Pulmonary suppurations

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- Lung abscess
- Necrotizing pneumonia
Lung abscess

- A localized area of necrotic destruction of lung parenchyma in which infection by pyogenic organisms results in tissue necrosis & suppuration.
- It manifests radiographically as a cavity with an air–fluid levels.

Necrotizing Pneumonia

- Necrosis with multiple micro abscesses (less than 2cm in diam).
- The most severe form of necrotizing pneumonia – pulmonary gangrene.
Ethiology & Pathogenesis

- Microorganism
- Mechanism of infection
- Host defense
Microorganisms

- Caused by a wide variety of different organisms & it's common to obtain a mixed bacterial growth from single abscess when pus is cultured.

- Anaerobes – 69% of community acquired cases
- Anaerobes – 7% hospital acquired cases
Microorganisms

- Most frequently implicated
- Main groups
  - **Gram negative bacilli** – Bacteroides- Bacteroides fragilis
  - **Gram positive cocci** mainly Peptostreptococcus
  - Long & thin gram negative rods – Fusobacterium – Fusobacterium nucleatum, Fusobacterium necrophorum
Microorganisms

- Tend to cause **lung abscess** as a part of necrotizing pneumonia
- **Gram positive aerobes**:
  - Staph.aureus – pneumonia, lung abscesses, pneumatoceles
  - Staph.aureus – leading cause of lung abscess in children
  - Strep.pyogenes
  - Strep.pneumoniae serotype 3
Microorganisms

▶ **Gram negative aerobes**
  - Klebsiella pneumoniae
  - Pseudomonas aeruginosa
  - Hemophilus influenzae
  - E.coli
  - Acinetobacter
  - Proteus
  - Legionella
Microorganisms

- **Fungal infection** – Histoplasma capsulatum

- Blastomyces dermatitidis
- Coccidioides immitis
- Aspergillus
- Cryptococcus neoformans

- No *Mycobacterium tuberculosis*
Mechanisms of Infection

- Commonest cause – Aspiration of oropharyngeal contents

- 75% of the abscesses occur in posterior segment of the Rt. upper lobe or Apical segments of either lower lobe.
Mechanisms of Infection

- Aspiration of Oropharyngeal flora
  - Dental / Periodontal sepsis
  - Paranasal sinus infection
  - Depressed conscious level
  - Impaired laryngeal closure (cuffed endotracheal tube, tracheostomy tube, recurrent laryngeal nerve palsy)
  - Disturbances of swallowing
  - Delayed gastric emptying / GERD / vomiting
Mechanisms of Infection

- Hematogenous spread from a distal site
  - UTI
  - Abdominal sepsis
  - Pelvic sepsis
  - Infective endocarditis
  - IV drug abuse
  - Infected IV cannulae
  - Septic thrombophlebitis
Mechanisms of Infection

- Pre existing lung disease
  - Bronchiectasis
  - Cystic fibrosis
  - Bronchial obstruction: tumour, foreign body, cong.abn
- Infected pulmonary infarct
- Trauma
- Immunodeficiency
Host defense

- in previously healthy patient or in a patient at risk for aspiration - *primary*

- Associated with a previous lung condition or immunocompromised status - *secondary*
Clinical Features - Symptoms

- The presenting features of lung suppurations vary considerably.

1. Symptoms progress over weeks to months
2. Fever, cough, and sputum production
3. Night sweats, weight loss & anemia
4. Hemoptysis, pleurisy
Clinical Features - Signs

- There are **no specific signs** for lung abscess
- Digital clubbing – develop within a few weeks if treatment is inadequate.
- Increase/decrease vocal fremitus
- Dullness to percussion/Hyperresonance
- Diminished breath sounds if abscess is too large and situated near the surface of lung.
- Amphoric / cavernous breath sounds
Diagnosis

Imaging tests

- Chest X ray
- CT CHEST

Microbiological tests

Assessment of severity of inflammation (CBC, ESR, CRP, LDH)
2. Microbiological exam
   - Gram stain: both +ve & -ve, mixed
   - Sputum culture on standard & anaerobic culture
   - Blood culture
   - AFB, Xpert MTB/Rif, MGIT, LJ
Diagnosis

Uncontaminated specimens

- BAL
- Transtracheal aspirates (TTA)
- Transthoracic needle aspirates (TTNA)
Differential diagnosis

- Cavitating lung cancer
- Localized empyema
- Infected bulla containing a fluid level
- Infected congenital pulmonary lesions
- Pulmonary haematomata
- Cavitated pneumoconiotic lesions
- Hiatus hernia
- Hydatid cysts
- Infection with paragonimus westermani
- Cavitating pulmonary infarcts
- Wegeners granulomatosis
Treatment – antibiotic therapy

1. Penicillin or clindamycin +/- metronidazole IV – in hospitalised pts.
2. Can change – according to sensitivity
Response to treatment

- Usually show clinical improvement with ↓ fever within 3-4 days after beginning antibiotics
- Should deffervesce in 7-10 days
- Persistent fevers beyond this time indicate delayed response, and such patients should undergo further diagnostic tests to define the underlying anatomy and microbiology of the infection
Duration of treatment

- Debated
- Some advocate 4-6 weeks
- Most treat until radiographic abnormalities resolve, generally requiring months of treatment
Surgical intervention

- Surgery rarely required
- **Indications:** failure of medical management, suspected neoplasm, or hemorrhage
- **Predictors of poor response to antibiotic therapy alone:** abscesses associated with an obstructed bronchus, large abscess (>6 cm in diameter), relatively resistant organisms, such as P. aeruginosa
- The usual procedure in such cases is a **lobectomy or pneumonectomy**
1. Alternative for patients who are considered with very high operative risks is **percutaneous drainage**.

2. **Bronchoscropy** - to facilitate drainage (relatively little use)
Complications

- Empyema
- Bronchopleural –fistula
- Pneumothorax , pyoneumothorax
- Metastatic cerebral abscess
- Sepsis
- Fibrosis, bronchiectasis, amyloidosis