Respiratory Distress Syndrome
(Hyaline Membrane Disease)

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INCIDENCE

- RDS occurs primarily in premature infants.
- Its incidence is inversely related to gestational age and birth weight.
- It occurs in 60-80% of infants less than 28 wk of gestational age, in 15-30% of those between 32 and 36 wk, in about 5% beyond 37 wk, and rarely at term.
Risk Factors

- Maternal diabetes.
- Multiple births.
- Cesarean section delivery.
- Precipitous delivery.
- Asphyxia.
- Cold stress.
- History of previously affected infants.
- The incidence is highest in preterm male or white infants.
The risk of RDS is reduced:

- In pregnancies with chronic or pregnancy-associated hypertension.
- Maternal heroin use.
- Prolonged rupture of membranes.
- Antenatal corticosteroid prophylaxis.
ETIOLOGY AND PATHOPHYSIOLOGY

- Surfactant deficiency (decreased production and secretion) is the primary cause of RDS.
- With advancing gestational age, increasing amounts of phospholipids are synthesized and stored in type II alveolar cells.
- They reduce surface tension and help maintain alveolar stability by preventing the collapse.
- Mature levels of pulmonary surfactant are usually present after 35 wk.
- Though rare, genetic disorders may contribute to respiratory distress.
CLINICAL MANIFESTATIONS

- Signs of RDS usually appear within minutes of birth.
- The characteristic hyaline membranes are rarely seen in infants dying earlier than 6-8 hr after birth.
- The natural course of untreated RDS is characterized by progressive worsening of cyanosis and dyspnea.
- In most cases, the symptoms and signs reach a peak within 3 days.
Improvement is often heralded by spontaneous diuresis and the ability to oxygenate the infant at lower inspired oxygen levels or lower ventilator pressures.

Death is rare on the 1st day of illness, usually occurs between days 2 and 7, and is associated with alveolar air leaks (interstitial emphysema, pneumothorax), pulmonary hemorrhage, or IVH.

Mortality may be delayed weeks or months if BPD develops in mechanically ventilated infants with severe RDS.
DIAGNOSIS

- The clinical course, x-ray of the chest, and blood gas and acid-base values help establish the clinical diagnosis.

- X-ray, the lungs may have a characteristic, but not pathognomonic appearance.

- A fine reticular granularity of the parenchyma and air bronchograms.

- Early-onset sepsis may be indistinguishable from RDS.

- In pneumonia manifested at birth, the chest roentgenogram may be identical to that for RDS.
Cyanotic heart disease (total anomalous pulmonary venous return) can also mimic RDS both clinically and radiographically.

Congenital alveolar proteinosis (congenital surfactant protein B deficiency) is a rare familial disease that manifests as severe and lethal RDS in predominantly term and near-term infants.

In atypical cases of RDS, a lung profile (lecithin:sphingomyelin ratio and phosphatidylglycerol level) performed on a tracheal aspirate can be helpful in establishing a diagnosis of surfactant deficiency.
PREVENTION

- Avoidance of unnecessary or poorly timed cesarean section.
- Appropriate management of high-risk pregnancy and labor.
- Prediction and possible in utero acceleration of pulmonary immaturity are important preventive strategies.
- Antenatal and intrapartum fetal monitoring may similarly decrease the risk of fetal asphyxia; asphyxia is associated with an increased incidence and severity of RDS.
PREVENTION

- Administration of betamethasone to women 48 hr before the delivery of fetuses between 24 and 34 wk of gestation significantly reduces the incidence, mortality, and morbidity of RDS.
- Repeated weekly doses of betamethasone until 32 wk.
- Prenatal glucocorticoid therapy decreases the severity of RDS and reduces the incidence of other complications of prematurity.
- Prenatal dexamethasone may be associated with a higher incidence of periventricular leukomalacia than betamethasone.
TREATMENT

- The basic defect requiring treatment is:

1- Inadequate pulmonary exchange of oxygen and carbon dioxide.
2- Metabolic acidosis.
3- Circulatory insufficiency.
4- Multidose endotracheal instillation of exogenous surfactant and mechanical ventilation dramatically improves survival and reduces the incidence of pulmonary air leaks.
- Calories and fluids should initially be provided intravenously.
- Excessive fluids (>140 cc/kg/day) contribute to the development of PDA and BPD.
- If an infant managed by CPAP cannot maintain an arterial oxygen tension above 50 mm Hg while breathing 70-100% oxygen, assisted ventilation is required.
Reasonable indications for mechanical ventilation use are:

1. Arterial blood pH < 7.20,
2. Arterial blood Pco2 of 60 mm Hg or higher,
3. Arterial blood Po2 of 50 mm Hg or less at oxygen concentrations of 70-100% and CPAP of 6-10 cm H2O.
4. Persistent apnea.
Repeated dosing is given via the endotracheal tube every 6-12 hr for a total of 2 to 4 doses.

**Inhaled nitric oxide (iNO)** decreases the need for extracorporeal membrane oxygenation (ECMO) in term and near-term infants with hypoxic respiratory failure.

**Metabolic acidosis** in RDS may be a result of perinatal asphyxia and hypotension.
COMPLICATIONS

- Asphyxia from obstruction of the tube.
- Cardiac arrest during intubation or suctioning.
- The subsequent development of subglottic stenosis.

**Post umbilical arterial/venous catheterization:**

Embolization, thrombosis, spasm, and vascular perforation; ischemic or chemical necrosis of abdominal viscera; infection; accidental hemorrhage; and impaired circulation to a leg with subsequent gangrene.
Bronchopulmonary dysplasia (BPD)

- Is a result of lung injury in infants have severe respiratory distress requiring prolonged periods of mechanical ventilation and oxygen therapy.
- **Treatment** of BPD includes nutritional support, fluid restriction, drug therapy, maintenance of adequate oxygenation, and prompt treatment of infection.
- Growth must be monitored because recovery is dependent on the growth of lung tissue and remodeling of the pulmonary vascular bed.
**PROGNOSIS**

- Early provision of intensive observation and care of high-risk newborn infants.
- Antenatal steroids, postnatal surfactant use, improved modes of ventilation, and developmentally appropriate care have resulted in low mortality from RDS (from 40% to \(\approx 10\%\)).
- Mortality increases with decreasing gestational age.