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Pre-Term Infants with Neonatal Respiratory Distress Syndrome: A Physiotherapeutic Case Study

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ABSTRACT

Background: A newborn's respiratory distress is frequently caused by neonatal respiratory distress syndrome, or RDS, which usually manifests within hours after birth, usually right after delivery. Term infants are rarely affected by RDS, which mostly affects preterm neonates. Smaller and preterm newborns have a higher incidence of RDS, which is inversely correlated with the infant's gestational age. Although prenatal corticosteroids, surfactants, and sophisticated neonatal respiratory care are among the therapeutic approaches that have improved the outcomes for individuals with RDS, the condition remains a major cause of morbidity and mortality among preterm infants.

Method: Chest physiotherapy manoeuvres are applied at regular intervals every day until the child is discharged.

Conclusion: Early physiotherapeutic interventions have proved to be extremely effective in saving the life of such affected individuals.

Keywords: RDS, Neonatal, Physiotherapy, Rehabilitation

INTRODUCTION

Neonatal respiratory distress syndrome, or RDS, is a common cause of respiratory distress in a newborn, presenting within hours after birth, most often immediately after delivery. RDS primarily affects preterm neonates, and infrequently, term infants. The incidence of RDS is inversely proportional to the gestational age of the infant, with more severe disease in the smaller and more premature neonates. While treatment modalities, including antenatal corticosteroids, surfactants, and advanced respiratory care of the neonate, have improved the outcomes for patients affected by RDS, it continues to be a leading cause of morbidity and mortality in the preterm infant[1]. Neonatal respiratory distress syndrome is caused by surfactant deficiency, especially in the context of immature lungs. The deficiency of surfactant increases the surface tension within the small airways and alveoli, thereby reducing the compliance of the immature lung. The delicate balance of pressures at the air-fluid interface is essential to prevent the collapse of the alveolus or the filling of the alveolus with fluid. The pathophysiology of RDS can be described using LaPlace law, denoted as: P=2T/R where P is pressure, T is surface tension, and R is the radius. Laplace law describes the relationship between the pressure difference across the interface of two static fluids to the shape of the surface. As the surface tension increases at the alveolar level, the amount of pressure required to maintain

alveolar shape increases. With reduced surfactant production, atelectasis occurs throughout the lung, causing reduced gas exchange. Widespread and repeated atelectasis eventually damages the respiratory epithelium, causing a cytokine-mediated inflammatory response. As pulmonary edema develops as a result of the inflammatory response, increasing amounts of protein-rich fluid from the vascular space to leak into the alveoli, which further inactivate surfactant[2,3]. A newborn with neonatal respiratory distress syndrome is frequently born prematurely and exhibits respiratory distress symptoms either minutes after birth or just after delivery. The baby might have fewer breath sounds and perhaps fewer peripheral pulses when they first arrive. Clinically, these neonates exhibit tachypnoea, expiratory grunting, nasal flaring, retractions (subcostal, subxiphoid, intercostal, and suprasternal), use of accessory muscles, cyanosis, and poor peripheral perfusion, all of which are indicators of increased work of breathing. Air entry is consistently reduced, according to auscultation. If left untreated, RDS symptoms will develop over the course of 48 to 72 hours, eventually leading to respiratory failure. The newborn may also become apnoeic and sluggish. In addition, the baby may have symptoms of reduced urine production and peripheral extremities edema. A comprehensive evaluation of the prenatal and delivery history is necessary for the timely diagnosis and treatment of neonatal respiratory distress syndrome due to its imprecise definition. This evaluation identifies perinatal risk factors, clinical presentation, radiographic findings, and blood gas analysis evidence of hypoxemia. The prognosis of infants managed with antenatal steroids, respiratory support, and exogenous surfactant therapy is excellent. Mortality is less than 10%, with some studies showing survival rates of up to 98% with advanced care. Increased survival in developed countries is in stark comparison to babies who received no intervention in low-income countries, where the mortality rate for premature infants with RDS is significantly higher, at times close to 100%.[4] Neurodevelopmental delay is another complication of RDS, especially with infants who received mechanical ventilation long-term.[5]

Numerous respiratory conditions that affect newborns share similarities in their clinical manifestations with neonatal respiratory distress. Nonetheless, imaging, a laboratory workup, and a rudimentary history can all aid in the diagnosis confirmation. Many teams, including doctors, nurses, respiratory therapists, dietitians, and pharmacists, must coordinate care in order to manage RDS. The clinical course of newborns with RDS is further complicated by a number of comorbidities, necessitating a high degree of professional skill. These include sepsis, pulmonary hypertension, patent ductus arteriosus, and respiratory issues such as pneumothorax, pneumomediastinum, and pulmonary interstitial emphysema.

From the moment the baby is stabilized in the delivery room until the long-term objectives of care are specified, clear goals of care must be established. After being discharged from the NICU, the patient is generally managed by a team headed by the neonatologist, occasionally seeking advice from a pulmonologist to arrange long-term care. Optimizing care for such delicate neonates requires specialized neonatal nursing care. The medical team uses a variety of ventilatory methods, and a skilled respiratory therapist is equally important in managing these strategies. These tactics could involve high-frequency ventilation, traditional mechanical ventilation, and several non-invasive ventilation techniques. If RDS is left untreated, the baby may become lethargic and apneic as the symptoms get worse over the course of 48 to 72 hours and eventually lead to respiratory failure.[6]

Tailored interventions addressing risk factors should be devised to improve the preterm neonate's survival in close attention to early stabilization and improve preterm care may further reduce the mortality associated with this disorder, especially to those with low birth weight and lower gestational age.[7]

CASE REPORT

A five-day-old male baby whose weight at the time of birth was 2.3 kg and was delivered via LSCS and age of gestation was 36 weeks was brought to the emergency of CSSH, Meerut with the complaint of difficulty in breathing as reported by the infant's father. The patient had presented with respiratory distress with grunting, nasal flaring, and intercostal retraction. The patient also had an acute history of fever (axillary temperature being 39.2°C). The patient also had a history of poor feeding (refusing breastfeeding as well as bottle feeds with minimal intake) and had lethargy (difficulty arousing with decreased spontaneous movement).

Prenatal history included maternal UTI which was treated with antibiotics during the third semester. Perinatal history included an emergency c-section performed due to foetal distress, APGAR score being 5 and 7 at 1 and 5 minutes respectively. The patient was also admitted to the NICU at Hapur Hospital immediately after birth due to respiratory distress requiring intubation and mechanical ventilation for 48 hours post which the patient was transferred to CSSH, Meerut for further management. The patient had no history of surgery or physiotherapy.

On observation, the patient was radiant warmer and his general complexion was pale. He was on a mechanical ventilator (CPAP- FiO_2 = 40%), PEEP=5. He also had peripheral cyanosis and multiple external appliances were present (which included a cardiopulmonary monitor, pulse oximeter, chest leads, sensor, NG tube, OG tube & IV cannula on the left hand).

Upon palpation no tenderness, oedema, or tracheal shift was present.

Examination revealed GCS of the baby was $E_4V_5M_6$, both S_1 and S_2 were present with no murmurs, abdomen was soft and non-tender. However, bilateral air entry was reduced (right more than left) crepitus was present on the right upper lobe and rhonchi were present on the left lung basal lobe. The patient also presented with head bobbing, subcostal retraction, intercostal retraction, and suprasternal retraction. Vitals on Day 1 of assessment was (BP- 70/30 mm of Hg; RR- 58/min; HR-124bpm; SPO_2- 95%). I:E was 1:2 and reflexes present were rooting, sucking, palmar grasp, plantar grasp, ATNR, Dancing, Spinal Galant, and Moro's.

Chest X-rays of the infant revealed bilateral diffused opacities which were suggestive of pneumonia. WBC count was 22,000 mm^3 with left shift. CRP level was 45mg/L and blood culture was positive for Klebsiella. The patient was managed with IV infusion of Meropenem, Vancomycin, Colistin, and Metrogyl. Along with this patient also underwent oxygen therapy, IV infusions for hydration, and NG tube feeds.

Short Term Goals: Weaning off from the ventilator, removal of bronchial secretions, improvement of ventilation, and maintaining blood oxygen saturation at room air.

Long Term Goals: improvement of mucociliary clearance, optimization of gaseous exchange, increasing efficiency of respiratory muscles.

PHYSIOTHERAPEUTIC INTERVENTIONS

S NO.	INTERVENTION	DOSIMETRY
1.	Nebulization	3 times a day

2.	Postural Drainage
	• Upper Lobe (Left Lung)
	  ○ Apical Segment
	  ○ Posterior Segment
	  ○ Anterior Segment
		5 minutes for each manoeuvre

	• Lower Lobe (Right Lung)
	  ○ Posterior Basal Segment
	  ○ Anterior Basal Segment
	  ○ Superior Segment	

3.	Chest PNF
	
	• Manual Pressure
	  ○ Apical Segment
	  ○ Basal Segment
	
	• Intercostal Stretch
		
		Ten repetitions in three sets with one minute rest time.[8]
		
		Three repetitions in two sets with one minute rest time[8]

4.	Percussion (using Percussion Cup)	Hourly
5.	Manual Vibration (with two fingers)	Two hourly
6.	Hand Bagging	Two hourly
7.	Endotracheal Suctioning	Whenever Required


Percussion


Postural Drainage


Chest PNF

CONCLUSION

To increase the preterm neonate's chances of survival, customized therapies that target risk factors should be developed. The mortality rate linked to this condition may be further decreased by paying close attention to early stabilization and enhancing preterm care, particularly for individuals with low birth weight and lower gestational age.

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