Introduction

- 12% of all live births are premature (<37 weeks gestation)
- 14% increase in preterm birthrate since 1990 (multiple birth rate, lowered threshold of fetal viability)
- 60% of preterm births occur in Africa and South Asia

Introduction

- premature newborns NICU (cost, expertise)
- complications of prematurity: ICH, NEC, sepsis
  - lung disease most common cause of morbidity
- improved survival - surfactant replacement, mechanical ventilation (modern ventilators, HFOV, ECMO)
- acute and chronic pulmonary disease and altered familiar radiologic patterns of disease.


Objectives

- Respiratory distress syndrome (RDS)
- RDS and surfactant era
- “old” bronchopulmonary dysplasia (BPD)
- “new” BPD
- Air leak complications
Neonatal Respiratory Distress Syndrome/ surfactant deficiency disorder

- RDS - clinical expression of surfactant deficiency
- premature or term infants (DM mothers)
- respiratory distress shortly after birth (< 24 hrs)
- Hyaline membrane disease – histologic appearance necrotic alveolar cells, fibrin - line terminal bronchioles
- natural history of RDS modified: corticosteroids, prophylactic/rescue surfactant replacement, sophisticated assisted ventilation techniques


Radiologic Features (‘classic” RDS)

- Surfactant deficiency - bilateral diffuse symmetric
- alveolar atelectasis – volume loss
- collapsed alveoli, transudate interstitium - reticulogranular, ground glass opacification
- obscuration pulmonary vessels, air bronchograms
- severe cases – dense consolidation ( white-out)
- mild RDS evolution typically: reticulogranular - generalized hazy opacities – clearing days to 2–3 weeks

Mild RDS

Severe RDS
Radiologic Features of RDS with surfactant

- surfactant replacement - “classic” RDS radiographic findings less common
- RDS radiographic patterns with surfactant administration - complicates image interpretation
- particularly when surfactant has been administered before baseline imaging
**Radiologic Features of RDS with surfactant**

- Localized segmental hyperinflation – may produce cystic lucencies - mimic interstitial air leak/PIE
- Unilateral improvement - resulting in hyperlucent lung with contralateral mediastinal shift - mimic tension pneumothorax.

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**Unilateral distribution of surfactant**

![Image of a chest X-ray showing unilateral distribution of surfactant](image-url)
Asymmetric surfactant effect

Radiologic Features of RDS with surfactant

- Pulmonary hemorrhage - rare complication
- clinically acute respiratory decompensation after initial surfactant response
- radiograph - sudden dense airspace consolidation
- mechanism unclear - improved ventilation, decreased pulmonary vascular resistance, promotes left-to-right shunting through ductus arteriosus

Pulmonary hemorrhage

Pulmonary hemorrhage post surfactant
Bronchopulmonary Dysplasia

“Classic” BPD

- chronic lung disease in premature infants (34 weeks, 2235g) with RDS
- treated with positive-pressure mechanical ventilation (> 3 days during first 2 weeks of life)
- required supplemental oxygen (beyond 28 days of life)
- developed characteristic radiographic abnormalities
- significant pulmonary dysfunction during first year of life
- Alveolar septal fibrosis the predominant residual feature

Radiologic criteria of “Classic” BPD

- stage I (2–3 days) reticulogranular/mild RDS
- stage II (4–10 days), near complete opacification
- stage III (10–20 days) small round lucencies (cysts) alternating with irregular opacity
- stage IV (> 1 month) larger lucencies alternating with thin strands of increased opacity, “bubbly lungs”
Proposed model for the pathogenesis of long-standing healed BPD

“Classic” severe BPD
Chest CT

- greater sensitivity than radiography,
- regional air trapping
- reticular and linear opacity - thickened interlobular septa,
- Subsegmental, lobar atelectasis,
- fibrosis;
- vascular attenuation with reduced bronchoarterial diameter ratios;
- bronchial wall thickening without bronchiectasis; and
- bullae or pneumatoceles

“Classic” severe BPD
“New” BPD

- very immature neonates (less than 30 weeks, <1200g), (did not survive previously)
- antenatal glucocorticoid administration, postnatal surfactant therapy, “gentler” ventilation
- synergy of oxidant injury and mechanical ventilation no longer considered major trigger
- Perinatal factors (low-grade chorioamnionitis) influence lung maturation likely play an important role in the pathogenesis of BPD
- fundamentally an inhibition of acinar and vascular growth during vulnerable stage of lung development


Lung development

- pseudoglandular phase (6–16 weeks gestation): airways to the level of the terminal bronchioles
- canalicular phase (16–28 weeks gestation): alveolar ducts(type II pneumocytes - surfactant) develop from respiratory bronchioles, thinning of the pulmonary interstitium allows gas exchange
- saccular phase (28–34 weeks gestation): increase in terminal sacs, thinning of interstitium, proliferation of the capillary bed, early development of true alveoli
- alveolar phase: 36 weeks gestation until 18 postnatal months,
- Factors influencing lung maturation (glucocorticoids, antepartum stressors)
Radiology of “new” BPD

- four radiographic stages of BPD less commonly observed
- normal/near normal initial chest radiograph
- gradual and subtle progression hazy reticulogranular opacity
- uniform pattern of coarse interstitial opacities without cystic lucencies
- eventually “bubbly” lungs (symmetric diffuse smaller cysts)

“New” BPD
Definition of BPD and Diagnostic Criteria

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Less Than 32 Wk</th>
<th>Greater Than 32 Wk</th>
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<tbody>
<tr>
<td>Time point of assessment</td>
<td>36 wk PMA or discharge to home, whichever comes first; treatment with &gt;21% oxygen for at least 28 d plus</td>
<td>&gt;28 d but &lt;56 d postnatal age or discharge to home, whichever comes first; treatment with &gt;21% oxygen for at least 28 d plus</td>
</tr>
<tr>
<td>Mild BPD</td>
<td>Breathing room air at 36 wk PMA or discharge, whichever comes first</td>
<td>Breathing room air by 56 d of postnatal age or discharge, whichever comes first</td>
</tr>
<tr>
<td>Moderate BPD</td>
<td>Need for &lt;30% oxygen at 36 wk PMA or discharge, whichever comes first</td>
<td>Need for &lt;30% oxygen at 56 d postnatal age or discharge, whichever comes first</td>
</tr>
<tr>
<td>Severe BPD</td>
<td>Need for ≥30% oxygen and/or positive pressure (PPV or nasal CPAP) at 36 wk PMA or discharge, whichever comes first</td>
<td>Need for ≥30% oxygen and/or positive pressure (PPV or nasal CPAP) at 56 d postnatal age or discharge, whichever comes first</td>
</tr>
</tbody>
</table>

Air Leak Phenomena

- Ventilation – airway barotrauma and volutrauma
- rupture bronchioloalveolar junctions, gas perivascular and peribronchial spaces - *pulmonary interstitial emphysema* (PIE)
- distinguished from true *emphysema*: permanent expansion of alveoli with absence of fibrosis.
- Gas dissect centrifugally: subpleural blebs, pneumothorax
- Centripetal: pneumomediastinum, -pericardium, systemic air embolism

Acute PIE.

Radiology of acute PIE

- tubular and cystic lucencies – not branching pattern of air bronchograms
- focal or diffuse, unilateral or bilateral
- unilateral PIE: pulmonary overexpansion and contralateral shift of mediastinum
- focal PIE: single or multiple well-defined thin walled cystic air collections - *pseudocysts*
Unilateral acute PIE

Focal acute PIE
**Persistent pulmonary interstitial emphysema**

- PIE lasts >1 week
- focal or diffuse
- cysts are composed of fibrous walls
- lobar persistent PIE - expanding masslike aggregate of smooth-walled cysts
- may compress adjacent lung parenchyma and cause mediastinal displacement


**Diffuse persistent PIE**
Diffuse persistent PIE

Localized persistent PIE
Localized persistent PIE

- single-lobe or multilobar
- hyperexpanded cystic lucencies
- characteristic linear and dotlike structures of soft-tissue attenuation within the cysts (bronchovascular bundles surrounded by interstitial gas)

CT of the chest for evaluation of persistent PIE

- temporal relationship between acute PIE and persistent PIE usually excludes other causes (congenital lobar overinflation, cystic pulmonary airway malformation)
- persistent PIE have been reported in neonates who received only nasal CPAP
- CT is superior to radiography in characterizing pulmonary lobar involvement

Pneumothorax

- supine infant, pleural air collects anteriorly
- pleural line often not discernible
- well-defined costophrenic sulcus (“deep sulcus sign”)
- anterior junction line - can indicate bilateral pneumothorax
- compress the thymus, “figure 8” or “pseudomass”

Pneumothorax

- Track the extrapleural space and outline the inferior aspect of the heart (“continuous diaphragm sign”)
- Dissect into soft tissues of the neck or chest
- pneumoretroperitoneum or -peritoneum
- Pericardial air (limited superiorly by pericardial reflection)
- Systemic air embolism
Pneumothorax

Bilateral pneumothoraces
Pneumomediastinum

Extensive air leak
Systemic air embolism

Conclusions

- the natural history of lung disease in premature infants have changed due to advances in perinatal medicine
- radiology face new or perplexing expressions of once predictable disease
- interpretation of a preterm neonate chest radiograph requires appreciation of surfactant effect, impact of sophisticated ventilation and patterns of chronic lung disease.
Thank you