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ORIGINAL ARTICLE

The Prognosis of Premature Newborn Who Suffer From Respiratory Distress Syndrome with and Without Patent Ductus Arteriosus

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ABSTRACT:

Background: The prognosis of premature newborns with respiratory distress syndrome (RDS) depends on factors such as gestational age, birth weight, infection, requirement for respiratory support and diagnosis of a patent ductus arteriosus (PDA). The aim of our study was to assess the morbidity and mortality of premature newborn who suffer from RDS and PDA and study the clinical, microbiological & echocardiographic aspects of those patients and demonstrate the differences in clinical profile, microbiology, echocardiography and clinical outcome between premature newborn who suffer from RDS and PDA and those who have RDS without PDA.

Methods: This retrospective observational study was carried out on 25 premature neonates with RDS requiring intubation and surfactant therapy, RDS and PDA identified on echocardiography. Patients were assigned into two groups: Group I: admitted with RDS with PDA and Group II: admitted with RDS without PDA during the same period.

Results: 1-min and 5-min Apgar score, hemoglobin, white blood cells, pH, HCO3, O2 saturation, and PaO2 were significantly lower in Group 1 than Group 2 as (P value <0.05). while Na and PaCO2 were significantly higher in Group 1 than Group 2. Mortality and morbidity were insignificantly different between the two groups.

Conclusion: RDS and PDA preterm infants show a lower Apgar score, hemoglobin level, and O2 saturation in comparison to RDS without PDA preterm infants. Mortality and morbidity are insignificantly different between RDS and PDA preterm infants and RDS without PDA preterm infants' group.

Keywords: Premature; Distress; Ductus; Arteriosus; Newborn.

INTRODUCTION

Preterm delivery (PTD), characterized by the birth of a live infant prior to the completion of thirty-seven weeks of gestation, remains the primary contributor to perinatal illness and death in developed nations **[1].** PTD is linked to a range of long-term negative health outcomes, including impairments in vision and hearing, as well as neurodevelopmental and behavioral challenges **[2].** The underlying causes of PTD are not yet fully understood. It is believed to result from a combination of obstetric, genetic, environmental, and demographic factors, which may function independently or in conjunction to trigger preterm birth **[3].** A combination of several factors significantly heightens the risk of delivering preterm **[4].** Respiratory distress syndrome (RDS) is recognized as a threatening cause of increased morbidity and mortality in neonates. It typically affects infants whose lungs are not fully mature, often due to

prematurity, though it may also result from genetic abnormalities affecting lung development. The infant's birth weight and gestational age, the presence of infection, presence of patent ductus arteriosus (PDA), and the need for mechanical breathing are some of the variables that affect the outcome and severity of RDS **[5].**

PDA refers to the continued patency of a fetal blood vessel connecting the two main arteries emerging from the heart. This can result in a left-to-right shunt, where blood flows from the aorta to the pulmonary artery, potentially causing various complications **[5].** While the ductus arteriosus is a normal anatomical feature during fetal development, it is expected to close soon after birth. When it fails to do so, the condition is termed PDA. PDA does not cause RDS but may worsen its severity by increasing pulmonary circulation and causing pulmonary edema **[6].** The aim of our work was to analyze the morbidity and mortality of premature newborn who suffer from RDS and PDA.

METHODS

This retrospective observational study was carried out on 25 premature neonates aged less than 36 weeks, both sexes, birth weight 600-1400 grams with RDS requiring intubation, surfactant therapy, RDS and PDA identified on echocardiography. The study was done from January 2024 to December 2024 after approval from the Ethical Committee of Zagazig University, Zagazig, Egypt (ZU-IRB#10813-6-6-2023). An informed written consent was obtained from relatives of the patients. Exclusion criteria were associated congenital anomalies as: nasopharyngeal pathology as: (choanal atresia, cleft lip and palate, etc.), major lethal congenital anomalies especially thoracic (pulmonary hypoplasia) and cardiac defects other than PDA. Patients were divided into two groups: Group I: admitted with RDS with PDA and Group II: admitted with RDS without PDA during the same period. The diagnosis of PDA was established by echocardiography, defined as persistent left-to-right shunt at the ductal level with ductal diameter >1.5 mm, left atrium-to-aortic root (LA/Ao) ratio >1.4, and evidence of pulmonary overcirculation or systemic hypoperfusion. The clinical features of RDS have been clearly defined by Silverman **[7],** Downes **[8],** and their co-workers. The diagnostic criteria for RDS in this study were:

- Onset of respiratory distress within 6 hours following birth and persisting for more than 4 hours.
- Prematurity, defined as a gestational age of less than 37 weeks.
- Presence of historical risk factors such as cesarean section delivery or a diabetic mother.
- Abnormal chest roentgenograms, consisting of a diffuse reticulogranular pattern associated with poor aeration of the lung parenchyma.
- Abnormal arterial blood gases, with the PACQ greater than 60 torr and Pao, below 40 torr when breathing room air.
- Need for nasotracheal intubation and mechanical respiratory assistance **[7, 8]**.

All patients were subjected to complete history taking, clinical examination, laboratory investigations [Complete blood count (CBC) with differential count and c-reactive protein (CRP) with titter, arterial blood gases (determination of pH, PCO_2, BE, HCO3, PO2, and O2 saturation), Na, K, Ca and phosphorus] and Imaging (Echocardiography, ECG and chest X-ray (CXR)). Although this study was observational, PDA management was guided by clinical status and echocardiographic findings. Infants with hemodynamically significant PDA (hsPDA) were referred for medical treatment with ibuprofen or paracetamol. In selected cases, surgical ligation was considered after failure of medical closure, following standard NICU protocols. Prior to starting medical treatment for PDAs, echocardiograms were taken using a Philips probe set to a frequency of 5 MHz in order to evaluate haemodynamics and ductal patency in the two groups. PDA size, LA/Ao ratio, systemic arterial blood pressure,

and right ventricular systolic pressure (RVP), which was obtained by doppler probe of the tricuspid regurgitant jet or PDA gradient, were all evaluated by echocardiography. To evaluate the direct impact of these surfactants on PDA haemodynamics, echocardiograms were also taken in a subset of infants just prior to and 20 to 30 minutes following the administration of the second dosage of surfactant.

Statistical Methods

SPSS version 26 (IBM Corp., Chicago, IL, USA) was used to analyze the data. The unpaired Student's t-test was used to compare the two groups, and quantitative results were presented as mean ± standard deviation (SD). Depending on its relevance, the Chi-square test or Fisher's exact test were used for statistical comparisons of categorical data, which were displayed as frequencies and percentages. It was determined that statistical significance was indicated by a two-tailed p-value of less than 0.05.

RESULTS

As indicated in Table (1), there was no significant difference between the two groups in terms of birth weight, gestational age, mode of delivery, sex, maternal chorioamnionitis, use of prenatal steroids, use of surfactants, and use of drugs. One minute and five minutes group 1's Apgar score was considerably lower (P values = 0.003 and 0.016, respectively). According to Table (2), there was no significance in the length of time spent in the NICU between both groups. White blood cells (WBCs) and haemoglobin were significantly lower in Group 1 than Group 2 (P value <0.05), but Na was significantly higher in Group 1 than Group 2 (P<0.002), according to Table (3). The two groups' differences in K, Ca, and phosphorus were negligible. In addition, Group 1 exhibited significantly lower levels of pH, bicarbonate (HCO_3), oxygen saturation (O_2 sat), and arterial oxygen pressure (PaO_2) in comparison to Group 2 (P < 0.05). Conversely, arterial carbon dioxide pressure ($PaCO_2$) was significantly elevated in Group 1 relative to Group 2 (P < 0.001). According to Table (5), there was no significant difference in mortality or morbidity between the two groups.

Table 1: Neonatal Demographic data and general history of the studied groups

		Group 1 (n=25)	Group 2 (n=25)	P value
Gestational age (weeks)		31.48 ± 2.24	31.28 ± 1.51	0.713
Birth weight (g)		989.24 ± 206.77	942.8 ± 226.14	0.452
Sex	**Male**	16 (64%)	12 (48%)	0.254
	Female	9 (36%)	13 (52%)	
Mode of delivery	**Vaginal delivery**	9 (36%)	7 (28%)	0.544
	CS	16 (64%)	18 (72%)	
Maternal chorioamnionitis		4 (16%)	5 (20%)	1
Use of antenatal steroids		20 (80%)	18 (72%)	0.741
Surfactant use		14 (56%)	11 (44%)	0.396
Medications use		14 (56%)	10 (40%)	0.258

Data are presented as Mean ± SD, Frequency (%), CS: cesarean section.

Table 2: Apgar score and duration of NICU staying of the studied groups

	Group 1 (n=25)	Group 2(n=25)	P value
1-min Apgar score	6.04 ± 1.9	7.56 ± 1.45	**0.003***
5-min Apgar score	7.36 ± 1.08	8.2 ± 1.29	**0.016***
Duration of NICU staying (days)	42 (30 - 55)	34 (22 - 45)	0.099

Data are presented as mean ± SD or median (IQR), *Significantly different as P value ≤0.05, NICU: Neonatal Intensive Care Unit.

Table 3: Lab investigations of the studied groups

	Group 1 (n=25)	Group 2 (n=25)	P value
Hemoglobin (g/dl)	11.27 ± 0.49	12.7 ± 0.96	**<0.001***
WBCs (x109/L)	5.7 ± 1.1	6.6 ± 1.02	**0.004***
Na (mEq/L)	121.52 ± 7.04	115.36 ± 6.08	**0.002***
K (mEq/L)	4.88 ± 0.96	4.36 ± 0.92	0.056
Ca (mmol/L)	0.98 ± 0.3	1.11 ± 0.2	0.078
Phosphorus (%)	2.01 ± 0.41	1.82 ± 0.35	0.089

Data are presented as Mean ± SD, *Significantly different as P value ≤0.05, WBCs: White blood cells.

Table 4: Arterial blood gases tests of the studied groups

	Group 1 (n=25)	Group 2 (n=25)	P value
pH	7.09 ± 0.02	7.15 ± 0.05	**<0.001***
$PaCO_2$ (mmHg)	68.96 ± 3.36	63.36 ± 2.53	**<0.001***
HCO_3 (mEq/L)	17.96 ± 1.31	20.96 ± 1.84	**<0.001***
PaO_2 (mmHg)	60.4 ± 7.54	66.92 ± 7.98	**0.005***
O_2 saturation (%)	88.24 ± 1.69	90.84 ± 1.31	**<0.001***

Data are presented as Mean ± SD, *Significantly different as P value ≤0.05.

Table 5: Mortality and morbidity of the studied groups

	Group 1(n=25)	Group 2 (n=25)	P value
Mortality	4 (16%)	1 (4%)	0.348
Morbidity	7 (28%)	3 (12%)	0.289

Data are presented as Frequency (%).

DISCUSSION

PDA can worsen Respiratory Distress Syndrome (RDS) by increasing pulmonary blood flow, but it is not a direct cause of RDS. RDS is primarily caused by a deficiency in surfactant, which leads to lung immaturity and difficulty breathing. While a PDA can exacerbate the symptoms of RDS, it's a consequence of the underlying lung condition and prematurity, not a root cause **[9, 10].** Concerning our results, gestational age, birth weight, sex, mode of delivery, maternal chorioamnionitis, use of antenatal steroids, surfactant use, and medications uses are insignificantly different between two groups. These results are in agreement with Hammoud and colleagues who showed that RDS with PDA and RDS without PDA showed no significant difference regarding mode of delivery and use of antenatal steroids **[11].** Moreover, Chen and colleagues are in accordance to what we found as they demonstrated that RDS with PDA and RDS without PDA groups showed no considerable difference regarding gender, antenatal steroids, and delivery method, but Chen study showed dissimilar results to ours as they observed that both groups had statistically difference regarding gestational age, birth weight, and surfactant use **[12].**

Group 1's 1- and 5-minute Apgar scores are significantly lower than Group 2's, according to our study (P values = 0.003 and 0.016, respectively). These findings are supported by Chen and colleagues as they showed that 1-min Apgar score was lower in RDS with PDA group (4.27 ± 2.612) than RDS without PDA group **(**5.17 ± 2.355) (P=0.126), also 5-min Apgar

score was lower in RDS with PDA group **(**6.67 ± 2.233) than RDS without PDA group **(**7.44 ± 1.761) (P=0.05) **[12]**. On the contrary, Ozer and colleagues had showed that 1-min and 5-min Apgar score was insignificantly different between Group 1 and Group 2 as (P value >0.05 for each) **[13].**

Regarding our statics, the duration of NICU staying is insignificantly different between the two groups. In agreement with us, Hammoud and colleagues revealed that RDS with PDA and RDS without PDA showed no difference in NICU stay which was related to insignificant difference in fetal distress between two groups **[11].**

According to our results, pH, HCO_3, O_2 saturation and PaO_2 are significantly lower in Group 1 than Group 2 (P value<0.05). $PaCO_2$ is significantly higher in Group 1 than Group 2 (P value<0.001). In the same line with our results, Medina-Serra, Chock and colleagues demonstrated that PDA in preterm infants showed hypoxia and hypercapnia **[14, 15].** The mechanism of hypoxia and hypercapnia is that the ductus is a remnant of the distal sixth aortic arch and connects the proximal descending aorta to the main pulmonary artery. If the ductus remains patent after birth it is associated with pulmonary edema. The increase in respiratory distress and hypoxia occur with pulmonary edema **[16].**

Our study clears that mortality and morbidity were insignificantly different among the two groups. In accordance with us, Ozer and colleagues stated that RDS with PDA group showed no significant difference regarding survival compared to RDS without PDA group (P>0.05) **[13].**

The study has several limitations, including that it is a single center study that might yield different results from other studies, a small sample size that might yield negligible results, and a failure to examine maternal risk factors for the development of PDA in preterm infants, such as age, diabetes, hypertension, and multipara.

CONCLUSIONS

Compared to RDS without PDA preterm infants, preterm newborns with PDA and RDS have lower haemoglobin levels, O2 saturation, and Apgar scores. The group of RDS with PDA preterm newborns and the group of RDS without PDA preterm infants have no differences in mortality and morbidity**.**

Conflict of interest: The authors declare that they have no competing interest.

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REFERENCES

1. Ananth CV, Vintzileos AM. Epidemiology of preterm birth and its clinical subtypes. J Matern Fetal Neonatal Med. 2006;19:773-82.
2. El-Gilany A-H, Elkhawaga G, Ghanem A. Incidence and occupational risk factors of preterm delivery among working mothers: A single center study in Egypt. TAF Preventive Medicine Bulletin. 2016;15:199.
3. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. Lancet. 2008;9606:75-84.
4. Zhang YP, Liu XH, Gao SH, Wang JM, Gu YS, Zhang JY, et al. Risk factors for preterm birth in five maternal and child health hospitals in beijing. PLoS One. 2012;12:52780.
5. WAMBACH JAH, A. M. Lagoski. Respiratory distress syndrome in neonate. Diseases of the fetus and infant. Philadelphia: Elsevier, Saunders.; 2015.
6. Benitz WE. Treatment of persistent patent ductus arteriosus in preterm infants: time to accept the null hypothesis? J Perinatol. 2010;30:241-52.
7. Silverman WA, Fertig JW, Berger AP. The influence of the thermal environment upon the survival of newly born premature infants. Pediatrics. 1958;22:876-86.

8. Downes JJ, Vidyasagar D, Boggs TR, Jr., Morrow GM, 3rd. Respiratory distress syndrome of newborn infants. I. New clinical scoring system (RDS score) with acid--base and blood-gas correlations. Clin Pediatr (Phila). 1970;9:325-31.
9. Behman RE, Jacob J, Gluck L, DiSessa T, Edwards D, Kulovich M, et al. The contribution of PDA in the neonate with severe RDS. J ped. 1980;96(1):79-87.
10. Balsan MJ, Jones JG, Guthrie RD. Effects of a clinically detectable PDA on pulmonary mechanics measures in VLBW infants with RDS. Ped pulmonol. 1991;11(2):161-5.
11. Hammoud MS, Elsori HA, Hanafi EA, Shalabi AA, Fouda IA, Devarajan LV. Incidence and risk factors associated with the patency of ductus arteriosus in preterm infants with respiratory distress syndrome in Kuwait. Saudi Med J. 2003;24:982-5.
12. Chen YY, Wang HP, Chang JT, Chiou YH, Huang YF, Hsieh KS. Perinatal factors in patent ductus arteriosus in very low-birthweight infants. Pediatr Int. 2014;56:72-6.
13. Özer Bekmez B, Tayman C, Buyuktiryaki M, Çetinkaya A, Cakır U, Derme T. A promising, novel index in the diagnosis and follow-up of patent ductus arteriosus: Red cell distribution width-to-platelet ratio. J Clin Lab Anal. 2018;32:22616.
14. Medina-Serra R, Palacios C, McMillan M. Alternative anaesthetic management in a reintervention for correction of a left-to-right shunting patent ductus arteriosus (PDA) in a dog. Vet Rec Case Rep. 2021;9:25.
15. Chock VY, Bhombal S, Variane GFT, Van Meurs KP, Benitz WE. Ductus arteriosus and the preterm brain. Arch Dis Child Fetal Neonatal Ed. 2023;108:96-101.
16. Jain P, Lazarus M, Tiwari A, Athwani VK. Prevalence and pattern of congenital heart disease in pediatric population—a study from central india. IJRSMS. 2022;9:48.

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