ventricular dysfunction contributes significantly to persistent hypotension that is exacerbated by excessive fluid resuscitation. Although the choice of inotropic agent depends on the clinical status of the patient, adrenaline and noradrenaline are still considered the first-line options for cardiac support used in the appropriate dose since a higher dose of adrenaline can cause adverse events such as tachyarrhythmias, hyperglycemia and lactic acidosis by acting on alpha receptors, so its use is recommended only in inotropic doses. On the other hand, norepinephrine only has vasoconstrictor effects, allowing it to increase peripheral vascular resistance, the use of dopamine is not recommended due to the adverse effect it has on raising lung pressure .Cardiovascular management, introduction, suspension, and precise titration of each drug should be carried out within the framework of specific hemodynamic management, treatment will be individualized to meet the unique needs and responses of each newborn and their specific cardiovascular status.

There is evidence that the underlying cardiovascular phenotype may vary between different patients with CDH. This phenotype may evolve during the early acute phase of hospital admission, underscoring the need for continuous multidisciplinary surveillance and the use of clinical information including ultrasound at the patient's bedside. However, although various phenotypes have been documented, no cohort studies of CDH have defined the benefits of employing specific cardiovascular management strategies for pulmonary hypertension, right ventricular dysfunction, left ventricular dysfunction, or biventricular dysfunction in this population. According to the American academic of Pedai It is recommended to perform an echocardiogram after birth, not only to check for possible suspected cardiac abnormalities but also to evaluate ventricular function, therefore at least 2 standardized echocardiograms should be performed, one between 24 and 48 hours of life and another between 2 and 3 weeks of life to evaluate your cardiac function, a repeat echocardiogram is indicated on days 5 and 7 years of life when there is clinical evidence of progression or improvement of pulmonary hypertension. PGE1 infusions should be used if pulmonary or systemic flow depends on the patency of the ductus arteriosus or in the presence of a concomitant anatomical cardiac lesion or PGE1 may be considered in the presence of suprasystemic right ventricular pressures or in case of right ventricular failure if the right-to-left ductal shunt exceeds the left-to-right shunt [7].

The use of targeted pulmonary vasodilator therapy is recommended in the context of CDH-associated pulmonary hypertension when standard cardiorespiratory maneuvers fail to maintain adequate oxygenation or cardiac function1. Inhaled nitric oxide may be considered part of the treatment regimen only if it demonstrates echocardiographic and clinical improvement, which should otherwise be discontinued. Milrinone, on the other hand, is a lusitropic drug that improves diastolic function and at the same time causes pulmonary and systemic vascular dilation, it also provides assistance to the compromised left ventricle, for all these properties milrinone is increasingly recommended in the management of PPHN with HDC. Milrinone is a phosphodiesterase 3 inhibitor that increases the concentration of cyclic adenosine monophosphate (cAMP) in smooth muscle and myocardium. It has inotropic lusitropic properties by relaxing the pulmonary arteries and decreasing the pressure of the pulmonary artery. Multiple case series have demonstrated the efficacy of intravenous milrinone in the treatment of nitric oxide-resistant PPHN [10,11]. Milrinone therapy has been used in the treatment of nitric oxide-resentful PPHN with HDC; it is usually commonly used in loading doses of 50mcgkg in 30 to 60 min, followed by maintenance doses of 0.33 to 0.66mcgkgmin; however, the loading dose of milrinone increases the risk of hypotension, so this loading dose is not recommended in patients with HDC with systemic hypotension. Extracorporeal membrane oxygenation is considered the last option to save the lives of newborns over 34 weeks of gestation or weighing more than 2kg with CDH whose admission criteria are as follows: inability to maintain preductal saturations >85% or postductal saturations > 70% together with increased PaCO2 and respiratory acidosis with pH <7.15 despite optimal ventilatory management PIP >28 cm H20 or MAP >17 to achieve saturations >85% or inadequate oxygen supply with metabolic acidosis or systemic hypotension resistant to fluid therapy resulting in urine output < 0.5mlkgh over a 12- to 24-hour period or IO being >40 [2].

Admission to surgery

The optimal time for HDC repair can be difficult to determine and is usually not offered before 48 to 72 hours of age. Delaying surgical repair until physiological stability is achieved, interpreted as cardiorespiratory function and sufficient oxygenation to avoid lactic acidosis with evidence of subsystemic pulmonary artery pressure, seems to improve the prognosis of CDH. The following criteria must be met before surgery: urinary output > 1mlkgh, FiO2 < 50%, oxygen saturation between 85 and 95%, blood pressure in P50 lactate <3 mmol/L, and pulmonary arterial pressures lower than systemic. We also have the oxygenation index as an indicator of physiology and it is useful to determine the time of surgical repair in the HDC, an IO < 9.4 led to an

increase in the days of mechanical ventilation and a delay in hospital discharge.

Methodology

An observational, descriptive and retrospective study of a cohort of newborns diagnosed with congenital diaphragmatic hernia admitted to the Neonatology Unit of the Nueva Aurora Luz Elena Arismendi Pediatric Obstetric Gynecological Hospital in Quito, Ecuador, was conducted. All newborns with confirmed diagnoses of congenital diaphragmatic hernia seen between January 2025 and May 2026 were included. The diagnosis was established through clinical and radiological findings and surgical confirmation. Patients with congenital malformations of the airway and pulmonary tract were excluded.

Results

10 newborns with a confirmed diagnosis of CDH were identified during the study period. The mean gestational age was 37.8 weeks and the mean birth weight was 2864g. The left location was predominant (90%) while prenatal diagnosis was present in 40% of cases. Overall survival was 80%. All patients with complex anatomical defects (type C, D or intrathoracic hepatic herniation) required advanced cardiorespiratory support, while patients with type B defects had a more favorable evolution and less need for combined therapeutic strategies. Mortality was observed exclusively in the advanced combined support group (33.3%), while no deaths were recorded in the conventional or advanced support groups without milrinone.

Discussion

This study included 10 newborns diagnosed with congenital diaphragmatic hernia. The mean gestational age was 37 weeks and the mean birth weight was 2864g. 50% were male and only 40% had a prenatal diagnosis. The left location was the most frequent, being observed in 90% of cases. In order to evaluate the relationship between clinical severity and therapeutic requirements, patients were stratified into 3 groups according to the intensity of cardiorespiratory support received: conventional support, advanced support without milrinone, and advanced combined support (milrinone, norepinephrine, and high-frequency oscillatory ventilation). In this cohort of 10 highly complex cases, it is observed that milrinone was used both in the preoperative and postoperative phases, with durations reaching up to 9 days preoperatively and 5 days later, suggesting a strategy aimed at optimizing myocardial function and reducing pulmonary vascular resistance in patients with left ventricular dysfunction. In congenital diaphragmatic hernia, milrinone is described as being used in cases of pulmonary hypertension with left ventricular dysfunction as it provides inotropism, lusitropism and pulmonary vasodilation; At the same time, norepinephrine is reserved for scenarios of systemic hypotension or the need to increase systemic vascular resistance to improve the relationship between systemic and pulmonary pressure and decrease right-to-left shunting. This pathophysiological approach is precisely the one reflected in this cohort. The evidence does not demonstrate universal benefit in all patients with CDH. A retrospective study in neonates with mild to moderate CDH did not find significant improvement in oxygenation index and pulmonary arterial pressure, although no adverse effects were observed with the use of milrinone.

In other words, milrinone does not seem to be the general solution for all cases, but rather a more useful drug in selected patients when there is a ventricular dysfunction phenotype associated with pulmonary hypertension. Therefore, milrinone is used in some patients and not in all, it is consistent with the trend of individualized therapy and guided by hemodynamic phenotype rather than an indiscriminate routine use as observed in the cohort analyzed. The evidence is still insufficient to confirm definitive clinical efficacy in this population; the literature says that milrinone has a solid physiological basis but robust trials are still lacking to establish with certainty which subgroups improve hard outcomes. Long-term use of milrinone in some patients should be interpreted more as a marker of severity and the need for targeted support. Norepinephrine can be used to raise systemic blood pressure with potential improvement of oxygenation in severe pulmonary hypertension, however, it should be used selectively, not as a vasopressor for all cases of CDH. In other words, this series reproduces the pattern described in highly complex centers: milrinone as support in the phenotype of myocardial dysfunction and pulmonary hypertension and norepinephrine as support when systemic hypotension predominates or blood pressure needs to be sustained to optimize hemodynamics. What does differentiate is that the clinical response depends a lot on the underlying cardiovascular phenotype, the time of onset, the severity of pulmonary hypoplasia, the presence of left ventricular dysfunction and the use of other therapies [10,11]. The pathophysiology recognizes that many newborns have not only pulmonary hypertension but also ventricular dysfunction, especially left or biventricular dysfunction, so inotropics are selected according to the hemodynamic phenotype, in this context dobutamine is used when the decrease in myocardial contractibility predominates9. Reviews describe dobutamine as a first-line adoption when there is a decrease in contractibility due to its beta-adrenergic inotropic effect, improving cardiac output and ventricular function, however, it can cause tachycardia if there is no alteration in cardiac function and this high frequency causes a drop in blood pressure, so it is not the ideal drug if the dominant problem is hypotension due to low systemic vascular resistance [13]. The fact that inotropics are needed in HDC is

associated with greater clinical severity, greater pulmonary hypertension, and greater ventricular dysfunction. A recent study found that the presence of PH and left ventricular death, clinically expressed by the need for higher doses of inotropic support, influenced mortality and length of hospital stay (Table 1).

***Table 1**: Clinical characteristics, hemodynamic management, ventilatory and outcome.*

NAME OF THE RN	MATERNAL AGE	EG	APGAR	SEX	WEIGHT	MILRINONE PREQX	MILRINONE POSQX	NOREPINIFRINE	DOBUTAMINE	VAFO	VMC	TYPE OF HERNIA	HOSPITALIZATION DAYS	EGRESS ALIVE/ DEAD
Patient 1	27	38.5	Q8 - Q9	F	2955	NO	NO	1 DAY	6 DAYS	0 DAYS	8 DAYS	LEFTT YPE B	19	ALIVE
Patient 2	21	36	8-9	M	2600	NO	NO	5 DAYS	NO	0 DAYS	5 DAYS	LEFT TYPE B	18	ALIVE
Patient 3	31	41	5-9	F	2526	NO	NO	1 DAY	6 DAYS	0 DAYS	6 DAYS	RIGHT	19	ALIVE
Patient 4	28	39.6	8-9	F	2370	8 DAYS	2 DAYS	9 DAYS	12 DAYS	6 DAYS	4 DAYS	LEFT TYPE B	36	ALIVE
Patient 5	26	32.5	8-9T	M	1840	2 DAYS	NO	2 DAYS	5 DAYS	1 DAY	0 DAYS	LEFT TYPE D	1	DEAD
Patient 6	31	37.6	Q8 - Q9	M	2570	NO	NO	20 DAYS	20 DAYS	9 DAYS	6 DAYS	LEFT TYPE C	49	ALIVE
Patient 7	34	40.4	3-6T-9T	M	3930	7 DAYS	NO	5 DAYS	7 DAYS	7 DAYS	12 DAYS	LEFT TYPE B	32	ALIVE
Patient 8	22	37	7T-9T	F	3200	9 DAYS	5 DAYS	12 DAYS	NO	10 DAYS	10 DAYS	LEFT TYPE B	30	ALIVE
Patient 9	44	38.5	7T-8T	F	3640	1 DAY	1 DAY	2 DAYS	1 DAY	1 DAY	0 DAYS	LEFT WITH LIVER IN THE CHEST	1	DEAD
Patient 10	43	37.1	9T-9T	M	2643	7 DAYS	1 DAY	1 DAY	NO	3 DAYS	19 DAYS	LEFT TYPE B	23	ALIVE

Patients who required combined support with milrinone, norepinephrine, and HFOV represented the group with the highest cardiorespiratory involvement, concentrating all mortality events and the highest requirements for ventilatory support. In contrast, patients managed with conventional ventilation without pulmonary vasodilator support had a more favorable outcome. These findings include that the intensity of the support required constitutes an indirect marker of pathophysiological severity and could reflect different hemodynamic phenotypes within the clinical spectrum of CDH. All patients were managed under a protective pulmonary ventilation protocol, conventional ventilation was the initial modality of respiratory support; High-frequency oscillatory ventilation was used as rescue therapy in patients with persistent hypercapnia, respiratory acidosis, or elevated inspiratory pressure requirements. The central point of the evidence is that in HDC the priority is not to use HFOV by itself but to maintain a lung protection strategy that avoids volutrauma and barotrauma in hypoplastic lungs and conventional ventilation such as HFOV can be part of that approach, the results show that patients who require HFOV are usually the most severe showing worse oxygenation index, more days of respiratory support. Neonates who required HFOV show worse oxygenation

rates in the first 72 hours, longer time to surgery, and higher mortality [3]. There is no evidence to say that HFOV should be the standard initial mode in all newborns with CDH, however, in severe CDH, early initiation of HFOV over conventional ventilation [12]. In severe HDC, early initiation of HFOV is associated with improvement in gas exchange, so HFOV in selected groups, especially when the aim is to maintain alveolar recruitment with protective ventilation and avoid high pressures.

The overall mortality of the cohort was 20%. Both deaths occurred in newborns with high-risk characteristics. The first case corresponded to a type D diaphragmatic hernia, characterized by an extensive diaphragmatic defect with almost complete absence of the hemidiaphragm, a condition associated with severe pulmonary hypoplasia. The second case presented intrathoracic hepatic herniation, one of the most consistent predictors of mortality reported in the literature. None of the patients with type B defects died during the study period. No deaths were reported in patients managed with conventional support or in those who required advanced support without milrinone. The need for combined therapeutic strategies is an indirect marker of greater pathophysiological severity, reflecting a greater severity of pulmonary hypoplasia, pulmonary hypertension and hemodynamic compromise since one of them had type D CDH, corresponding to a complete absence of the diaphragm and the second patient had an intrathoracic hepatic herniation, a finding widely described in the literature as one of the main prognostic factors adverse due to its close relationship with a higher degree of fetal lung compression, severe pulmonary hypertension. The main limitations include its retrospective nature, the small sample size and the absence of ECMO in our center, which limits the generalizability of the results. However, the cohort reflects real clinical practice in a highly complex neonatal referral center in Latin America. In conclusion, CDH continues to represent one of the most complex neonatal pathologies in which the prognosis is determined not only by the severity of pulmonary hypoplasia, but also by the degree of associated hemodynamic compromise. The stratification of patients according to the intensity of cardiorespiratory support allowed the identification of different hemodynamic phenotypes associated with different clinical severity and outcome, which could be a useful tool for the prognostic evaluation of centers without ECMO. This analysis highlights the importance of continuing to generate local evidence that allows optimizing clinical decision-making and adapting international recommendations to the reality of care, with the aim of improving outcomes in this highly vulnerable population. Despite being a highly complex cohort, overall survival reached 80%, a result comparable to the series reported by international centers specialized in the management of congenital diaphragmatic hernia.

The findings, opinions, and points of view contained in the article are particular to the author and not as a result of an official position of the Institution. The author has no conflict of interest.

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