

Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)

ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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EVALUATION OF RISK FACTORS AND OUTCOME IN RESPIRATORY DISTRESS SYNDROME IN SULAIMANI MATERNITY TEACHING HOSPITAL

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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)

ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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ABSTRACT

Historically the most frequent respiratory condition of premature infants has been hyaline membrane disease or respiratory distress syndrome. This does not change the fact that preterm infants with respiratory problems such as tachypnea, retractions, grunting respirations, nasal flaring, and requiring a larger percentage of inhaled O_2 remain a substantial clinical concern.

The goal of this study was to assess the prognosis and probable causes of respiratory distress syndrome in preterm infants. **METHODS.** The prospective data collection took place at the Neonatal Care Unit of Sulaimani Maternity Teaching Hospital. The neonatal intensive care unit has 18 incubators, 2 resuscitators, 6 CPB machines, and 1 ventilator to manage both full-term and preterm infants. Data collection for 75 incidents was completed from July 1, 2015, to September 30, 2015. In this study, we evaluated both the data of moms and babies and determined the p-value. We considered a p-value below 0.05 to be significant and below 0.001 to be extremely significant.

RESULT. The study showed that there was a statistically significant association between respiratory distress syndrome and gestational age (<28 weeks) (p-value was <0.001). The p-value is 0.001, indicating that maternal smoking is protective. Respiratory distress syndrome has not been associated with maternal medications, mode of delivery, or presence of maternal illness. Moreover, there is no association between birth defects and neonatal respiratory distress syndrome. Pneumothorax was less common than apnea, aspiration, and sepsis as a complication of RDS. In this study, nine percent of the patients died.

Keywords: Premature infants, respiratory distress syndrome(RDS), complication, hyaline membrane disease (HMD), Continuous Positive Airway Pressure (CPAP).

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Introduction

Definition

Premature infants often develop respiratory distress syndrome (RDS), which was once named hyaline membrane disease (HMD). The primary cause of this illness is the lack of pulmonary surfactant in immature lungs. Pulmonary surfactant is produced during lung development and allows normal gaseous exchange between the environment and the blood. This surfactant prevents alveoli from collapsing when breathing after birth. RDS is a leading cause of death in premature babies. RDS is generally caused by a developmentally regulated lack of pulmonary surfactant (1, 2).

1.2 Pulmonary surfactant:

The fetal lung is filled with fluid and does not perform any respiratory function until birth. During the third trimester of pregnancy, the lungs make surfactant in preparation for inhaling air. Surfactant reduces the surface tension of the alveoli, allowing the alveoli to expand more freely in an effort to reduce the possibility of alveolar collapse atelectasis. The most common cause of surfactant deficit is prematurity since the generation of surfactant is developmentally controlled. Hereditary respiratory failure of term newborns and surfactant deficiency or malfunction can also be caused by genetic anomalies of the genes encoding surfactant proteins SP-B and SP-C and the ATP-binding cassette (ABC) transporter A3 (ABCA3). 3 and 4

Pulmonary surfactant is composed largely of lipids (90 %) and proteins (about 10 %), the lipids being largely phospholipids. three and four surfactants are comprised of lipids, most of which are phosphatidylcholine species. The key factor for decreasing the surfactant tension is lecithin, which constitutes roughly 60% of it (Figure 1-1). (5)

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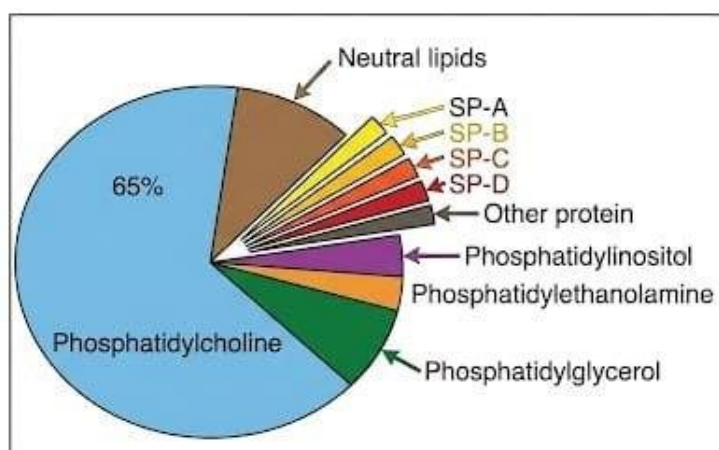


Figure 1-1 Composition of surfactant recovered by alveolar wash. The quantities of the different components are similar for surfactant from the mature lungs of mammals.

Neutral lipids SP-A SP-B SP-C Phosphatidylinositol Phosphatidylethanolamine Phosphatidylglycerol Other protein

Protein—The roles of four surfactant-specific proteins have been partially elucidated. These include the hydrophilic surfactant protein SP-A and SP-D and the hydrophobic surfactant protein SP-B and SP-C. [1, 5]

Synthesis, secretion, and adsorption processes: Alveolar type II cells synthesize surfactant by synthesizing phospholipids in the endoplasmic reticulum and then processing them through the Golgi apparatus to the lamellar bodies. Lamellar bodies include a surfactant lipoprotein complex composed of phospholipids and the surfactant proteins, SP-B and SP-C. Lamellar bodies are transported to the apical surface of the type II cell and then to the alveolar surface through exocytosis. When lamellar bodies break down in the alveoli, they generate a lipoprotein array called tubular myelin. This array is composed of SP-A, SP-B, and SP-C proteins and phospholipids and helps in the formation of a surface film in the alveoli and airways. It also decreases the alveolar surface tension. Surfactant released in the airspaces is translocated to type II cells and re-endocytosed into the cell as multivesicular bodies and then as lamellar bodies.

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Surfactant recycling, both internal and external, is of tremendous advantage to the surfactant pool. There is no tubular myelin in SP-A-deficient mice. [1, 7] Premature birth is related to a decrease in surfactant activity that presents as red diaper syndrome (RDS) due to a decrease in the amount and quality of surfactant. The production of surfactant declines steeply with decreasing gestational age, and there are significant differences in the lipid and protein content of surfactant between term and preterm babies, with the latter having less active surfactant. Surfactant from mature lungs contains 10% phosphatidylinositol, while surfactant from immature lungs has 8% phosphatidylglycerol and less than 1% phosphatidylglycerol (figures (1-2)(1-3)). More developed surfactant has a larger concentration of phosphatidylglycerol and, hence, a higher surface activity. The phosphatidylglycerol level of the amniotic fluid increases after 35 weeks of gestation, which is indicative of fetal lung maturity. (8) There are also more measures of fetal lung development available. For example, lamellar body counts and the lecithin/sphingomyelin (L/S) ratio are two measures of fetal lung development. As described previously, lamellar structures appear in the airway epithelial cells, indicating the commencement of surfactant production at roughly 20 weeks of gestation. Counting lamellar bodies in amniotic fluid is a clinical tool used to determine fetal lung maturity and surfactant production. Preterm lung surfactant has a low protein content compared to lipid content. In the human lung, lamellar bodies in type II cells normally appear at 22 weeks of gestation, while surfactant protein mRNA expression is minimal until later in pregnancy. The expression of the four surfactant proteins varies with gestational age: SP-A mRNA expression increases after 32 weeks of gestation, SP-C mRNA expression is high at the tip of the branching airways in the early stages of lung development, and SP-D mRNA expression is low until late gestation. the ninth Prenatal glucocorticoid therapy improves neonatal lung function and hence reduces the risk of RDS in premature infants by stimulating enzymes that promote

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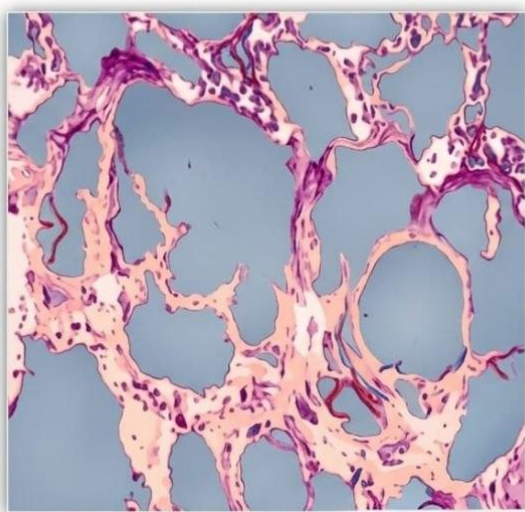
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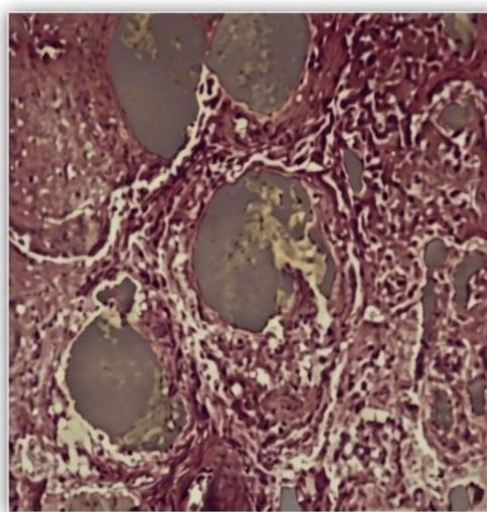
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phospholipid formation and surfactant release and by promoting maturational changes in lung architecture. in ten minutes



Figure(1,2) Alveolar system of normal lung.



Figure(1-3) Lung morphology in a Severe affected lung.

Incidence

Respiratory Distress Syndrome (RDS) in Neonates is an important developmental condition characterized by the deficit of surfactant in preterm newborns. Here is a detailed and structured summary of the text you supplied:

1. appearance

The risk is 93% higher for extremely preterm babies, those delivered before 28 weeks of gestation. Risk is inversely related to the gestational age. Late preterm incidence is for newborns born at 34 and 36 weeks and 6 days.

Gestational age-specific incidence rates: 10.5 percent in 34 weeks, 6.0 percent in forty-five weeks. 2.8% 3 months

1.0% at 37 weeks' gestation After 38 weeks, the proportion is 0.3%.

2. The Science of Disease

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This condition is mainly due to the lack of pulmonary surfactant, which results in high surface tension at the end of exhalation, instability of the lungs, poor lung capacity, and impaired compliance. According to LaPlace's Law ($P = 2T/R$), high pressure (P) is required to keep alveoli open when surface tension (T) is high and alveolar radius (R) is short. If there is inadequate pressure, the alveoli do not collapse.

Impact of downward trickling:

Diffuse atelectasis is a diffuse alveolar collapse. This condition leads to a mismatch between ventilation and perfusion (V/Q mismatch) and right-to-left shunting. It produces severe hypoxemia. Loss of surfactant causes damage to the respiratory epithelium, which leads to the production of cytokines, influx of neutrophils, and accumulation of protein-rich pulmonary fluid (edema) that causes inflammation and lung injury. The surfactant is substantially less efficient when it is exposed to meconium, blood, proteinaceous edema, and the stress of mechanical ventilation (volutrauma).

3. Symptoms in Clinic

- Onset: Cases commonly worsen over the first 48 hours of life without treatment, and symptoms typically present between minutes and hours after birth.

Clues to the Important:

1. Increased rate of breathing
2. A burning nose

Breathing out noise

four forms of retractions, intercostal subxiphoid, and subcostal cyanosis (due to shunts from right to left) In the first twenty-four hours there will be less breath sounds, pallor of skin, weaker peripheral pulses, and less urine production. Then the urine flow will increase dramatically and the patient will recover.”

Diagnosis

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- Maternal diabetes, preterm labor, and neonatal hypoxia history.

The chest x-ray shows air bronchograms, low lung volumes, and the characteristic diffuse reticulogranular ground-glass appearance.

Level 1: Minimal reticulogranular pattern.

Level II: Mottled air bronchogram.

Patchy mottling level III.

Bilateral confluent opacification (Grade IV) “white-out” of the lungs

- Controlled environment research: Arterial blood gas (ABG) shows hypoxemia, and arterial carbon dioxide tension (PaCO_2) is somewhat elevated initially but increases with progression of the disorder.

Hyponatremia is caused by fluid retention (managed by restricting fluid consumption).

Blood cultures are necessary to rule out sepsis and Group B Strep (GBS) pneumonia.

Differential Diagnosis

1. TTN (Transient Tachypnea of the Newborn)
2. Germicidal bronchitis (GBS especially)
3. Pneumothorax and other air-leak syndromes
4. Congenital heart disease with cyanosis
5. Metabolic acidosis, hypothermia, anemia, hypoglycemia, and other systemic diseases not related to the lungs
6. Chronic Aspiration and Pulmonary Hypertension
7. Birth defects (CPAM, diaphragmatic hernia)

Prevention

1. we need to do our utmost to prevent unnecessary or planned C-sections at inconvenient times.
2. Fetal observation during pregnancy and delivery to prevent hypoxia.

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3. Giving prenatal corticosteroids to mothers between 24 and 34 weeks gestation significantly reduces the frequency and severity of RDS, infant mortality, intraventricular hemorrhage, neural tube abnormalities, and developmental delays.

4. surfactant prophylaxis should be administered early to high-risk preterm babies.

Treatment & Care

Treatment With Substitute Surfactant

Emergency Treatment: Recommended for neonates found with a high oxygen requirement.

The treatment is given if the arterial-to-alveolar oxygen ratio is less than 0.22 or 0.3, or if PaO₂ is less than 80 mmHg at a blood oxygen saturation above 40%.

Prevention or early treatment: Given promptly to infants younger than 30 weeks gestation to reduce the risk of air leaks and death.

Single-dose regimens are associated with higher rates of pneumothorax and mortality than several doses (3-4 doses depending on oxygen demands).

B. Mechanical Ventilation & CPAP

The main goals of Continuous Positive Airway Pressure (CPAP) in non-failing preterm infants ($\geq 1500\text{ g}$) are to preserve functional residual capacity, prevent atelectasis, and minimize the dangers of mechanical ventilation (such as Bronchopulmonary Dysplasia—BPD). Mechanical ventilation with positive end-expiratory pressure (PEEP) is used in babies with severe respiratory failure or chronic hypoxemia for treatment of atelectasis and facilitation of surfactant administration. supports CPAP in babies with a birth weight of 1500 g or more. However, few data are available on the benefits of CPAP in newborns with an exceptionally low birth weight (less than 1500 g at birth). A meta-analysis showed CPAP to be a better modality for larger babies with respiratory distress

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syndrome, as there was a lower mortality and morbidity rate in babies born prematurely with a birth weight of more than 1500 g who were put on CPAP as compared to those who were initially on mechanical ventilation. ^(36,37) .The most recent recommendation is that infants born before 30 weeks of gestation or with a birth weight less than 1500 g and respiratory distress syndrome (RDS) can be extubated and stabilized on CPAP after intubation and surfactant administration, provided they are active, demonstrate signs of spontaneous respiratory effort, and are not in respiratory failure. “This approach can be applied to very low birth weight infants.” CPAP is the primary treatment for respiratory distress in larger preterm infants. There is no apparent consensus on the ideal way to ventilate patients requiring mechanical ventilation. not breathing properly because of it. There are several ways of mechanical ventilation, including pressure control, patient-triggered, volume control, and high-frequency positive pressure breathing. To summarize, there are no present studies showing a better long-term prognosis for any of the classical methods of ventilation. Depending on the clinician’s expertise and background, certain modes may be more suited in different clinical settings. Regardless of the style of ventilation, the reduction of the length of mechanical ventilation and the improvement of management to facilitate weaning from ventilator support are of paramount importance. = ^(37,38) If respiratory failure occurs, the patient should be placed on assisted ventilation according to the criteria for mechanical ventilation. One of these is a sign of respiratory failure:

1. Respiratory acidosis as demonstrated by an arterial pH less than 7.20 and PaCO₂ more than 60 mm Hg.
2. Hypoxia, defined as an arterial PaO₂ < 60 mm Hg, despite a 70 percent oxygen supplement on nasal CPAP.
- No. 3, severe sleep apnea.

The advantage of treatment of chronic pulmonary hypertension in term or late preterm children with inhaled nitric oxide (iNO) is well documented. Moreover,

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in animal trials, iNO improved surfactant function, attenuated hyperoxic lung injury, boosted lung growth, and reduced inflammation.

POSTNATAL GLUCOCORTICOID—Circulatory and pulmonary function is improved, and the incidence of BPD is reduced in premature infants who are started on postnatal glucocorticoid therapy on the first day of life. The use of corticosteroids after birth has been severely restricted because of short-term issues (intestinal perforation, metabolic instability) and, more importantly, poor long-term neurodevelopmental results in treated infants. The American Academy of Pediatrics committee on pregnancy and newborns suggests postnatal steroid medication only when the infant is not doing well on maximal supportive care.

1.9.5 Antibiotic Treatment – A newborn may have pneumonia at birth that appears to be RDS both clinically and on x-ray. Therefore, it is advised that all neonates diagnosed with RDS should have a full blood count (CBC), blood cultures, and empiric antibiotic treatment with ampicillin and gentamicin. If there is no growth in the blood culture after 48 hours, the antibiotic treatment is usually stopped unless there is a reason to continue it based on the patient's pregnancy history or clinical state. Moreover, further supportive care of preterm newborns is required to optimize the effectiveness of the specific procedures for treating RDS already mentioned. ^(40,41) We want to put these babies in a temperature-neutral environment so they don't have to work so diligently to maintain their core temperature. Thermostatically correct behavior reduces oxygen.

Antibiotic Treatment—RDS and pneumonia may be indistinguishable on radiograph and clinical examination at birth. Therefore, it is advisable that all babies diagnosed with RDS have a complete blood count (CBC), blood cultures, and empiric antibiotic treatment with ampicillin and gentamicin. If blood cultures are negative after 48 hours, antibiotics are stopped unless the clinical picture or obstetric history suggests a longer course.

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SUPPORTIVE CARE—More supportive care for premature babies is needed to enhance the effectiveness of the targeted treatments for RDS that have been described. ^(40, 41) **Thermoregulation**—Infants must be kept in a temperature-neutral environment to maintain a constant core body temperature. Proper thermoregulation aims to reduce oxygen use and caloric needs. Avoid rectal temperatures in infants with RDS because of increased risk of injury or perforation. Therefore, the servo-controlling temperatures of radiant heaters and incubators should be set at stomach temperatures.

In small preterm infants (<1000 g), the ambient temperature should be maintained at 36.9°C for the anterior abdominal skin and at 36.5°C for the larger infants, which is equivalent to 37°C for rectal temperatures.

Fluid balance—Because excess fluid may raise the risk of patent ductus arteriosus, necrotizing enterocolitis (NEC), pulmonary edema, and mortality, it is important to manage fluid balance closely. Excessive fluid may also increase the risk of bronchopulmonary dysplasia (BPD). by excessive fluid. ^(40, 41)

Cardiovascular treatment, systemic hypotension, and failure to close the patent ductus arteriosus increase the risk of NEC and BPD.

The early stages of RDS commonly have symptoms of systemic hypotension. The umbilical artery catheter is used to continuously monitor arterial pressure in mild to severe RDS patients. Hypotension is treated using vasopressors and careful administration of crystalloid solutions to dilate the blood vessels in the body. In cases of persistent hypotension in spite of vasopressor and volume therapy, evaluation of serum cortisol levels and administration of stress doses of hydrocortisone may be indicated.

Patent ductus arteriosus (PDA) is common in premature newborns with RDS. If so, the patient is more likely to develop BPD and have difficulty being weaned off mechanical ventilation.

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COMPLICATIONS

The mortality and morbidity of RDS The use of prenatal corticosteroids and exogenous surfactant therapy has reduced the mortality and morbidity associated with RDS. Prenatal corticosteroids and exogenous surfactant therapy have reduced the mortality and morbidity associated with RDS. The mortality and morbidity associated with RDS have Prenatal corticosteroids and exogenous surfactant therapy have reduced the mortality and morbidity associated with RDS. by the use of prenatal corticosteroids and exogenous surfactant therapy. But there are still difficulties, and there are still fatalities. Treatment is one of the common causes of complications. Methods used to manage these complications include endotracheal tube placement, supplemental oxygen, arterial catheters, and positive pressure ventilation. Endotracheal tube problems—The endotracheal tubes are commonly displaced or misplaced. Endotracheal intubation of a main stem bronchus leads to hyperinflation of the ventilated lung and atelectasis of the contralateral lung. In hyperinflation, air-block syndrome and air leakage may occur. Two further issues with intubation are atelectasis at extubation and subglottic stenosis. Perforations of the throat or esophagus are extremely rare; these may stay in the mediastinum or extend into the pleural space.^(42,43) The most common acute complication of RDS is pulmonary air leakage, also known as air block syndrome. Positive pressure from mechanical ventilation most often causes the rupture of an expanded alveolus, leading to air leaks. Air is released from the ruptured alveoli into the perivascular connective tissue sheaths. If this dissection extends to the hilum, pneumomediastinum and pneumothorax may develop. The rarer conditions of pneumopericardium, subcutaneous emphysema, and pneumoperitoneum result from the rupture of air into the pericardial space, subcutaneous tissue, and peritoneal space, respectively. The most common long-term sequel of respiratory distress syndrome (RDS) is chronic lung disease of the newborn or bronchopulmonary dysplasia (BPD). Although there is improved management of RDS, the incidence of borderline personality disorder (BPD)

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remains high. BPD is a condition with a complex etiology. Volutrauma, barotrauma, oxygen toxicity, and infection increase inflammation, which plays an important role in the course of this disease. The lung seems to be most susceptible prior to the onset of alveolar formation, which takes place around 31 to 34 weeks of gestation. This phenomenon is because a preterm lung has immature airway support structures, antioxidant systems, compliance, and fluid clearance. The lung also has no surfactant and is anatomically and functionally immature.

Patients & Methods

Settings of the study

The Neonatal Care facility (NCU) of Sulaimani Maternity Teaching Hospital was the main aseptic neonatal care facility in the city, serving patients both from the city and from its outskirts. There was a prospective study done. This neonatal intensive care facility looks after both premature and full-term infants and has 18 incubators, 2 resuscitators, 6 CPAP machines, and 1 ventilator.

Methods and sample size:

The researcher collected data for the 75 cases that comprise the sample from July 1, 2015, to September 30, 2015, visiting the NCU three times a week. The initial appointment is to choose cases. During the second visit, investigations that do not take much time. The researcher conducts quick investigations during the second visit. Finally, at the third visit, results of very time-consuming studies, e.g., blood cultures, are taken.

Cases selection:

The study included both male and female neonates delivered by regular vaginal delivery or by cesarean sections assisted by RDS by clinicians who used the clinical markers and available investigations.

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2.4 Sources of the informations:

The questionnaire contains the following information:

A. Background:

- i. Background information Name of the baby Place of residence Age in hours Sex Estimated Gestational age (based on last menstrual period & early abdominal ultrasound) Weight first- or second-born
- ii. Maternal illnesses such as DM, HT (if +ve, is it required or gestational), UTI, APH, PROM duration, or others.
- iv. Pregnancy: history of drug usage.
- iv. Whether the mother smoked throughout pregnancy, either actively or passively.
- v. Type of Delivery...
- vi. A history of respiratory distress syndrome (RDS) in previously born siblings.

Part B. The Check-up:

- i. Physical examination of the neonate.

For diagnosis of anomalies in the occipitofrontal region. Other abnormalities: fever, paleness, lethargy, excessive oral secretions, weight, length, vital signs, apgar score, and dysmorphic features.

- ii. Signs of respiratory difficulty:

- a) Tachypnoea or respiratory rate in breaths per minute in the relaxed infant;
- b) The use of respiratory accessory muscles demonstrated by intercostal, subcostal, and suprasternal muscular retractions.
- c) Laughing. dic) Central cyanosis.
- a) Narcolepsy.

Section iii: Listening to chest:

BotTachypnoea indicates rapid breathing, abnormal air entry, and enhanced heart sounds. This finding indicates the presence of rapid breathing, abnormal air entry, additional sounds, and enhanced heart sounds.

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C. Research

- i. CBC (hemoglobin, WBC total and differentiation, and platelet count).
 - ii) Blood test for total and immature neutrophils.
 - iii) Serum testing.
 - iv. Blood gas analysis (GGA) for blood pH, pH_2 , and HCO_3 (note: the PO_2 notation is reliable as the blood was not collected from the artery).
oximetry to determine O_2 saturation.
 - vi. Random blood sugar (RBS) to rule out hypoglycemia.
- Chapter VII: Mother and Child Blood Types and Rh Factor
- vi. X-ray Chest (CXR)

Result (D):

These main challenges, and the destiny of the infant. The statistics for this were obtained directly from the pediatricians treating the child and their follow-up file in the patient's medical record.

2.5 Design of the study:

The final sample of 75 cases. The RDS research sample was examined. We also discovered and examined the occurrence of apnea, air leak, sepsis, bronchopulmonary dysplasia, aspiration, intraventricular hemorrhage, and necrotizing enterocolitis. For each newborn baby, an inquiry document was filled out. Questionnaires were also completed for infants with respiratory problems. These sheets contained information on the type of respiratory support given to the baby (mechanical ventilation, oxygen therapy through mask, nasal cannula, or head box), the number of major complications the mother had, her age, the number of pregnancies, the delivery method, and whether corticosteroids had been used to induce fetal lung maturity. Also on the form were immunization, diabetes in pregnancy (preconception and prenatal), placenta praevia or placental

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abruption, intrauterine growth retardation, maternal fever > 24 hours, and premature rupture of fetal membranes (PROM).

Diagnostic criteria

The radiological criteria for RDS are a characteristic pattern with decreased air content and a reticulogranular pattern of the lung. Increased dependence on oxygen within the first twenty-four hours after delivery, absence of infection, and the onset of RDS within six hours of birth.

Exclusion criteria

All children with respiratory problems who had a history of infection in the mother were excluded.

1. Possible history of infection in the mother.
3. Positive septic screen, which can be defined as immature/total neutrophil (I/T) ratio, positive C-reactive protein, or positive blood culture.
4. TTN
5. MAS
2. Clinical signs of infection, which can be defined as temperature changes, poor feeding, decreased activity, or lethargy.
3. Sixth pneumonia.

Statistical methods

Risk variables were evaluated in relation to the onset of RDS. The p-value was considered statistically significant if it was < 0.05 and highly significant if it was < 0.001.

2.6 Evaluation of breathlessness:

The degree of respiratory distress was determined by using the Downe's score as shown in the table (2-1).

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Table (2-1) Evaluation of Respiratory Distress

Test	Score		
	0	1	2
Resp. rate	< 60/min	60-80/min	> 80/min
Retraction	No	Mild	Severe
Cyanosis	No	Relieved by O2	With O2
Air entry	Good bilateral	Mild decrease	No air entry
Grunt	No	Audible by stethoscope	Audible by ear

Evaluation	
Total Score	Diagnosis
< 4	No respiratory distress
4 - 5	Mild
6 - 7	Moderate
> 7	Severe

RESULTS

Table 3.1: Association Between Sex and RDS Development

Sex	No RDS N(%)	Mild N(%)	Moderate N(%)	Severe N(%)	Total N(%)
Male	18 (39.1%)	12 (26.1%)	13 (28.3%)	3 (6.5%)	46 (61.3%)
Female	4 (13.8%)	13 (44.8%)	10 (34.5%)	2 (6.9%)	29 (38.7%)
Total	22 (29.3%)	25 (33.3%)	23 (30.7%)	5 (6.7%)	75 (100.0%)

p-value = 0.110 (Not significant). Males accounted for 61.3% of total cases.

There was no association between both sexes and the development RDS

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GESTATIONAL AGE

Table (3.2) the association of gestational age (in weeks) and RDS.

Gestational age (in weeks)	Presence & severity of RDS								Total	
	no RDS		Mild		moderate		Sever		No.	%
	No.	%	No.	%	No.	%	No.	%		
≤ 28wks.	1	20,0%	1	20,0%	0	0,0%	3	60,0%	5	6,7%
> 28-32wks.	7	30,4%	7	30,4%	9	39,1%	0	0,0%	23	30,7%
> 32-37wks.	10	35,7%	8	28,6%	10	35,7%	0	0,0%	28	37,3%
> 37-42wks.	3	25,0%	5	41,7%	4	33,3%	0	0,0%	12	16,0%
> 42wks.	1	14,3%	4	57,1%	0	0,0%	2	28,6%	7	9,3%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

The majority of the cases (74.7%) were below 38 weeks of gestation, so, there is a highly significant increase in the development of RDS in preterm neonate.

**p-value was less than 0.001 & the high percentage with gestational age between (32-37) weeks.*

Birth weight:

Table (3.3) shows the association of the birth weight and RDS, about (56%) of cases were below 2.5 Kg at birth which is considered as low birth weight. P-value was > 0.05 so there is no association with RDS.

Table 3.3: Association Between Birth Weight and RDS

Birth Weight	No RDS N(%)	Mild N(%)	Moderate N(%)	Severe N(%)	Total N(%)
< 1.0 kg	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (100.0%)	1 (1.3%)
1.0 – 1.499 kg	10 (37.0%)	7 (25.9%)	8 (29.6%)	2 (7.4%)	27 (36.0%)
1.5 – 2.499 kg	2 (14.3%)	6 (42.9%)	6 (42.9%)	0 (0.0%)	14 (18.7%)
2.5 – 4.0 kg	10 (31.3%)	11 (34.4%)	9 (28.1%)	2 (6.3%)	32 (42.7%)
> 4.0 kg	0 (0.0%)	1 (100.0%)	0 (0.0%)	0 (0.0%)	1 (1.3%)
Total	22 (29.3%)	25 (33.3%)	23 (30.7%)	5 (6.7%)	75 (100.0%)

p-value > 0.05 (Not significant). 56.0% of neonates had low birth weight (<2.5 kg).

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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Type of delivery

Table (3.4), reveals that 62.7% of the cases were delivered by cesarean section. Association was not found between both cesarean section and normal vaginal delivery and development of RDS, P-value was > 0.05 .

Table (3.4) The association between mode of delivery and RDS.

Mode of delivery	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
C/S	16	34,0%	14	29,8%	15	31,9%	2	4,3%	47	62,7%
NVD	6	21,4%	11	39,3%	8	28,6%	3	10,7%	28	37,3%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

Table (3.5) The association between maternal diseases during pregnancy and RDS.

Maternal disease	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
D.M.	6	60,0%	2	20,0%	2	20,0%	0	0,0%	10	13,3%
HT	1	33,3%	1	33,3%	1	33,3%	0	0,0%	3	4,0%
UTI	5	31,3%	6	37,5%	5	31,3%	0	0,0%	16	21,3%
APH	0	0,0%	1	50,0%	0	0,0%	1	50,0%	2	2,7%
PROM	2	25,0%	1	12,5%	4	50,0%	1	12,5%	8	10,7%
Non	8	22,2%	14	38,9%	11	30,6%	3	8,3%	36	48,0%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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Maternal smoking

Table (3.7) shows the association between the maternal smoking during pregnancy and the development of RDS. There was protective effect and significant because of p-value was 0.025.

Table (3.7) the association between the maternal smoking during pregnancy and RDS.										
Smoking history	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
Present	5	83,3%	0	0,0%	1	16,7%	0	0,0%	6	8,0%
Absent	17	24,6%	25	36,2%	22	31,9%	5	7,2%	69	92,0%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

Table (3.9) The association between the multiple pregnancies and the development of RDS.

Pregnancy	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
Single	19	29,2%	22	33,8%	19	29,5%	5	7,7%	65	86,7%
Twin	3	30,0%	3	30,0%	4	40,0%	0	0,0%	10	13,3%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

Table (3.10) The association between neonatal congenital anomalies and the development of RDS.

Congenital anomalies	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
Positive	2	20,0	3	30,0	5	50,5	0	0,0	10	13,3
Negative	20	30,8	22	33,8	18	27,7	5	7,7	65	86,7
Total	22	29,3	25	33,3	23	30,7	5	6,7	75	100,0

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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Maternal drug intake

Tables (3.6) show the association between maternal drugs intake and the development of RDS. The data shows there was no association between these drugs and the development of the RDS. P-value was > 0.05 .

Table (3.6) The association between maternal drug intake during pregnancy and the development of RDS.

Drugs	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	No.	%
Steroid	1	100,0%	0	0,0%	0	0,0%	0	0,0%	1	1,3%
Insulin	6	50,0%	3	25,0%	3	25,0%	0	0,0%	12	16,0%
Aldomet	0	0,0%	1	50,0%	1	50,0%	0	0,0%	2	2,7%
Antibioti cs	3	27,3%	4	36,4%	4	36,4%	0	0,0%	11	14,7%
Non	12	24,5%	17	34,7%	15	30,6%	5	10,2%	49	65,3%
Total	22	29,3%	25	33,3%	23	30,7%	5	6,7%	75	100,0%

Table (3.11) Outcome result of RDS.

Table (3.10) shows association between the severity of RDS & the outcome result which is a highly significant; P-value was less than 0.01

Outcome result	Presence & severity of RDS								Total	
	no RDS		mild		moderate		sever			
	No.	%	No.	%	No.	%	No.	%	N o.	%
Well discharged	22	36,1	25	41,0	14	23,0	0	0,0	61	81,3
Died	0	0,0	0	0,0	9	64,3	5	35,7	14	18,7
Total	22	29,3	25	33,3	23	30,7	5	6,7	75	100,0

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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Table (3.2.12) the association between complications & severity of RDS

Complications	Presence & severity of RDS			Total
	Mild	Moderate	Sever	
Aspiration.	1	2	1	4
Sepsis.	2	0	3	5
Pneumothorax.	1	0	0	1
Necrotizing enterocolitis.	0	1	2	3
Intraventricular hemorrhage.	0	0	1	1
Bronchopulmonary dysplasia.	0	0	1	1
Apnea.	0	1	3	4
Total	4	4	11	19

Discussion

RDS, or HMD, is a disease that affects preterm newborns and has a high incidence and morbidity rate internationally, especially in developing countries. Therefore, to reduce the incidence of RDS, it is important to investigate this condition thoroughly to find preventive risk factors. Sex distribution revealed that men were more likely to be impacted than females, but there was no difference in the association between the sexes and RDS, since the p-value would not have been significant either way. These results are in agreement with the results of other studies (Aseel, Basrha, Iraq 2010) and (Wisam, Karbla, Iraq 2012). Depending on the gestational age and the course of the disease. The great majority of these events happened before the 37th week of gestation. The probability of RDS is very high (p-value < 0.001), and the highest probability is between 32 and 37 weeks' gestation. However, this risk decreases with increasing gestational age. This phenomenon is because the lungs mature to a point where they can manage

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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respiration, and the incidence of hyaline membrane illness falls considerably with each week that passes. That discovery is consistent with other research, such as the studies conducted in 2003. Fetal malnutrition. In: studies 2003; The association of RDS with birth weight was comparable. This result is also in agreement with other studies like Neil (2003) and Aseel (2010), which reported that 32% of cases had birth weight between 2.5 and 4 kg. There was no significant difference in the connection between RDS and the two modes of delivery. However, C/S represented 62.7% of the occurrences. This result corroborates prior studies such as Aseel (2010) and Wisam (2012). This conclusion may The limited sample size and the inclusion of both elective and emergency C/S in our study may explain this conclusion. This conclusion may The incorporation of both elective and emergency cesarean sections in our study, coupled with the constrained sample size, may elucidate this conclusion. by the incorporation of both elective and emergency cesarean sections in our study, coupled with the constrained sample size. This conclusion may The incorporation of both elective and emergency cesarean sections in our study, coupled with the constrained sample size, may elucidate this conclusion. This conclusion may The inclusion of both elective and emergency C/S in our study, along with the limited sample size, may explain this conclusion. by the inclusion of both elective and emergency C/S in our study, as well as the limited sample size. This discovery is consistent with other studies; for example, a study from 2003, and our study also considered the impact of maternal diseases on the onset of RDS. The data showed no link between maternal illness and RDS. The results are consistent with prior studies, including one by Wisam in 2012. Contraception and medication use during pregnancy The study did not show any link between drug usage and the start of RDS (P -value > 0.05). This is in accordance with prior studies, e.g., (Wisam, 2012).

The protective effect and the correlation between maternal smoking and the development of RDS were statistically significant ($p = 0.025$). This phenomenon

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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is because when a mother smokes during pregnancy, fetal lung development and the production of respiratory surfactant are both stimulated, leading to a state of chronic stress. This finding is congruent with other studies, for instance, (Wisam, 2012). The effect of a pre-existing disease on the course of RDS. The research found no link. This is statistically significant ($p\text{-value} > 0.05$). Similar outcomes have been reported in research like (Neil, 2003). Many pregnancies are associated with the development of RDS. The RDS was not correlated with the twins. The results: Some studies, including Neil (2003) and Aseel (2010), support the results. The results are supported by some studies, including Neil (2003) and Aseel (2010).

Statistical analysis of the data did not reveal any link between RDS and neonatal congenital anomalies. Neil (2003) and other related investigations have observed similar results.

The data also showed 81.3% of patients were treated and released, while 18.7% died.

In very preterm and severe cases of RDS, there is no prospect of survival. The results of this study are compatible with several studies such as those of Aseel (2010) and Wisam (2012). It was less likely to be pneumothorax. The most prevalent complications connected with RDS in the study were apnea, aspiration, and sepsis. Some studies agree with the results, such as those of Wisam (2012) and Aseel (2010). The death toll reached 18.7 percent.

Conclusions

The main findings of this study are

1. The risk of RDS was inversely associated with both gestational age and birth weight.
2. The influence of NVD and C\S on the RDS development was the same.
3. Mothers who smoke are at considerably lower risk for RDS.
4. No association between RDS and maternal diseases.

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ISSN 2760-4942 (Online) Volume 2, Issue 9, September 2026



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5. Moreover, RDS was not related to sex or neonatal congenital anomalies. There have been 18.7 deaths. 6.

Recommendations

1. Increase prenatal care to lower the likelihood of kids being born premature or with low birth weight.
2. All neonates with high risk factors for RDS should be given early surfactant therapy shortly after birth.

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