A Study on Clinical Profile of Preterm Neonates with Respiratory Distress

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KEYWORDS

Respiratory distress, preterm neonates, bubble continuous positive airway pressure, gestational age, birth weight, oxygen therapy, hospital stay

ABSTRACT

Preterm neonates commonly develop respiratory distress, requiring NICU admission especially among <34 weeks of gestation. The timely intervention by CPAP can significantly reduce the mortality and morbidity. CPAP is non invasive method to support spontaneously breathing babies, which is less expensive, easy to operate, requires less skilled staff, less injury than mechanical ventilator. Hence this study was conducted to know the outcome of bubble continuous positive airway pressure in the form of CPAP success and failure rate. A prospective study was conducted on preterm neonates with respiratory distress admitted to a tertiary care hospital. The study analysed the efficacy of B-CPAP therapy, considering various factors such as gestational age, birth weight, duration of CPAP, and length of hospital stay. Statistical data analysis was performed using Microsoft Excel and Epi Info software. Relevant inferential statistical tests like chi- square and t-test were used for better interpretation of the results. A p-value of less than 0.05 was considered statistically significant. In our study babies diagnosed with respiratory distress syndrome were113(80.7%), birth asphyxia were 12 (8.6%) meconium aspiration were 14(10%) and only one congenital pneumonia case was present. Out of 140 preterm neonates treated with B-CPAP, 85% had a successful outcome, while 15% experienced failure.

Introduction

Respiratory Distress Syndrome (RDS) is the leading cause of respiratory failure and mortality in neonates, as well as a common reason for admission to Neonatal Intensive Care Units (NICUs). Respiratory distress is characterized by at least two of the following symptoms: rapid breathing (tachypnea of over 60 breaths per minute), central cyanosis while breathing room air, expiratory grunting, retractions in the subcostal, intercostal, or jugular areas, and nasal flaring. Although RDS predominantly affects preterm infants, it can also occur in those born at term.[1] About 15% of term infants and 29% of late preterm infants who are admitted to NICUs experience significant respiratory issues, with rates even higher for infants born before 34 weeks' gestation.[2]

Globally, RDS is a common neonatal condition, with prevalence rates ranging from 1.44% to 20.5%. The incidence of RDS is inversely related to gestational age, occurring in nearly all preterm infants born at 22-28 weeks' gestation, about 3% of late preterm infants born at 34-36 weeks, and 0.12% of term infants born after 37 weeks. Additional risk factors include meconium-stained amniotic fluid, cesarean delivery, gestational diabetes, maternal chorioamnionitis, and prenatal ultrasound findings such as oligohydramnios or structural lung abnormalities. Other causes of respiratory distress include transient tachypnea of the newborn, meconium aspiration syndrome, congenital pneumonia, persistent pulmonary hypertension, congenital heart disease, and several neurological and metabolic conditions.[3,4]

The clinical signs of respiratory distress in neonates include apnea, cyanosis, grunting, inspiratory stridor, nasal flaring, poor feeding, and tachypnea (over 60 breaths per minute), with possible retractions in various chest areas. The prognosis of RDS depends on its severity and the underlying cause. Without prompt recognition and management, respiratory distress can progress to respiratory failure and cardiopulmonary arrest.[5,6] Thus, it is crucial for healthcare practitioners to quickly identify and differentiate the signs and symptoms of respiratory distress and implement appropriate management strategies to prevent severe complications or death. Diagnostic evaluation, including blood gas analysis, pulse oximetry, and chest X-ray, is essential when respiratory distress is suspected.

Methodology:
- **Study Place:** Department of Paediatrics – Neonatal Intensive Care Unit
- **Type of Study:** Hospital Based Prospective Study
- **Sampling method:** Purposive sampling
- **Sample Size:** 138.9

Method of Collection of Data
- The study received approval from the institution.
- Written and Informed Consent taken from the parents or guardian of all the neonates.
- All preterm neonates with respiratory distress delivered at Adichunchanagiri hospital and research institute during the study period taken for the study.

Neonates selected using inclusion and exclusion criteria as follows:

Inclusion Criteria
- Preterm babies with respiratory distress with SILERMAN ANDERSON SCORE 4-6.

Exclusion Criteria
1. Neonates with Respiratory distress with SILVERMAN ANDERSON SCORE >7
2. Hemodynamically unstable babies
3. Out born babies
4. Intra-ventricular hemorrhage
5. Congenital malformation

Results

Table 1: Distribution of participants based on gender (n=140)

Gender	Frequency (n=140)	Percent (%)
Male	75	53.6
Female	65	46.4
Total	140	100

Among 140 babies 75 were male babies and 65 were female babies, majority of the babies were male babies.

Table 2: Distribution of participants based on gestation age at birth (n=140)

Gestation age	Frequency (n=140)	Percent (%)
<28 weeks	3	2.1
28 to 32 weeks	103	73.6
33 to 36 weeks	34	24.3
37 and above	0	0
Total	140	100

In our study majority of the babies belongs to 28-32 weeks(73.6%). Only 3 babies were there within 28 weeks.

Table 3: Distribution of participants based on delivered mode (n=140)

Mode of delivery	Frequency (n=140)	Percent (%)
LSCS	27	19.3
Vaginal delivery	113	80.7
Total	140	100

Majority of the babies were delivered by vaginal delivery (80.7%) and by LSCS only 27(19.3%).

Table 4: Distribution of participants based on birth weight (n=140)

Birth weight	Frequency(n=140)	Percent (%)
ELBW (<1000gm)	16	11.4
VLBW (< 1500 gm)	31	22.1
LBW (1500- 2500 gm)	93	66.4
Total	140	100

In our study 93 babies had LBW (66.4%), 31 babies falls in VLBW (22.1%), and 16 babies in ELBW (11.4%).

Table 5: Distribution of participants based on mothers medical condition during ANC period (n=140)

Mothers medical condition	Frequency (n=140)	Percent (%)
Diabetes	4	2.8
APH	19	13.6
PIH	31	22.2
Normal	86	61.4
Total	140	100

Table 6: CPAP and Outcome

CPAP Outcome	Frequency	Percentage
Failure	21	15
Success	119	85
Total	140	100

The table indicates that 85% of the subjects had a successful outcome with CPAP treatment, while 15% experienced failure.

Table 7: Distribution of participants based on diagnosis (n=140)

Diagnosis	Frequency (n=140)	Percent (%)
Respiratory distress syndrome	113	80.7
Birth Asphyxia	12	8.6
Meconium Aspiration syndrome	14	10
Congenital Pneumonia	01	0.7
Total	140	100

In our study babies diagnosed with respiratory distress syndrome were113(80.7%), birth asphyxia were 12 (8.6%) meconium aspiration were 14(10%) and only one congenital pneumonia case was present.

Table 8: Distribution of participants based on duration of CPAP(n=140)

Duration of CPAP	Frequency (n=140)	Percent (%)
< 6 hrs	12	8.6
6 to < 12 hrs	25	17.8
12 to 24 hrs	48	34.3
>24 hrs	55	39.3
Total	140	100

In this study majority of the cases required CPAP duration more than 24 hours (39.3%), whereas only 12 cases required CPAP duration less than 6 hours (8.6%).

Discussion

Neonatal Respiratory Distress Syndrome (RDS) arises from a deficiency or delay in the production and secretion of pulmonary surfactant, a protein-phospholipid complex that reduces surface tension at the air-

liquid interface within the alveoli. Insufficient surfactant leads to increased alveolar surface tension, causing atelectasis and impaired gas exchange. Surfactant is produced by alveolar Type II cells, stored in lamellar bodies (specialized intracellular organelles), and then released into the alveolar lumen through exocytosis. In the alveolar lumen, lamellar bodies disintegrate to form tubular myelin, a lattice-like structure where phospholipids gather to create the air-liquid interface. [7]Type II cells begin to differentiate during the canalicular stage of fetal lung development, and lamellar bodies are present by about 22 weeks' gestation. Surfactant production continues to increase until around 35 weeks' gestation. Factors such as acidosis, cold stress, hypovolemia, and hypoxemia can impair surfactant production even when Type II cells are mature.[8] Postnatal factors can also affect surfactant production. For example, invasive mechanical ventilation can expose the infant to high oxygen levels, excessive ventilatory pressures (leading to barotrauma), and overdistention of the lungs (causing volutrauma). These conditions can stimulate the release of proinflammatory cytokines and chemokines, damaging the alveolar epithelial lining and impairing surfactant synthesis. Moreover, the leakage of fibrin and other proteins from the alveolar surface can inactivate surfactant. [9,10]

Additionally, immature pulmonary epithelial membrane transport proteins contribute to the respiratory issues seen in surfactant deficiency. During fetal development, fluid is actively secreted into the alveolar spaces along with chloride ions through sodium-

potassium-chloride co-transporters and other ion transporters. These transporters are down-regulated towards the end of gestation, which slows the accumulation of fetal lung fluid. At birth, elevated epinephrine levels stimulate sodium absorption via epithelial sodium channels and sodium-potassium-ATPase. The expression of epithelial sodium channels peaks late in gestation. Consequently, premature birth or delivery without labor can result in excess fetal lung fluid at birth. Failure to clear this fluid postnatally can lead to pulmonary edema, worsening respiratory distress.[11,12]

Conclusion

In our study babies diagnosed with respiratory distress syndrome were113(80.7%), birth asphyxia were 12 (8.6%) meconium aspiration were 14(10%) and only one congenital pneumonia case was present.

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