Critical Care Radiology: The Role of Imaging in Acute Respiratory Distress Syndrome
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Overview

- Case presentation: index patient
- Use of chest radiography to distinguish between causes of airspace opacification
- Use of chest radiography to distinguish between cardiogenic and non-cardiogenic pulmonary edema
- Acute Respiratory Distress Syndrome (ARDS):
  - Definition
  - Associated disorders
  - Diagnostic criteria
  - Pathophysiology
- Imaging choices for ARDS
- Radiologic findings in ARDS by stage
- Implications of imaging for understanding ARDS pathophysiology
- Implications of imaging for managing ARDS
Index Patient: Brief History

• 33 year-old man
• Two-day history of “feeling unwell”
• Day of admission: onset of “worst headache of [his] life,” followed by nausea and vomiting
• At outside hospital, CT showed extensive subarachnoid hemorrhage and intraventricular hemorrhage
Index Patient: Brief Hospital Course

• Within an hour of arrival in the ED, becomes lethargic and is intubated
• Admitted to ICU
• Week 1: Tachycardia, hypertension, and fever
  – Therapeutic hypothermia protocol initiated
• Week 2: Pneumonia
• Week 3: Extubated
  → dyspnea, rapid hypoxemia
  → re-intubated
Index Patient

The patient’s chest radiograph follows.

It was taken 24 hours after the onset of his dyspnea and hypoxemia.

Attempt to interpret the film independently, then continue to view findings.
Index Patient:
Chest radiograph, 24 hours after onset of respiratory distress
Index Patient: Chest radiograph

Diffuse, hazy opacities

Source: Beth Israel Deaconess Medical Center PACS
Index Patient:
Chest radiograph

Diffuse, hazy opacities
Index Patient:
Chest radiograph

Source: Beth Israel Deaconess
Medical Center PACS
Index Patient: Chest radiograph

Diffuse, hazy opacities

Air bronchograms

Diffuse, hazy opacities

Source: Beth Israel Deaconess Medical Center PACS
Given only a chest radiograph with an airspace/alveolar pattern of opacification, what can we determine about its etiology?

Quite a bit.
## Airspace Opacification

<table>
<thead>
<tr>
<th>Cause of Opacification</th>
<th>Radiographic Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic pulmonary edema</td>
<td>• Diffuse</td>
</tr>
<tr>
<td></td>
<td>• Symmetric</td>
</tr>
<tr>
<td></td>
<td>• Perihilar</td>
</tr>
<tr>
<td></td>
<td>• ± Dependent</td>
</tr>
<tr>
<td>Non-cardiogenic pulmonary edema (e.g., ARDS)</td>
<td>• Patchy</td>
</tr>
<tr>
<td></td>
<td>• Asymmetric</td>
</tr>
<tr>
<td></td>
<td>• Peripheral</td>
</tr>
<tr>
<td></td>
<td>• ± Dependent</td>
</tr>
<tr>
<td></td>
<td>• ± Air bronchograms</td>
</tr>
<tr>
<td>Bronchopneumonia</td>
<td>• Patchy</td>
</tr>
<tr>
<td></td>
<td>• Asymmetric</td>
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<tr>
<td></td>
<td>• Peripheral</td>
</tr>
<tr>
<td></td>
<td>• Non-dependent</td>
</tr>
<tr>
<td>Aspiration pneumonia</td>
<td>• Patchy</td>
</tr>
<tr>
<td></td>
<td>• Asymmetric</td>
</tr>
<tr>
<td></td>
<td>• Dependent</td>
</tr>
<tr>
<td>Septic infarcts</td>
<td>• Peripheral</td>
</tr>
<tr>
<td></td>
<td>• Wedge-shaped</td>
</tr>
</tbody>
</table>

Our patient’s film showed airspace opacification that was **diffuse, peripheral, and worse at the bases** (i.e., potentially dependent) with **air bronchograms**.

It demonstrates many, but not all, of the common radiographic features of **non-cardiogenic pulmonary edema**.
Pulmonary Edema

• Abnormal accumulation of fluid in the extravascular compartments of the lung
• Net fluid movement = $K_f([P_c - P_i] - \sigma[\pi_c - \pi_i])$
• Pathophysiologic categories of pulmonary edema:
  – increased hydrostatic pressure edema
  – permeability edema with diffuse alveolar damage (DAD)
  – permeability edema without DAD
  – mixed edema

Having narrowed down the cause of our patient’s airspace opacification to pulmonary edema, we can use the features of his chest radiograph to determine the type of pulmonary edema.

The chest radiograph is a powerful tool in the critical care setting; it can provide an assessment of volume status and vascular flow patterns.
Radiographic Features of Pulmonary Edema: Cardiac vs. Non-cardiac

<table>
<thead>
<tr>
<th>Signs</th>
<th>Cardiogenic Edema</th>
<th>Fluid Overload</th>
<th>ARDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiomegaly</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Vascular redistribution</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Widened vascular pedicle</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Pleural effusions</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Kerley lines</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Peribronchial cuffing</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Airspace opacification</td>
<td>Diffuse perihilar</td>
<td>Central perihilar</td>
<td>Patchy peripheral</td>
</tr>
</tbody>
</table>

Based on our patient’s clinical presentation, the airspace opacification pattern on his chest radiograph, and the absence of CXR features seen in cardiogenic or fluid-overload pulmonary edema, we suspect that he has Acute Respiratory Distress Syndrome (ARDS).

Let’s continue by briefly examining the etiology and pathophysiology of ARDS.
ARDs: Definition and Associated Disorders

- A clinical syndrome of abrupt-onset dyspnea and hypoxemia in the setting of diffuse pulmonary infiltrates
- Disorders associated with ARDS:

<table>
<thead>
<tr>
<th>Direct lung injury</th>
<th>Indirect lung injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Aspiration of gastric contents</td>
<td>Shock</td>
</tr>
<tr>
<td>Pulmonary contusion</td>
<td>Severe trauma</td>
</tr>
<tr>
<td>Drowning</td>
<td>Multiple transfusions</td>
</tr>
<tr>
<td>Fat/amniotic fluid embolism</td>
<td>Salicylate or narcotic overdose</td>
</tr>
<tr>
<td>Smoke/toxic gas inhalation</td>
<td>Pancreatitis</td>
</tr>
</tbody>
</table>

Sources:
ARDS: Diagnostic Criteria

1. Acute onset (<7 days)
2. \( \text{PaO}_2/\text{FIO}_2 <200 \text{ mmHg} \)
   - Acute Lung Injury: \( \text{PaO}_2/\text{FIO}_2 <300 \text{ mmHg} \)
3. Diffuse, bilateral pulmonary infiltrates on frontal radiograph
4. Absence of left atrial hypertension
   - PCWP <18 mmHg if measured, or
   - No clinical evidence of elevated LA pressure

Sources:
ARDS: Pathophysiology

• Alveolar capillary membrane: 2 separate barriers
  – Vascular endothelium
  – Alveolar epithelium (type I pneumocyte)

• ARDS: Injury to, and compromise of, either barrier
  – Increased vascular permeability
    • Alveolar flooding (exudative/protein-rich)
      – Hyaline membrane formation
    • Loss of diffusion capacity
    • Damage to type II pneumocytes \( \rightarrow \) widespread surfactant abnormalities

Imaging Choices in ARDS

- **Chest radiograph**
  - To support a diagnosis of ALI/ARDS in patients fulfilling clinical criteria
  - To detect or confirm a suspected subclinical complication (e.g., nosocomial pneumonia)
  - To monitor progression or regression of prior findings

- **CT**
  - To quantify the extent of lung abnormality in patients with equivocal CXR
  - To determine the etiology of the ARDS
  - To identify areas of dependent, dense, parenchymal opacification (compression atelectasis)

Sources:
Stages of ARDS

• ARDS is comprised of three stages, each of which demonstrates distinct radiographic findings.
  1. Exudative Stage
  2. Proliferative Stage
  3. Fibrotic Stage
Stages of ARDS: Exudative Stage

• Pathophysiologic:
  – Interstitial edema, rapidly progressing to the filling/flooding of alveolar spaces with an exudate
  – Hyaline membrane formation

• Radiologic (plain film):
  – First 24 hours: normal CXR
  – Early: interstitial edema (perihilar)
  – Later: alveolar consolidation (peripheral); air bronchograms

Sources:
Companion Patient 1: 
**Early exudative stage**

Chest radiograph

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 1: **Early exudative stage**

Chest radiograph

Perihilar, interstitial opacities
Index Patient: Later exudative stage

Chest radiograph

Source: Beth Israel Deaconess Medical Center PACS
Index Patient:

**Later exudative stage**

Chest radiograph

Peripheral, alveolar consolidation

Source: Beth Israel Deaconess Medical Center PACS
Stages of ARDS: Exudative Stage

• CT findings: Gravitational gradient
  – Ventral-dorsal
    • Anterior: normal lung
    • Posterior: dense consolidation
    • In between: ground-glass opacification
  – Cephalocaudal
    • Increasing abnormal density caudally

• CT findings: Airway changes
  – Bronchial dilatation

Sources:
Companion Patient 2: Later exudative stage

CT, axial Image 1

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 2: Later exudative stage

CT, axial
Image 1

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 2: Later exudative stage

CT, axial Image 1

Ground-glass opacities
Companion Patient 2: Later exudative stage

CT, axial Image 1

Dense consolidation
Companion Patient 2: Later exudative stage

CT, axial Image 1

Normal lung
Ground-glass opacities
Dense consolidation

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 2: Later exudative stage

CT, axial Image 1
Companion Patient 2: Later exudative stage

CT, axial Image 2

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 2: Later exudative stage

CT, axial
Image 3
Companion Patient 3: Later exudative stage

CT, axial Image 1
Companion Patient 3: Later exudative stage

CT, axial Image 1

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 3: Later exudative stage

CT, axial
Image 1

Ground-glass opacities

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 3: Later exudative stage

CT, axial
Image 1

Dense consolidation
Companion Patient 3:
Later exudative stage

CT, axial Image 1

Dense consolidation

Normal lung

Ground-glass opacities
Companion Patient 3: Later exudative stage

CT, axial Image 1

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 3: Later exudative stage

CT, axial
Image 2
Companion Patient 3: Later exudative stage

CT, axial Image 3

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 3: Later exudative stage

CT, axial Image 4
Companion Patient 3: Later exudative stage

CT, axial Image 5

Source: Beth Israel Deaconess Medical Center PACS
Companion Patient 3: 
Later exudative stage

CT, axial 
Image 6
Stages of ARDS: Proliferative Phase

• Pathophysiologic:
  – Organization of fibrinous exudate
  – Regeneration of alveolar lining

• Radiologic:
  – Inhomogeneous areas of ground-glass opacity
  – Thickening of alveolar septae

Stages of ARDS: Fibrotic Stage

• Pathophysiologic:
  – Scarring/fibrosis
  – Formation of subpleural and intrapulmonary cysts

• Radiologic:
  – Distortion of interstitial and bronchovascular markings
  – Cystic lesions
  – Complications of cysts or barotrauma:
    • Aberrant air: pneumothorax, pneumatocele

Sources:
Implications of Imaging for Understanding ARDS

• CT allows quantitative analysis of volumes of gas and tissue
• CT data of the whole lung have changed our understanding of the pathophysiology of ARDS
  – Lung volume = tissue volume + gas volume
  – We now understand that there is a marked reduction in overall lung volume at the expense of the volume of the lower lobes
    • Increase in tissue in upper lobes (edema, inflammation)
    • Loss of aeration of lower lobes (compression by heart, abdominal contents)
  • Old understanding of ARDS: overall volume of lung preserved because gain of tissue was expected to exceed loss of gas
  • New understanding of ARDS: reduction in overall lung volume because loss of gas is greater than gain of tissue
  – Loss of aeration differs between patients

Lower-lobe predominant pattern

- 40% of patients
- Loss of aeration:
  - Mainly in lower lobes
  - Minimal involvement of upper lobes
- Mortality: 40%

CT scan from a 74-year-old patient with ARDS caused by severe bronchopneumonia

- Upper lobes: some parts remain normally aerated (black)
- Lower lobes: either poorly aerated (gray) or nonaerated (red).

Lower-lobe exclusive pattern

- One-third of patients
- Loss of aeration:
  - Exclusively in lower lobes
- Mortality: 40%

CT scan from 50-yr-old patient with ARDS caused by aspiration pneumonia

- Upper lobes: normally aerated (black).
- Lower lobes: either poorly aerated (gray) or nonaerated (red).

Diffuse pattern

- 25% of patients
- Loss of aeration:
  - Massive
  - Equally-distributed throughout lung
- Mortality: 70%

CT scan from in a 53-year-old patient with ARDS caused by *Pneumocystis jirovecii*

• Entire lung: nonaerated (red) or poorly aerated (gray).

Implications of Imaging for ARDS Management

• CT has led to safer and more-effective management of ARDS
  – Understanding that overall lung volume and cephalocaudal lung dimensions are reduced at the expense of the lower lobes
    • Prone and semi-recumbent positioning of patients
  – Assessment of alveolar recruitment and detection of lung overinflation
    • Optimization of PEEP: maximizing recruitment while limiting barotrauma

Summary

- The different causes of airspace opacification on plain film have distinctive radiographic appearances.
- Pulmonary edema is one such cause. It is possible to deduce the origin of pulmonary edema (cardiac, fluid overload, or ARDS) based on the radiographic features of a chest film.
- ARDS is a clinical syndrome of severe dyspnea of rapid onset and hypoxemia in the setting of diffuse pulmonary infiltrates.
- ARDS is caused by diffuse lung injury that leads to leakage of alveolar capillaries, allowing flooding of alveolar spaces with an exudate.
- The menu of imaging for ARDS includes plain film and CT.
- ARDS has three phases (exudative, proliferative, and fibrotic), each of which has distinct radiographic features.
- CT has changed our understanding of the pathophysiology of ARDS.
- CT has changed our approach to the management of ARDS.
References

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