Acute Bacterial Infections of the Lung and Lung Abscess

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Outline

• Pneumonia
• Acute Bronchitis
• Exacerbation of COPD
• Hospital-Acquired Pneumonia
• Pneumonia in the Immuno-compromised Host
• Aspiration Pneumonia
• Lung Abscess
Introduction

• Globally, lower respiratory tract infections represent principal cause of mortality.
  – In North America, they are major cause of death due to infectious disease.

• Lower respiratory tract infections may have complications requiring intervention and complicate post-op course for surgical patients.
Introduction

• Suppurative diseases of lung (empyema, lung abscess, bronchiectasis) provided major impetus for development of thoracic surgery as a specialty.

• Until 40 years ago, it represented major focus of practice.
Pneumonia
Pneumonia

• In North America, it is the 6th most common cause of death, and most common cause from infectious disease (despite advent of ABX and vaccines)

• 4 million cases of CAP each year, 20% of which are hospitalized.

• Mortality is 1-5% in outpatient setting, approaches 25% for hospitalized patients, and may be even higher for those admitted to an ICU.
Definition

• Infection of the lower respiratory tract that involves secondary lobules of the lung:
  – Respiratory bronchioles, alveolar ducts, and alveoli.

• Presentation
  – Fever, cough with/without sputum, dyspnea, and chest discomfort
  – May have abnormal findings on physical exam with infiltrates on CXR
Classification

• Initially, the pathologic organism is rarely known.

• Algorithmic classification based on clinical characteristics of pts and epidemiologic evidence aids in antibiotic selection
Community-Acquired Pneumonia (CAP)

- Usually caused by bacterial or viral pathogens especially in patients who have not recently (within 14 days) been hospitalized in an acute or chronic facility

- Common in older pts and those with comorbid conditions (COPD, CHF, DM, CRI, CLD)
Hospital-Acquired Pneumonia (HAP)

• Develops 48 to 72 hours after admission to a hospital

• Reflects some type of compromise in host defense mechanisms

• Usually bacterial (often due to Gram-negative bacilli), though fungal infections are possible
PNA in Immunocompromised Host

• Can occur based on nature of immunocompromised state
  – HIV
  – Chemotherapy
  – Organ transplantation
## Classification

<table>
<thead>
<tr>
<th>Type of Pneumonia</th>
<th>Subclassification</th>
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<tbody>
<tr>
<td>Community-acquired pneumonia</td>
<td>Age &lt;60 year, no other illness</td>
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<td>Age &gt;60 year or coexisting illness</td>
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<td></td>
<td>Severe or rapidly progressing pneumonia</td>
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<td>Pneumonia in nursing home residents</td>
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<tr>
<td>Nosocomial pneumonia</td>
<td>Ventilator-associated pneumonia</td>
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<td></td>
<td>Non–ventilator-associated pneumonia</td>
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<tr>
<td>Aspiration pneumonia</td>
<td>Infectious pneumonia</td>
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<td></td>
<td>Chemical pneumonitis</td>
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<tr>
<td>Pneumonia in the immunocompromised host</td>
<td>HIV/AIDS</td>
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<td>Chemotherapy for neoplastic diseases</td>
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<td>Organ or bone marrow transplant recipient</td>
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<td>Diabetes</td>
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<td>Other immunocompromised states (e.g., hypogammaglobulinemia, neutrophil deficiencies, cellular)</td>
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</table>
Clinical diagnosis of pneumonia

Categorize host pneumonia presentation

Outpatient CAP with no modifying factors
  - Macrolide or doxycycline
    - Second-generation macrolide or doxycycline
      - Fluoroquinolone or amoxicillin/clavulanate or second-generation cephalosporin ± macrolide
    - Fluoroquinolone or amoxicillin/clavulanate or second generation cephalosporin ± macrolide

Outpatient CAP with COPD
  - No corticosteroids or antibiotics in the last 3 months
    - Corticosteroids or antibiotics in the last 3 months
      - Fluoroquinolone or amoxicillin-clavulanate or second-generation cephalosporin ± macrolide

Nursing home CAP
  - Fluoroquinolone or amoxicillin-clavulanate or second- or third-generation cephalosporin ± macrolide

Hospital ward
  - Third-generation cephalosporin ± macrolide or piperacillin/tazobactam or fluoroquinolone

Intensive care unit
  - Blood culture, sputum Gram stain culture; consider saline-induced sputum or more invasive procedures such as bronchoscopy

Tailor treatment to predominant organism when results become available

Risk of *Pseudomonas aeruginosa*

Anti-pseudomonal fluoroquinolone (ciprofloxacin) + anti-pseudomonal β-lactam (ceftazidime, meropenem, or piperacillin/tazobactam) or macrolide + two anti-pseudomonal agents (aminoglycoside + ceftazidime, cefepime, meropenem, or piperacillin/tazobactam)
Acute Bronchitis
Acute Bronchitis

• Inflammation involving the mucosa of the trachea and bronchi

• **Most common cause:** infection due to viruses (RSV, rhinoviruses, echoviruses, influenza viruses, etc.), *Mycoplasma* bacteria, other bacteria and parasites

• Irritants include constituents of the smoke of tobacco and cannabis, gases (NH3), etc.
Bacterial Bronchitis

- Most common organisms:
  - *H. influenzae*
  - *S. pneumoniae*
  - *M. catarrhalis*

- Less common:
  - *Bordetella pertussis*
  - *Legionella*
  - *Mycoplasma pneumoniae*
  - *C. pneumoniae*

- Less common, but likely in patients who have resided in tropical climates:
  - *Strongyloides*
  - *Ascaris*
  - *Synagmus larngeus*
Clinical Syndrome

• Similar to that of pneumonia except:
  – CXR is often unremarkable
  – There is a production of mucoid or purulent sputum
  – There is often a low-grade fever
  – Small amounts of hemoptysis and substernal chest pain of a burning quality
  – Dyspnea is not a usual component of bronchitis unless other comorbidities are present (i.e. COPD)
Dx and Tx

• Diagnosis:
  – Gram stain of sputum →
    – Predominance of PMNs in bacterial bronchitis
    – Predominance of mononuclear cells in viral bronchitis

• Treatment:
  – Depends on type of patient and infection
  – Young → viruses; smokers → *H. influenzae*; elderly → bacterial; significant purulent sputum production → bacterial
Dx and Tx:

• If Gram stain suggests bacterial source, ABX is appropriate
  – Amoxicillin and clavulanic acid
  – Doxycycline
  – TMP-SMX
  – Clarithromycin
  – Azithromycin
  – Ciprofloxacin

• Also, oral hydration and humidified air are helpful adjuncts.
Exacerbation of COPD
COPD

• Group of disorders that commonly coexist:
  – Chronic bronchitis
  – Asthma
  – Emphysema
  – Bronchiectasis
  – Other small airways diseases

• Characterized by limitation of airflow due to obstruction
  – Asthma → obstruction is reversible
  – COPD → largely irreversible
Acute Exacerbations of COPD

• Common during winter months
• Sx: increase in dyspnea, wheeze, cough, sputum production

• Can be due to concomitant viral or bacterial infection, cigarette smoke, air pollution, allergies, drug toxicity, and other medical illnesses (i.e. CHF)
Acute Exacerbations of COPD

• Most guidelines recommend multifaceted approach in management:
  
  – Bronchodilators (inhaled ipratropium > beta-agonists)
  – Systemic glucocorticoids (steroids)
  – Antibiotics
  – Expectorants
  – Addressing coexisting medical conditions
Hospital-Acquired Pneumonia
Hospital-Acquired Pneumonia

• 2\textsuperscript{nd} most common hospital-acquired infection (following nosocomial UTI) with highest fatality rate of all nosocomial infections

• Mortality increases if mechanical ventilation is required

• Thought to be due to microaspiration or silent aspiration of oropharyngeal flora
Hospital-Acquired Pneumonia

• Normally, flora of healthy individuals is composed of benign commensal organisms

• With illness or trauma requiring hospitalization, flora shifts to include Gram (-) bacilli.

• Also, GPCs become more common in ICU patients, diabetics, and in head-trauma patients.
Hospital-Acquired Pneumonia

• Often, no gross episode is identified.

• Microaspiration contributes to development of PNA.

• Intubation results in increased mucus production, impaired mucociliary clearance, and loss of the normal protective barrier of the glottis.
Hospital-Acquired Pneumonia

- Accumulation of subglottic secretions in the vented pt represents an important reservoir for Gram (-) bacteria and source for VAP.

- Painful incisions may result in decreased tidal volumes, atelectasis, and decreased FRC, with impaired ability to cough and clear secretions.
Risk Factors

• Modifiable
  – Alcohol-based hand disinfection
  – Strict infection control
  – Monitoring and early removal of invasive devices
  – Programs to reduce ABX prescription
  – Active surveillance of pathogens (especially for multidrug resistance)

• Non-modifiable
  – Male gender
  – Multiple organ dysfunction
  – Underlying lung disease
  – Intubation
Ventilator-Associated Pneumonia

- Refers to nosocomial pneumonia that has developed in pts on mechanical ventilation
  - Occurs 48-72 hours after intubation, and results usually from aspiration complicating the intubation process
  - Caused by \textit{S. aureus}, \textit{H. influenzae}, \textit{S. pneumoniae}, and others.
Ventilator-Associated Pneumonia

• After first 72 hours, infection is commonly caused by antibiotic-resistant organisms:
  – MRSA
  – *Pseudomonas aeruginosa*
  – *Acinetobacter* species
  – *Enterobacter* species

• Risk of VAP is 1%/vented day and 68% for those vented more than 30 days
VAP: Risk factors

• Main
  – Need for re-intubation
  – Gastric aspiration

• Other
  – Old age
  – Thoracic or upper abdominal surgery
  – Malnutrition
  – Obesity
  – Chronic lung disease
  – Concurrent ABX use
VAP: Dx and Tx

• Dx:
  – Infiltrates on CXR
  – PaO₂ decreased
  – Sputum production
  – Presence of fever or leukocytosis
  – Bronchoscopy for BAL culture

• Tx:
  – General supportive care
  – Broad spectrum antibiotics
Important Tx Guidelines, ATS 2005

1. Prescribe early, appropriate, broad spectrum antibiotic therapy that includes agents from a different antibiotic class than what the patient received previously.

2. Consider de-escalation or discontinuation of antibiotics at 48 to 72 hours after initial prescription, based on the results of lower respiratory tract cultures and the patient’s clinical status.
3. A shorter duration of antibiotic therapy (7-8 days) is recommended for uncomplicated HAP and VAP in patients who initially received appropriate antimicrobial therapy and had a good clinical response.
Pneumonia in the Immunocompromised Host
Susceptibility to different infections

- Occurs at different time points in the clinical course of an organ transplantation recipient
  - First month
  - 1-6 months post-transplant
  - >6 months post-transplant
## DDx of Fever and Pulmonary Infiltrates in Organ Transplant Recipients

<table>
<thead>
<tr>
<th>Radiographic Pattern</th>
<th>Rate of Progression of Illness</th>
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<tbody>
<tr>
<td></td>
<td>Acute (&lt;24 hr)</td>
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<tr>
<td>Consolidation</td>
<td>Pulmonary edema</td>
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<tr>
<td></td>
<td>Pulmonary hemorrhage</td>
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<td></td>
<td>Bacterial pneumonia</td>
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<tr>
<td></td>
<td>Thromboembolism</td>
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<tr>
<td>Peribronchovascular</td>
<td>Pulmonary edema</td>
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<tr>
<td>Nodular infiltrate</td>
<td>Bacterial pneumonia</td>
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</table>

### Acute (<24 hr)
- Pulmonary edema
- Pulmonary hemorrhage
- Bacterial pneumonia
- Thromboembolism
- PCP
- Drug-related

### Subacute-Chronic (day-wk)
- Fungal
- Nocardial
- Tuberculous
- Viral
- PCP
- Nocardial
- Tuberculous
- Tumor (post-transplantation lymphoproliferative disorder)
- Fungal
- Nocardial
- Tuberculous
- PCP
In the transplant recipient...

• Two major causes of PNA in the first post-op month
  – Recurrence of an incompletely treated PNA present before transplantation
  – Infection due to aspiration of nosocomial flora resulting from post-op vomiting
From one to six months post-op...

- CMV and other immuno-modulating viruses are the predominant opportunistic infections

- CMV
  - Directly causes viral pneumonia
  - Can also contribute to graft-vs-host disease (GVHD), leading to need for more immunosuppression and increased risk of opportunistic infections like *Cryptococcus*, *Aspergillus*, and *PCP*
Beyond six months post-transplantation...

- Pts should have more modest immunosuppression and are susceptible to:
  - RSV
  - Influenza
  - Pneumococcal pneumonia

- Pts with poorer graft function – requiring more immunosuppression – are at higher risk of infection with *Cryptococcus*, *PCP*, *Nocardia*, and *Aspergillus*.
Diagnosis

• Dx:
  – **Bronchoscopy with BAL +/- transbronchial biopsy** (useful for PCP and Cryptococcal infection)
  – **Percutaneous needle biopsy**: procedure of choice for invasive diagnosis of focal pulmonary processes.
  – **Thoracoscopic biopsy**:
    – provides better specimen for culture and pathologic assessment
    – has less associated morbidity & mortality than open lung biopsy
Aspiration Pneumonia
Aspiration

• Three syndromes

  – Gastric acid aspiration

  – Bacterial pneumonia

  – Aspiration of a foreign body causing airway obstruction
(Gastric) Acid Aspiration Syndrome

- Low incidence with anesthesia induction:
  - 0.00002% to 0.0003%
  - Increases to 0.001% with emergency procedures.

- In one series of post-op general surgery patients, prevalence of aspiration PNA on DC summaries was 0.8%.
Acid Aspiration Syndrome

• Severity of injury is related to pH and amount of acid aspirated.

• Volumes greater than 50 mL with pH less than 2.5 → significant and potentially life-threatening lung injury
  – More damaging with presence of particulate matter
Diagnosis

• If unwitnessed, diagnosis is difficult to prove.

• Situations in which aspiration is more likely to occur:
  – Loss of airway-protective reflexes
  – Structural and functional abnormalities of pharynx and esophagus.
  – GERD
  – Post-op acute gastric dilation
Diagnosis

• Presentation:
  – Respiratory distress
  – Hypoxia

• CXR:
  – Localized air space disease in single segment
    (most commonly the superior segment of RLL) or
    in multiple dependent areas of the lung.
Diagnosis

• Bronchoscopy
  – Can reveal particulate debris, gastric fluid, or bile.
  – May suggest diagnosis if lipid laden macrophages are detected.
Pathophysiology

• Biphasic pattern of injury
  – Direct tissue injury by acid
    • Immediate damage to ciliated respiratory epithelium
    • Destruction of surfactant
    • Damage of alveolar lining
    • Increased capillary permeability
  – Neutrophil activation and systemic inflammatory response
  – Secondary bacterial infection
Pathophysiology

• Chemical pneumonitis
  – may resolve quickly without significant sequelae
  – patient may die from respiratory failure, progression to ARDS, or multisystem organ failure within days.
Treatment

• Supportive
• Prophylactic antibiotics and steroids are NOT indicated.
• Bronchoscopy to suction out debris
• Bronchodilators for any bronchospasm (anticholinergic and adrenergic)
• Avoidance of hyperoxygenation
• Aggressive treatment of any secondary pneumonias
Bacterial Aspiration Pneumonia

- Following an aspiration event, for pneumonia to occur, there must be:
  - Breakdown of host defenses
  - Large volume of aspirate
Bacterial Aspiration Syndrome

• Causes:
  – general anesthesia
  – sedatives/analgesics
  – CNS trauma
  – structural abnormalities of larynx/pharynx/esophagus
  – RLN paralysis
  – Neuromuscular disease
  – Achalasia or Zenker’s diverticulum
  – Malignant obstruction
  – Reflux
To help reduce incidence, factors that are...

• **Efficacious**
  – Vented pts in semirecumbent position
  – Oral decontamination
  – Attention to dental hygiene

• **Not efficacious**
  – Subglottic drainage
  – Nasojejunal enteral feeding
  – Prokinetics
  – Acid suppression
  – ABX strategies
  – Monitoring of gastric residual volumes
  – Gastrostomy vs jejunostomy
  – NG vs NJ feeding tubes
Diagnosis

• 

  – Based on clinical suspicion on appropriate setting

  – CXR may show infiltrate on dependent areas of lung
    • Posterior segment of RUL and superior segment of RLL

  – To detect silent aspiration and assess function of swallowing → video fluoroscopy
Diagnosis

• Dx:

  – Use of lipid-laden macrophage index greater than 100 on BAL →
    • Sensitivity of 94%
    • Negative predictive value of 98%
    • Specificity of 89%
    • Positive predictive value of 71%... For dx of aspiration.

  – Detection of pepsin in tracheal secretions or on BAL
Treatment

• Antibiotics with documented infection

• In non-hospitalized population:
  – Coverage against Gram (+) organisms and oral anaerobes

• In hospitalized population:
  – Coverage must also be effective against Gram (-) organisms
Lung Abscess
Lung Abscess

• Localized collection of pus contained in a cavity that is formed by the destruction of lung parenchyma
  – Excludes infected bullae and cysts

• Most often solitary.

• Considered chronic if present more than six weeks.
Lung Abscess

• Classified by etiology as primary or secondary

• Primary lung abscesses:
  – Necrotizing pulmonary infections
  – Aspiration of GI contents or oropharyngeal secretions

• Secondary lung abscesses:
  – Complication of bacteremia or bronchial obstruction
  – Extension of adjacent suppurative infections
  – Infection of previously destroyed or damaged lung parenchyma
Classification of Lung Abscesses

• **Primary**
  
  – Aspiration
  – Impaired level of consciousness (e.g., general anesthesia, drug or alcohol, stroke)
  – Poor oral hygiene (gingivodental sepsis)
  – Esophageal disease (achalasia, Zenker's diverticulum, gastroesophageal reflux disease)
  – Necrotizing pneumonia
  – Virulent organisms (*Staphylococcus aureus, Klebsiella pneumoniae*, Friedländer's bacillus)
  – Fungi
  – Tuberculosis
  – Immunodeficiency
  – Immunosuppression for organ transplantation
  – Steroid therapy
  – Cancer chemotherapy
  – Diabetes
  – Malnutrition

• **Secondary**
  
  – Bronchial obstruction
  – Cavitating lesions (neoplasm, infarct)
  – Direct extension (amebiasis, subphrenic abscess)
  – Hematogenous

• **Congenital or Acquired Cysts**
  
  – Hydatid, tuberculosis, bronchogenic cyst, bullae
Lung Abscesses

• Location
  – Superior Segment of RLL
  – Posterior Segment of RUL

• Incidence of lung abscesses diminished in 1940-50s with advent of antibiotics
Microbiology

• Aspiration related:
  – Aerobic Gram (+) cocci and facultative Gram (-) bacilli
  – S. aureus, S. pyogenes, K. pneumoniae, E. Coli, and Pseudomonas species

• Necrotizing pneumonia:
  – S. pyogenes, K. pneumoniae, S. aureus, S. viridans, S. pneumoniae, H. influenzae

• Immunosuppressed host:
  – Salmonella, Legionella, PCP, atypical mycobacteria, fungi
Clinical Presentation

• Cough, fever, chills, malaise, fatigue, weight loss, pleuritic chest pain, dyspnea, hemoptysis (less common).

• PUTRID SPUTUM once cavitation takes place, with large quantities!!!
Clinical Presentation

• Sudden onset of septic shock and respiratory failure if abscess ruptures and forms pyopneumothorax.

• Mortality is 5-10% (or 9-28% if immunosuppressed).
Diagnosis

• CBC
• Sputum culture
• CXR and CT scan: cavitating lesion with air-fluid level
• Bronchoscopy to evaluate for endobronchial obstruction
  – BAL for culture information
Management

• Broad spectrum antibiotics
  • IV ABX until patient is no longer toxic
  • Oral ABX for 4-8 weeks
• Chest physiotherapy and toilet bronchoscopy (if necessary)
• Most abscesses (85-90%) respond within 2 weeks and resolve over 2-5 months.
• If no response, re-culture to determine appropriate antimicrobial therapy
Indications for Intervention

• Failure to resolve with antibiotics
• Abscess under tension
• Abscess increasing in size despite ABX
• Contralateral lung contamination
• Abscess larger than 4-6 cm diameter
• Rising fluid level
• Persistent vent dependency
• Necrotizing infection with multiple abscesses
• Hemoptysis
• Rupture into pleural space with pyopneumothorax
• Inability to exclude a cavitating carcinoma
Management

• Percutaneous drainage → TREATMENT OF CHOICE (with exception of massive hemoptysis and inability to rule out cancer)

  – Effective 73-100% of the time
Indications for Surgery

• Suspicion of, or inability to rule out, cavitating lung cancer → lobectomy
  – Lack of ABX response, absence of fever or leukocytosis, thick-walled cavity are clues

• Massive hemoptysis → lobectomy

• Ruptured abscess → tube thoracostomy
  – Thoracotomy for better drainage +/- lobectomy
On Surgery

• PROTECTION OF CONTRALATERAL LUNG IS KEY TO ANY INTERVENTION!!!

• Use double-lumen tube, bronchial blocker, or contralateral mainstem intubation to minimize contamination of the dependent lung during surgery.

• Particularly important during cases of massive hemoptysis
On Surgery

• To minimize spillage
  – Early clamping of involved bronchus
  – Minimal manipulation of involved lobe
  – Anticipation of increased vascularity and lymphadenopathy secondary to infection, making access and control of the hilum difficult
RESULTS

• Mortality 5-20%

• 75-88% of pts are cured by medical tx alone

• Surgical treatment has 90% success rate, with mortality of 1-13%.

• Percutaneous drainage success rate: 73-100%.
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