OUTLINE: Respiratory Distress Syndrome (RDS)

- Discuss the pathophysiology of RDS.
- Explore the risk factors for RDS.
- Review the presentation and diagnosis of RDS.
- Review the differential diagnosis of RDS.
- Discuss the management of RDS.

(No Disclosures)
What is RDS?

- Respiratory distress syndrome (RDS) *Hyaline membrane disease* is a major cause of respiratory distress in preterm infants.

- It is caused by *surfactant* deficiency.
A complex mixture of lipids and proteins that lowers alveolar surface tension.

--the lipid is mainly phosphatidylcholine (70%).
--the proteins include SP A, B, C and D
- SP-B and SP-C are hydrophobic proteins
- SP-A and SP-D are hydrophilic proteins
SP B

• SP-B is important and is needed for:
  – Processing surfactant
  – Storage of surfactant
  – Secretion of surfactant in the type II respiratory epithelial cells.

• Deficiency SP B causes severe respiratory distress.

• Autosomal recessive inheritance pattern of deficiency has been recognized.
SP C

- SP-C promotes the formation of the phospholipid film lining the alveolus.
- Deficiency causes interstitial pulmonary fibrosis in early childhood.
- Autosomal dominant inheritance is also recognized.
SP A and D

- SP-A and SP-D facilitate the uptake and killing of bacteria and viral pathogens by immune cells.

- May have direct antimicrobial role.
Functional Properties of Surfactant

The hydrophobic and hydrophilic properties of the phospholipids cause head to tail orientation in the air-liquid interface inside the alveolus, hence:

- lower the surface tension of the liquid lining of the alveoli
- decrease the pressure needed to keep the alveoli open and inflated.
The Laplace Relationship

Explains the relationship between intra-alveolar pressure (P) needed to counteract the tendency of the alveoli to collapse under the force of surface (wall) tension (ST) and the radius (r).
The Laplace Relationship

The pressure \((P)\) needed to stabilize the respiratory system from within is directly proportional to twice the surface tension \((ST)\) and inversely proportional to the radius \((r)\) of the structure.

\[ P = \frac{2 \times ST}{r} \]
The Laplace Relationship

The smaller the alveolar radius

\[ P \propto \frac{1}{r} \]

The greater the pressure needed
The greater the surface tension

\[ P \propto ST \]

The greater the Pressure needed
The Laplace Relationship

• **Larger alveoli** need less pressure to open up and are less likely to collapse

• **Smaller alveoli** need more pressure to open and are more likely to collapse

• **Smaller alveoli** are more likely to empty into the larger alveoli

• **More Surfactant** in the smaller alveoli will reduce the need for high pressure and reduces the likelihood of collapse
The Laplace Relationship

S. Tension (ST) = 1
Radius (r) = 1

\[ P_1 = \frac{2 \times ST}{r} \]
\[ P_1 = \frac{2 \times 1}{1} \]
\[ P_1 = 2 \]

S. Tension (ST) = 1
Radius (r) = 2

\[ P_2 = \frac{2 \times ST}{r} \]
\[ P_2 = \frac{2 \times 1}{2} \]
\[ P_2 = 1 \]

\[ P_1 > P_2 \]
Physiologic Effects of Surfactant Deficiency

- Reduced compliance
- Reduced functional residual capacity
- Uneven increased airway resistance
- Decreased alveolar ventilation
Physiologic Effects of Surfactant Deficiency

- Increased physiologic dead space
- Ventilation-perfusion imbalance
- Hypoxemia and Hypercarbia
- Respiratory and metabolic acidosis
Pathologic Events in RDS

FIGURE 42-27. Schematic representation of the complex series of acute and chronic events that lead to neonatal respiratory distress syndrome and the accompanying lung injury secondary to therapeutic intervention in these infants.
Risk Factors for RDS

- Young gestational age and low birth weight:
  - 71% incidence in infants of 501g - 750g
  - 54% incidence in infants of 751g – 1000g
  - 36% incidence in infants of 1000g - 1250g
  - 22% incidence in infants of 1251g - 1500g
Risk Factors for RDS

- Maternal diabetes
- Perinatal asphyxia
- Genetic Disorder
Clinical Presentation

- Premature infants are more affected
- Tachypnea
- Nasal flaring
- Grunting
- Oxygen requirement
Clinical Presentation

- Intercostal / subxiphoid retractions
- Cyanosis
- Pallor / decreased perfusion
- Decreased breath sounds
- Decreased urine output and peripheral edema
Diagnosis of RDS

- Good history
- Clinical presentation
- **CXR** – reticulogranular, ground-glass appearance with air bronchograms; low lung volumes
- **ABG**: hypoxemia, hypercarbia
Differential Diagnosis of RDS

- Transient Tachypnea (TTN)
- Bacterial pneumonia – esp. GBBStrep.
- Air leak syndrome
- Congenital anomalies of the lungs or heart
- Persistent Pulmonary Hypertension (PPHN)
- Aspiration Syndromes, esp. Meconium
CHEST X-RAY
CHEST X-RAY FEATURES

- Reticulo-granular pattern
- Ground glass appearance
- Homogenous and symmetrical
- Air bronchograms
- Reduced lung volume
CHEST X-RAY
CHEST X-RAY
Chest X-Ray

- Prominent vascular markings
- Fluid in the minor fissure
CHEST X-RAY
CHEST X-RAY

- Lobar streaky densities (focal or multi-focal)
- Basal confluent opacities (unilateral or bilateral)
CHEST X-RAY
CHEST X-RAY FEATURES

- Coarse pulmonary densities usually bilaterally
- Areas of hyperinflation and consolidation
- Possible air leaks (interstitial emphysema, pneumothorax and/or pneumomediastinum)
Natural History of RDS

- Typically worsen in first 48 – 72 hrs
- Followed by increased endogenous surfactant production
- A period of marked diuresis
- Resolution of disease by one week
Prevention of RDS

- Prevent premature delivery

Determine lung maturity to plan delivery by

- Biochemical tests
- Biophysical tests
Prevention of RDS

Biochemical tests to determine lung maturity:

- Lecithin/sphingomyelin (L/S) ratio: the ratio is 1:1 until 32-33 weeks GA, then lecithin concentration increases while sphingomyelin concentration remains the same.

A ratio >2 indicates low risk for RDS.
Prevention of RDS

Biochemical tests to determine lung maturity:

Phosphatidylglycerol (PG): concentration in amniotic fluid rises several weeks after rise in lecithin concentration.
- It indicates advanced lung maturity.
- Value >0.3 is associated with low risk for RDS.

Fluorescence polarization test: measures competitive binding of surfactant and albumin to a probe in amniotic fluid.
- A value of 55mg per gram of albumin indicates lung maturity.
Prevention of RDS

- Biophysical test to determine lung maturity:
  - Foam stability index: serial dilution of ethanol is added to a fixed amount of amniotic fluid to quantitate the surfactant concentration.

- Lamellar body count:
  - Direct measurement of surfactant in amniotic fluid using coulter counter. Values of 30,000-50,000/ml indicates lung maturity.
  - Measuring optical density of amniotic fluid at a wavelength of 650nm. A value >0.15 imply lung maturity.
Prevention of RDS

- Antenatal Cortocosteroid (ACS) Use
  - Leads to improvement in neonatal lung function by:
    - Enhancing maturation changes in lung architecture.
    - Induction of lung enzymes leading to biochemical maturation.
Prevention of RDS

Types of ACS Used:

- Betamethasone 12mg IM q 24hrs X 2 doses
- Dexamethasone 6mg IM q 6hrs X 4 doses.
Antenatal Corticosteroid Use (ACS).

- ACS should be given to all women at high risk for preterm delivery at < 34 weeks unless impending delivery is anticipated. Benefits accrue within few hours of administration.

- Multiple course of ACS is not encouraged, however rescue therapy may be considered if several wks have elapsed since initial course and GA is still <28-30 wks.
Antenatal Corticosteroid Use (ACS).

- Multiple weekly dosing is not encouraged.

- ACS is recommended despite presence of prolonged ROM at GA 24-32 wks when there is no evidence of chorioamnionitis.

- ACS is not recommended before 24 wks or after 34 wks GA.
Benefits of ACS

- For GA 24 – 28 wks, ACS result in:
  - Reduction in severity of RDS
  - Reduction incidence of IVH
  - Reduction in mortality
  (But no reduction in incidence of RDS)
Benefits of ACS

- For GA 29-34wks ACS result in:
  - Reduction in incidence of RDS
  - Reduction in mortality
Management of RDS

- Respiratory support

- Supplemental oxygen by
  - Nasal Cannula
  - Nasal CPAP by prongs or mask
  - Endotracheal intubation to keep O2 saturations above 88 % on pulse oximetry.
Management of RDS

- Surfactant through ET

- Prophylactic surfactant is given within 20 minutes of delivery (GA < 30-32 Weeks). Reduces incidence of:
  - pneumothorax
  - PIE
  - mortality in those <30 wks

- Early surfactant is given within 2 hrs of delivery

- Selective or rescue surfactant is given to established cases based on diagnostic criteria and mostly requiring high or prolonged oxygen supplementation.
Management of RDS

Deciding on the level of support may be based on:

- GA
- weight
- severity of illness
Respiratory Support

- Infants <1000g: prophylactic surfactant and mechanical ventilation.

- Infants 1000-1500g for mild to moderate RDS: early nasal CPAP; if FiO2 >50%, consider surfactant and mechanical ventilation.

- Infants >1500g: supplemental O2 by oxy-hood or CPAP. If FiO2 >50%, consider surfactant.
Management of RDS

- **ABG**:
  - PaO2 50-80 mmHg
  - PaCO2 40- 55mmHg
  - PH at least 7.25

- **Thermoregulation**: Neutral thermal environment reduces oxygen and energy requirement of the infant.
Management of RDS

- Fluids, electrolytes and nutrition:
  - Fluid restriction lowers morbidity and mortality.
  - Typically urine output is reduced (increased vasopressin and decreased atrial natriuretic peptide).
  - Pulmonary edema is common.
  - Maintenance electrolytes are provided after 24 hrs.
Management of RDS

- Antibiotics are started after blood culture is drawn.
- Blood transfusion, if indicated, tends to hasten recovery.
- Address PDA, if present
• Polin, Yoder and Burg Practical Neonatology, 3rd edition, 155-180, 2001