RDS

hyaline membrane disease
Respiratory distress syndrome, also known as hyaline membrane disease, occurs almost exclusively in premature infants.

The incidence and severity of respiratory distress syndrome are related inversely to the gestational age of the newborn infant.
Pathogenesis:

respiratory distress syndrome develops because of impaired surfactant synthesis and secretion leading to atelectasis, ventilation-perfusion (V/Q) inequality, and hypoventilation with resultant hypoxemia and hypercarbia.

Blood gases show respiratory and metabolic acidosis that cause pulmonary vasoconstriction, resulting in impaired endothelial and epithelial integrity with leakage of proteinaceous exudate and formation of hyaline membranes.

The relative deficiency of surfactant decreases lung compliance and functional residual capacity, with increased dead space.
Risk factors
1. prematurity
2. maternal diabetes
3. cesarean delivery
4. Asphyxia
5. White male infants
6. Second-born twins
7. Infants with a family history of respiratory distress syndrome
In contrast, the incidence of respiratory distress syndrome decreases with the following:

1. Use of antenatal steroids
2. Pregnancy-induced or chronic maternal hypertension
3. Prolonged rupture of membranes
4. Maternal narcotic addiction
Clinical presentation:
1. Tachypnea
2. Expiratory grunting
3. Subcostal and intercostal retractions
4. Cyanosis
5. Nasal flaring
6. Extremely immature in neonates may develop apnea and/or hypothermia
Differential Diagnoses:
1. Anemia
2. Aspiration pneumonia
3. GERD
4. Congenital pneumonia
5. TTN
6. Hypoglycemia
Fetal lung maturity tests:
Prediction of fetal lung maturity is derived by estimating the lecithin-to-sphingomyelin ratio and/or by testing for the presence of phosphatidylglycerol in the amniotic fluid obtained with amniocentesis.
Surfactant is a complex lipoprotein contains: 70-80% phospholipids, 8-10% protein, and 10% neutral lipids, primarily cholesterol. Dipalmitoyl phosphatidylcholine (DPPC), or lecithin, is functionally the principle phospholipid. Phosphatidylglycerol makes up 4-15% of the phospholipids; although it is a marker for lung maturity, it is not necessary for normal lung function.
Complications:

1. Septicemia
2. Bronchopulmonary dysplasia (BPD)
3. Pulmonary hemorrhage
4. Apnea/bradycardia
5. Necrotizing enterocolitis
6. ROP
7. Failure to thrive
8. Intraventricular hemorrhage (IVH)
Management:
1. The use of antenatal steroids to enhance pulmonary maturity
2. Appropriate resuscitation facilitated by placental transfusion and immediate use of continuous positive airway pressure (CPAP) for alveolar recruitment
3. Early administration of surfactant
4. The use of gentler modes of ventilation, including early use of "bubble" nasal CPAP to minimize damage to the immature lungs
5. Supportive therapies, such as the diagnosis and management of PDA
6. Fluid and electrolyte management
7. Trophic feeding and nutrition,