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Retrospective Study of Predictor of CPAP Failure in Neonates

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Abstract

Background: To assess predictor of CPAP failure in neonates. **Methodology:** Two hundred seventy preterm infants less than 37 weeks of gestation with respiratory distress (RD) of both genders were selected. CPAP was delivered using bubble CPAP device. Baseline maternal and infant characteristics, CPAP care practices, morbidities and adjuvant therapies were recorded. The morbidities and outcomes were compared among infants with CPAP failure and CPAP success. **Results:** Male infant were110in group I and 30 in CPAP group II, birth weight < 750 g was seen in 9 in group I and 2 in group II, birth weight was 1280 grams in group I and 1194 grams in group II. Small for gestation was 30 in group I and 6 in group II, cesarean section was seen in 201 in group I and 45 in group II, antenatal steroids was seen in 198 in group I and 43 group II, 5 min APGAR was seen 8 in both groups, FiO2 at starting CPAP was 0.41 group I and 0.41 group II, CPAP (cm of H2O) pressure at initiation of CPAP was 5 in each group. Type of interface was fisher and paykel prongs was 16 group I and 3 in group II, Hudson prongs was 190 group I and 45 group II and Benveniste prongs 14group I and 2 group II, surfactant was used in 110 group I and 40 in group II. A significant difference was seen in both groups (P< 0.05). **Conclusion:** Early starting of CPAP, early surfactant administration, and lower FiO2 at beginning of CPAP were predictors of CPAP failure in neonates of respiratory distress syndrome.

Keywords: CPAP, Neonates, Respiratory distress syndrome, Surfactant

INTRODUCTION

Respiratory distress syndrome (RDS) is a common reason for neonatal intensive care unit (NICU) admission.[1] RDS was originally indicated as idiopathic respiratory distress syndrome (iRDS) or "hyaline membrane disease" (HMD), based on the histological presence of alveolar layers of fibrin and necrotic cells originally described in the Lancet in 1953.[2] It was finally re-named RDS after it was shown to be caused by primary surfactant deficiency. Respiratory distress syndrome remains the single, most important cause of mortality among preterm neonates despite advances in neonatal intensive care.[3]

According to the 2016 European Guidelines on the management of RDS, early nasal CPAP is recommended as the first-choice treatment in infants <30 weeks' gestation, who are at risk of RDS, but do not require mechanical ventilation (MV).[4] Widespread use of nasal continuous positive airway

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pressure (CPAP) as the initial means of respiratory support for preterm infants has fundamentally changed their management in early life.[5] Large clinical trials have indicated that CPAP started soon after birth, without prior intubation for surfactant therapy, is associated with equivalent or superior respiratory outcomes compared to a group that are intubated primarily.[6] At gestation beyond 29 weeks, most preterm infants with signs of respiratory distress syndrome (RDS) now receive an initial trial of CPAP which has become the standard of care.[7] The key clinical benefit of the early initiation of CPAP is the potential avoidance of invasive ventilation with all the related sequelae. However, the efficacy of early CPAP may vary, and the success rate largely depends on the gestational age.[8]

Surfactant deficiency has been suggested to be the main cause of CPAP failure. Maximum fraction of inspired oxygen (FiO2) in the first hours of life, immature gestation, lower birth weight, male gender, need for positive pressure ventilation at the time of birth and moderate to severe RDS etc. are other causes of CPAP failure.[9] Considering this, the present study was attempted with the aim to assess predictor of CPAP failure in neonates.

MATERIALS & METHODS

This retrospective study comprised of two hundredseventy preterm infants less than 37 weeks of gestation with respiratory distress (RD) of both genders. The approval for the study was initially obtained from higher authorities (Review & Ethical committee). Parental written consent was obtained before starting the study. Infants requiring intubation soon after birth, 5 min Apgar score less than 3, life-threatening malformations such as congenital diaphragmatic hernia, tracheo-esophageal fistula and malformations were excluded from the study.

CPAP was delivered using bubble CPAP device. Few infants required the Jet-CPAP device, a standalone variable flow CPAP device with a Benveniste valve. The desired pressure was achieved by adjusting the flow rate. The infant was continued on T-piece CPAP from birth till admission to Neonatal intensive care unit (NICU).

We used surfactant at the dose of 100 mg/kg of phospholipid given by In Sur E (Intubate Surfactant Extubate) technique, if an infant had respiratory distress syndrome and had an FiO2 requirement greater than 0.30. Baseline maternal and infant characteristics, CPAP care practices, morbidities and adjuvant therapies were recorded. The morbidities and outcomes were compared among infants with CPAP failure and CPAP success. Results of the present study after recording all relevant data were subjected for statistical inferences using chi- square test. The level of significance was significant if p value is below 0.05 and highly significant if it is less than 0.01.

RESULTS

Table 1: Distribution of patients

Total- 270		
Gender	**Boys**	**Girls**
Number	140 (51.8%)	130 (48.2%)

Out of 270 neonates, 140 (51.8%) were boys and 130 (48.2%) were girls (Table 1).

Table 2: Comparison of predictor variables

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Variables	CPAP success (Group I) (220)	CPAP failure (Group II) (50)	P value
Male infant	110	30	<0.05
Birth weight < 750 g	9	2	<0.05
Birth weight (grams)	1280	1194	0.17
Small for gestation (SGA)	30	6	<0.05
Cesarean section	201	45	<0.05
Antenatal steroids	198	43	<0.05
5 min APGAR	8	8	1
FiO2 at starting CPAP	0.41	0.41	1
CPAP (cm of H2O) pressure at initiation of CPAP	5	5	1
Type of interface Fisher and Paykel prongs	16	3	<0.05
Hudson prongs	190	45	
Benveniste prongs	14	2	
Surfactant	110	40	<0.05

Male infant were110 in group I and 30 in CPAP group II, birth weight < 750 g was seen in 9 in group I and 2 in group II, birth weight was 1280 grams in group I and 1194 grams in group II. Small for gestation was 30 in group I and 6 in group II, cesarean section was seen in 201 in group I and 45 in group II, antenatal steroids was seen in 198 in group I and 43 group II, 5 min APGAR was seen 8 in both groups, FiO2 at starting CPAP was 0.41 group I and 0.41 group II, CPAP (cm of H2O) pressure at initiation of CPAP was 5 in each group. Type of interface was fisher and paykel prongs was 16 group I and 3 in group II, Hudson prongs was 190 group I and 45 group II and Benveniste prongs14 group I and 2 group II, surfactant was used in 110 group I and 40 in group II. A significant difference was seen in both groups (P< 0.05) (Table 2, graph 1).

Graph 1 Comparison of predictor variables

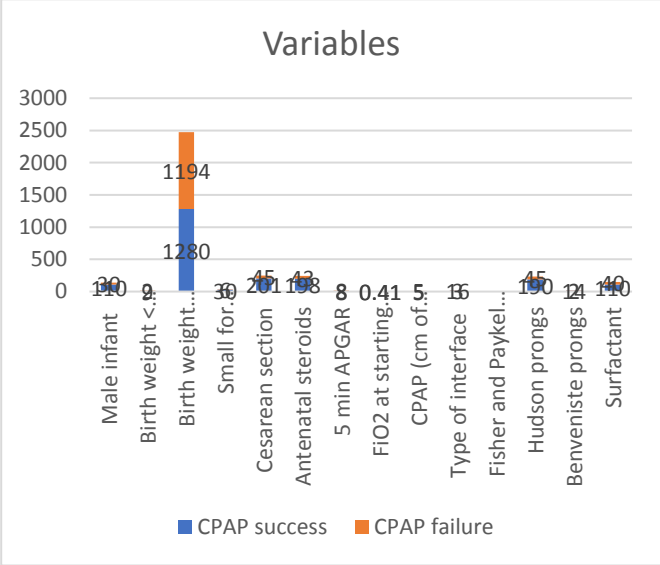

Table 3: Predictors of early CPAP failure

Predictor variable	Odd ratio 95% CI
Gestation (weeks)	0.91 (0.80-1.06)
Age of starting CPAP (hours)	1.007 (1.002- 1.012)
Starting CPAP Pressure (cm of H2O)	1.25 (1.20-1.32)
Surfactant	2.8 (2.2-3.4)
Age at surfactant administration (hours)	1.13 (1.12- 1.14)
Starting FiO2	1.04 (1.02- 1.05)

Gestation (weeks) {0.91 (0.80-1.06)}, age of starting CPAP (hours) {1.007 (1.002- 1.012)}, starting CPAP Pressure (cm of H2O) {1.25

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(1.20-1.32)}, surfactant {2.8 (2.2-3.4)}, age at surfactant administration (hours) {1.13 (1.12- 1.14)} and starting FiO2 {1.04 (1.02-1.05)} were predictor for CPAP failure (Table 3).

DISCUSSION

The present study was attempted with the aim to assess predictor of CPAP failure in neonates. enrolled 270 neonates, out of which 140 (51.8%) were boys and 130 (48.2%) were girls The key clinical benefit of the early initiation of CPAP is the potential avoidance of invasive ventilation with all the related sequelae.[10,11] However, the efficacy of early CPAP may vary, and the success rate largely depends on the gestational age.[12] Of note, infants who fail CPAP are at increased risk of death, pneumothorax, and bronchopulmonary dysplasia (BPD), among other morbidities.[13]Available data regarding the specific level of FiO2, which is predictive of CPAP failure, are still limited. Many experts pose the following questions: "which new borns have the highest risk of CPAP failure?" and "when should the decision on surfactant administration be made.[14]

Our study showed that Male infant were110 in CPAP success and 30 in CPAP failure group, birth weight < 750 g was seen in 9 in group I and 2 in group II, birth weight was 1280 grams in group I and 1194 grams in group II. Small for gestation was 30 in group I and 6 in group II.Murki et al,[15] identified risk factors and outcomes associated with early failure of nasal continuous positive airway pressure (CPAP) in premature infants with respiratory distress (RD). Six hundred and fifty-two infants were enrolled in the study. Early CPAP failure was seen in 96 infants (14.7%, 95% CI: 12%–17.5%). On logistic regression, adjusting for gestation and year of study, time of starting CPAP in hours (OR 1.01, 95% CI: 1.003–1.013), time of surfactant administration in hours (OR 1.12, 95% CI: 1.05–1.19), InSurE (Intubate Surfactant Extubate) (OR 2.7, 95% CI: 1.43–5.06) and higher starting FiO2 (OR 1.03, 95% CI: 1.01–1.05) predicted early CPAP failure. Neonatal morbidities and hospital duration were significantly higher in infants who failed CPAP.

It was seen that cesarean section was seen in 201 mothers in group I and 45 in group II, antenatal steroids was seen in 198 in group I and 43 group II, 5 min APGAR was seen 8 in both groups, FiO2 at starting CPAP was 0.41 group I and 0.41 group II, CPAP (cm of H2O) pressure at initiation of CPAP was 5 in each group. Gulczyńska et al,[16] investigated factors predictive of CPAP failure in the first 72 hours of life, with special attention to the prognostic role of FiO2. The risk was increased by 4.2 and 7.5% for each 0.01 increase in FiO2 in the first and second hours of life, respectively. In the final multivariate model, birth weight and FiO2 in the second hour of life were the predictive measures. The prognostic threshold was FiO2 = 0.29 in the second hour of life (AUC 0.7; $p < 0.0001$), with a sensitivity of 73% and a specificity of 57%. CPAP failure implied a more than 20-fold higher risk of death and pneumothorax and a 2- to 5-fold higher risk of typical complications of prematurity, including bronchopulmonary dysplasia and severe intraventricularhemorrhage.

In this study it was found that type of interface was fisher and paykel prongs was 16 group I and 3 in group II, Hudson prongs was 190 group I and 45 group II and Benveniste prongs 14 group I and 2 group II, surfactant was used in 110 group I and 40 in group II.Pillai et al,[17] prospectively observed 62 pre-term very low birth weight neonates initiated on nasal continuous positive airway pressure (CPAP) for respiratory distress in

the first 24 hours of life to devise a clinical score for predicting its failure. CPAP was administered using short binasal prongs with conventional ventilators. On multivariate analysis, we found three variables—gestation gestation<28 weeks, pre-term premature rupture of membranes and product of CPAP pressure and fraction of inspired oxygen >1.28 at initiation to maintain saturation between 88% and 93% to be independently predictive of failure. A prediction model was devised using weighted scores of these three variables and lack of exposure to antenatal steroids. The clinical scoring system thus developed had 75% sensitivity and 70% specificity for prediction of CPAP failure.

CONCLUSION

Results of this study showed that early starting of CPAP, early surfactant administration, and lower FiO2 at beginning of CPAP were predictors of CPAP failure in neonates of respiratory distress syndrome.

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