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RESEARCH ARTICLE

CHALLENGES OF NEONATAL RESPIRATORY DISTRESS SYNDROME

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Abstract

One of the most prevalent issues that newborns face in their first few days of life is respiratory distress. Numerous factors, not just respiratory or cardiovascular ones, might induce respiratory distress in a newborn. The newborn experiences respiratory failure if it is unable to maintain the additional effort required to meet its respiratory needs. Infants with the condition exhibit noticeable tachypnea. Impaired breathing (respiratory acidosis) or oxygenation (cyanosis) are two possible symptoms of this malfunction. Initiating respiratory assistance when necessary and assessing the newborn for the degree and cause of respiratory distress are both components of proper therapy.

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Introduction:-

Preterm infants frequently experience respiratory failure due to Neonatal Respiratory Distress Syndrome (NRDS), commonly referred to as Hyaline Disease.^[1] The new-born's breathing rate is between 30 and 60 breaths per minute.^[2] A new-born's respiratory distress is identified by one or more indicators of increased respiratory effort, such as tachypnea, grunting, nasal aring, or chest retractions.^[3] This can progress to respiratory failure and cardiopulmonary arrest. This syndrome occurs about 7% of deliveries in world wide.^[1] Respiratory distress syndrome affects over 1% of new-born's and causes about 860 fatalities annually, mostly in premature infants.^[1] When admitted to the neonatal intensive care unit, 29% of late preterm infants and 15% of term infants experience substantial respiratory morbidity.^[4] Prior to delivery, it is not always possible to determine which newborn's will exhibit symptoms. Prenatal treatment is required for prevention, as it reduces the number of cases of respiratory distress associated with premature births and caesarean deliveries. Inadequate prenatal care can result in infants born with lower birth weights and a higher chance of being admitted to the neonatal intensive care unit. Antenatal corticosteroid used between 24 and 34 weeks of pregnancy, dramatically lowers the frequency and intensity of respiratory distress in premature births. These neonates receive better treatment when they are identified early.^[5]

Etiopathogenesis:-

Numerous and multisystemic factors might contribute to a newborn's respiratory distress syndrome .Neonatal Respiratory Distress Syndrome is often attributed to a lack of surfactant, which is the primary reason. A complicated blend of 90% lipids and 10% proteins, surfactant is primarily produced by the type II alveolar epithelial cells, known as pneumocytes, in the lungs.^[6] To ensure efficient gas exchange and avoid alveolar collapse during exhalation, its primary role is to reduce surface tension within the alveoli. Surfactant is essential for efficient gas exchange and maintaining alveolar stability. Production begins between the 24th and 28th weeks of pregnancy. Typically, sufficient levels are achieved by weeks 35–37. Especially in the later stages of pregnancy, thyroid hormones and the

stress hormone cortisol encourage the production of surfactants. Certain conditions like maternal diabetes can delay the production of surfactants by affecting fetal lung development. Perinatal hypoxia, or lack of oxygen, can damage alveolar cells, which reduces surfactant production. Surfactant may become inactive because of factors such as inflammation, meconium aspiration, or infection, which reduces its effectiveness. Shortly after birth or when symptoms of RDS emerge, an exogenous surfactant—either natural or synthetic—is administered via the endotracheal tube. This improves survival rates, oxygenation, and lung compliance.

Signs And Symptoms:-

The term “respiratory distress” refers to a collection of symptoms that reflect a diverse disease.

Tachypnea (rapid breathing). Respiratory rate exceeds 60 breaths per minute. This occurs as the infant attempts to adjust for low oxygen levels and hindered gas exchange.^[7,8]

A grunting. A quick, quiet sound produced when exhaling. Prevents alveolar collapse and maintains positive end-expiratory pressure (PEEP) naturally.

Flaring of the nasal passages. When breathing, the nostrils widen which indicates a greater effort being made to get air into the lungs.^[8,9]

Retractions of the chest wall . During inspiration, the soft chest wall is drawn inward, causing intercostal, subcostal, and suprasternal retractions. Indicates a greater effort required to breathe.^[9]

Cyanosis. Low oxygen levels in the blood (hypoxaemia) are indicated by bluish discolouration of the lips, tongue, and extremities.^[1,10,11]

Reduced sounds of breathing. Poor alveolar expansion may result in reduced breath sounds on auscultation.

Heart palpitations. Higher heart rate (>160 beats per minute) as a coping strategy to enhance oxygen delivery.

Inadequate nourishment neonates may have trouble sucking and eating due to exhaustion and respiratory issues.

Apnoea . Breathing stoppage for a brief period of time, particularly in infants who are critically ill or very premature.^[12]

Sluggishness or reduced activity . Reduced spontaneous movements and poor response as a result of insufficient oxygenation

Failure of the respiratory system. If left untreated, progressive worsening results in bradycardia, bradypnea (slowed breathing), and ultimately respiratory collapse.

By the third day, RDS symptoms typically reach their peak and may go away as soon as the infant starts to diurese, or expel extra water in their urine. A baby’s requirement for oxygen and artificial assistance to breathe decreases as they become better.

Clinical Diagnosis:-

Newborn’s experiencing respiratory distress must be evaluated with a infant’s history, clinical symptoms, diagnostic tests and physical examination.

Prematurity : There is a high risk for babies born before 34–36 weeks of pregnancy.

Maternal risk elements: Diabetes mellitus causes lung maturity to be delayed.^[1,9,13]

Labor-free caesarean birth (reduces stress-induced surfactant release).^[1,9,13]

Birth difficulties or hypoxia throughout pregnancy.

Check to see if the new-born is breathing normally or if there are any indications of respiratory distress.

Observe how the chest excursions are symmetrical and in tune with the movement of the abdominal wall.^[14]

Neonates are rarely palpated or percussed, but by feeling the tracheal position, identifying the apex beat, palpating for crepitus or murmurs, and percussion for proper resonance, one might obtain important information.

Auscultation: Determine if the inspiratory or expiratory phases are prolonged, if the breath sounds are symmetrically and equally heard throughout the body, and if other sounds such as wheeze, stridor, rales, and rhonchi are present.

Investigations:-

An infection is suggested by complete blood counts if the ratio of immature to total neutrophils is greater than 0.2. Stress, sobbing, and oxytocin-induced labour can all change this ratio.

A thick and uneven pleural line, sub-pleural tiny consolidations, and a widespread alveolar-interstitial pattern with no spared zones are all visible on lung ultrasonography.^[15]

Detecting hypoxia and the requirement for oxygen supplementation is done by pulse oximetry.^[16]

The AP and Lateral CXR will show elevated diaphragm, air bronchograms, and reticulogranular (ground glass) look of both lung fields.

An important diagnostic technique for determining a new-born's respiratory and metabolic health is arterial blood gas (ABG) analysis. It aids in determining the severity of acid-base imbalances, hypercapnia, and hypoxaemia.

ABG PARAMETERS IN NRDS

Parameter	Normal neonatal range	Findings in NRDS	interpretation
pH	7.35-7.45	<7.35 (Acidosis)	indicates respiratory acidosis
PaO ₂	60-80mmHg	<60mmHg (Hypoxemia)	due to inadequate oxygen exchange
PaCO ₂	35-45mmHg	>45mmHg (hypercapnia)	indicates poor alveolar ventilation and Carbon Dioxide retention
Bicarbonate	22-26mmol/L	>26mmol/L (in chronic cases)	metabolic compensation
Base Excess	-2 to +2 mmol/L	Negative (<-2)	indicates metabolic acidosis
SaO ₂	>90%	<90%	reflects poor oxygenation

Differential Diagnosis:-

There are a variety of underlying causes of respiratory distress in new-born's, and the lungs are not necessarily the source.

Congenital heart problems, airway abnormalities, inborn metabolic disorders, neurologic, and haematologic causes are less common extrapulmonary etiologies.^[2]

When a new-born exhibits cyanosis and respiratory distress, pulmonary hypertension should be taken into consideration.^[1]

At birth, meconium aspiration syndrome manifests as cyanosis, retractions, grunting, and noticeable tachypnea.

Transient Tachypnea of the Neonate (TTN) affects 3.6–5.7 out of every 1000 term infants and up to 10 out of every 1000 preterm infants. It can occur in near-term, term, and late preterm children.^[1,9] Alveolar fluid from the lungs and delayed resorption cause TTN.^[1,3]

Chronically high PVR causes right-to-left shunting through the foramen ovale and the ductus arteriosus, which results in hypoxaemia and persistent pulmonary hypertension of the new-born (PPHN).^[17]

Infancy sepsis has an incidence of one or two per 1,000 live births and can affect both full-term and preterm babies. In the new-born phase, symptoms could start later. Prematurity and maternal fever are risk factors, as the membrane rupture occurring more than eighteen hours before to delivery.^[18]

Congenital heart defects in newborn's with cyanotic heart disease, respiratory discomfort is often accompanied with severe cyanosis. At least 24 hours after birth, but before being released from the hospital, new-born's should be checked.^[1]

For proper care, it is essential to distinguish NRDS from other causes of new-born respiratory distress. Accurate diagnosis requires a mix of laboratory tests, radiographic findings, and clinical characteristics.

Risk Factors:-

Premature rupture of membrane and gestational diabetes were found to be important risk factors for RDS in full-term infants.^[19]

Pneumothorax, defined as air in the pleural space, can be a cause of neonatal respiratory distress when pressure within the pulmonary space exceeds extrapleural pressure. It can occur spontaneously or as a result of infection, meconium aspiration, lung deformity, or ventilation barotrauma. The incidence of spontaneous pneumothorax is 1 to 2 percent in term births, but it increases to about 6 percent in premature births.^[20,21]

Meconium aspiration syndrome causes significant respiratory distress immediately after delivery. Hypoxia occurs because aspiration takes place in utero.

PIE, or pulmonary interstitial emphysema. Air leaks and gets stuck between the lungs' tiny air sacs, or alveoli. Bronchopulmonary dysplasia, another name for chronic lung disease.^[22]

Infants with patent arteriosus ductus.

Preeclampsia, maternal hyperglycemia, chorioamnionitis, and illegal substance use are risk factors of NRDS.

Management:-

The following are the fundamentals of treatment:

To help with differential diagnosis, take into account the patient's or mother's medical history and risk factors.

Determine the level of distress.

Extreme distress (apnoea, cyanosis, intense grunting or nasal flaring).

Verify mild distress, (such as grunting or mild tachypnea), watch for ten to twenty minutes.

Consider a blood glucose meter: newborn's who are at risk for hypoglycemia, such as premature newborn's, low birth weight infants, or infants of diabetes mothers, should ideally have glucose levels kept above 50 mg/dL (2.8 mmol/L).^[1]

Evaluate breathing, circulation, temperature, and airway.

Check blood pressure, heart rate, and urine production.

In the birthing room, a prophylactic surfactant is injected. Surfactant is given to newborn's at significant risk for RDS (those under 30 weeks gestation) before to the start of ventilation and for up to 20 minutes of life. Provide treatment to mothers at risk of preterm delivery (24–34 weeks gestation) to enhance fetal lung development.^[22,23]

In severe situations, pulmonary vasodilators like sildenafil (Revatio) or inhaled nitric oxide, as well as ventilator or vasopressor assistance, may be beneficial.

Conclusion:-

Identifying respiratory distress in the infants/neonates quickly and comprehending the physiologic anomalies linked to each of the many causes can help determine the best course of action.

Reference:-

1. Hermansen CL, Mahajan A. Newborn respiratory distress. *American family physician*. 2015 Dec 1;92(11):994-1002.
2. Warren JB, Anderson JM. Newborn respiratory disorders. *Pediatr Rev*. 2010;31(12):487–495, quiz 496
3. Edwards MO, Kotecha SJ, Kotecha S. Respiratory distress of the Term newborn infant. *Paediatr Respir Rev*. 2013;14(1):29–36
4. Hibbard JU, Wilkins I, Sun L, et al; Consortium on Safe Labor. Respiratory morbidity in late preterm births. *JAMA*. 2010;304(4):419–425
5. Sweet DG, Carnielli V, Greisen G, et al; European Association of Peri-Natal Medicine. European consensus guidelines on the management of neonatal respiratory distress syndrome in preterm infants—2013 Update. *Neonatology*. 2013;103(4):353-368.
6. [Possmayer F. Biochemistry of pulmonary surfactant during fetal development and in the perinatal period. *Pulmonary surfactant*. 1984:295-355.]
7. Minnes AS. Respiratory distress in the newborn. Retrieved from <<http://Learnpediatrics.sites.olt.ubc.ca/files/2010/07/RespiratoryDistress.pdf>>.
8. Reuter S, Moser C, Baack M. Respiratory distress in the newborn. *Pediatr Rev* 2014;35 (10):417–28.
9. Pramanik AK, Rangaswamy N, Gates T. Neonatal respiratory distress: a Practical approach to its diagnosis and management. *Pediatr Clin N Am* 2015;62:453–69.
10. Parkash A, Haider N, Khoso ZA, Shaikh AS. Frequency, causes and outcome of Neonates with respiratory distress admitted to Neonatal Intensive Care Unit, National Institute of Child Health, Karachi. *JPMA* 2015;65:771–5.
11. Ma XL, Xu XF, Chen C, Yan CY, Liu YM, Liu L, et al. Epidemiology of respiratory Distress and the illness severity in late preterm or term infants: a prospective Multi-center study. *Chin Med J* 2010;123(20):2776–80.
12. Ogunlesi TA, Ogunfowora OB. Pattern and determinants of newborn apnea In an under-resourced Nigerian setting. *Niger J Clin Pract* 2012;15 (2):159–64.

13. Dani C, Reali MF, Bertini G, Wiechmann L, Spagnolo A, Tangucci M, et al. Risk Factors for the development of respiratory distress syndrome and transient Tachypnoea in newborn infants. *Eur Respir J* 1999;14:155–9.
14. World Health Organization. *Managing newborn problems: a guide for doctors, Nurses, and midwives*; 2003. P. F-49
15. Raimondi F, Yousef N, Migliaro F, et al. Point-of-care lung ultrasound in neonatology: Classification into descriptive and functional applications. *Pediatr Res* 2021;90:534-531
16. Evans JJ. Prediction of respiratory-distress syndrome by shake test on newborn Gastric aspirate. *N Engl J Med.* 1975;292(21):1113–5.
17. Konduri GG, Kim UO. Advances in the diagnosis and management of persistent pulmonary hypertension of the newborn. *Pediatr Clin North Am* 2009;56:579-600.
18. Jesitus J. Sepsis: neonates require high suspicion, quick action. January 1, 2015. *Contemporary Pediatrics*. <http://contemporarypediatrics.Modernmedicine.com/contemporary-pediatrics/news/sepsis-neonates-Require-high-suspicion-quick-action>. Accessed October 9, 2015.
19. Liu J, Yang N, Liu Y. High-risk factors of respiratory distress syndrome in term neonates: a retrospective case-control study. *Balkan Med J.* 2014;31:64–8. Doi: 10.5152/balkanmedj.2014.8733. [DOI] [PMC free article] [PubMed] [Google Scholar]
20. Davis C, Stevens G. Value of routine radiographic examination of the newborn, based on a study of 702 consecutive babies. *Am J Obstet Gynecol.* 1930;20:73.
21. Horbar JD, Badger GJ, Carpenter JH, Fanaroff AA, Kilpatrick S, LaCorte M, et al.; Members of the Vermont Oxford Network. Trends in mortality and morbidity for very low birth weight infants, 1991–1999. *Pediatrics.* 2002;110(1 pt 1):143-51.
22. Jobe AH, Bancalari E. Bronchopulmonary dysplasia. *Am J Respir Crit Care Med.* 2001;163(7):1723-1729
23. Yost CC, Soll RF. Early versus delayed selective surfactant Treatment for neonatal respiratory distress syndrome. *Cochrane Database Syst Rev* 2000;2:CD001456.