## Anatomic Alterations of the Lungs

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| **Lung abscess** | necrosis of lung tissue that in severe cases leads to a localized air- and fluid-filled cavity |
| | also known as "**necrotizing pneumonia**" or "**lung gangrene**" |
| | The fluid in the cavity is a collection of purulent exudate that is composed of liquefied white blood cell remains, proteins, and tissue debris. |
| *Pyogenic membrane* | encapsulates the air- and fluid-filled cavity |
| | consists of a layer of fibrin, inflammatory cells, and granulation tissue |
| Early stages | pathology is indistinguishable from that of any acute pneumonia |
| | Polymorphonuclear leukocytes and macrophages move into the infected area to engulf any invading organisms. This action causes the pulmonary capillaries to dilate, the interstitium to fill with fluid, and the alveolar epithelium to swell from the edema fluid. In response to this inflammatory reaction, the alveoli in the infected area become consolidated |
| | As the inflammatory process progresses, **tissue necrosis** involving all the lung structures occurs. |
| | In severe cases, tissue necrosis ruptures into a bronchus and allows a partial or total drainage of the liquefied contents from the cavity |
| | An air- and fluid-filled cavity also may rupture into the intrapleural space via a **bronchopleural fistula** and cause ***pleural effusion*** and ***empyema*** |
| After a period of time | **fibrosis** and **calcification** of the tissues around the cavity encapsulate the abscess |

**Major pathologic or structural changes**

## Anatomic Alterations of the Lungs (cont)

- Alveolar consolidation
- Alveolar-capillary and bronchial wall destruction
- Tissue necrosis
- Cavity formation
- Fibrosis and calcification of the lung parenchyma
- Bronchopleural fistulas and empyema
- Atelectasis
- Excessive airway secretions

## Etiology and Epidemiology

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| **Lung abscesses** | most commonly occur as a complication of **aspiration pneumonia** |
| | —i.e., the pathologic events that follow shortly after aspirating either acidic gastric fluids or a variety of anaerobic organisms that are normally found in oropharyngeal secretions |
| | **Anaerobic organisms** often colonize in the small grooves and spaces between the teeth and gums in patients with poor oral hygiene; they are frequently associated with gingivitis and dead or abscessed teeth. |

**Aspiration** often occurs in the patient with a decreased level of consciousness.

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| Predisposing factors | (1) alcohol abuse, |
| | (2) seizure disorders, |
| | (3) general anesthesia, |
| | (4) head trauma, |
| | (5) cerebrovascular accidents, and |
| | (6) swallowing disorders. |

Anatomically, lung abscesses most commonly develop in lung regions that are dependent in the recumbent position

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| e.g. | posterior segments of the upper lobes |
| | superior segments of the lower lobes |

The **right lung** is more commonly involved than the left.

## Etiology and Epidemiology (cont)

**Flash burn** aspiration of acidic gastric fluids is associated with immediate injury to the tracheobronchial tree and lung parenchyma

**A lung abscess may also develop as a result of:**

(1) **bronchial obstruction with secondary cavitating infection** (e.g., distal to bronchogenic carcinoma or an aspirated foreign body)

(2) **vascular obstruction with tissue infarction** (e.g., septic embolism, vasculitis)

(3) **interstitial lung disease with cavity formation** (e.g., pneumoconiosis [silicosis], Wegener's granulomatosis, and rheumatoid nodules)

(4) **bullae or cysts that become infected** (e.g., congenital or bronchogenic cysts)

(5) **penetrating chest wounds that lead to an infection** (e.g., bullet wound)

## Organisms Known to Cause Lung Abscess

| Common Organisms Associated with Aspiration | |
|---|---|
| Anaerobic gram-positive cocci | Peptostreptococci |
| | Peptococci |
| Anaerobic gram-negative bacilli | *Bacteroides fragilis* |
| | *Prevotella melaninogenica* |
| | *Fusobacterium* species |

**Less Common Organisms**

*Klebsiella*

*Staphylococci*

*Mycobacterium tuberculosis* (plus atypical organisms *Mycobacterium kansasii* and *Mycobacterium avium*)

*Histoplasma capsulatum*

*Coccidioides immitis*

*Blastomyces*

*Aspergillus fumigatus*

**Parasites**

*Paragonimus westermani*

## Organisms Known to Cause Lung Abscess (cont)

*Echinococcus*

*Entamoeba histolytica*

**Rare Causes**

*Streptococcus pneumoniae*

*Pseudomonas aeruginosa*

*Legionella pneumophila*

## CLINICAL DATA OBTAINED AT THE PATIENT'S BEDSIDE

**The Physical Examination**

**Vital Signs** **Increased Respiratory Rate (Tachypnea)**

- Stimulation of peripheral chemoreceptors (hypoxemia)
- Decreased lung compliance–increased ventilatory rate relationship
- Stimulation of J receptors
- Pain, anxiety, fever

**Increased Heart Rate (Pulse) and Blood Pressure**

**Pleuritic Chest Pain, Decreased Chest Expansion**

**Cyanosis**

**Cough, Sputum Production, and Hemoptysis**

Early Stages: inflammatory pneumonia-like phase; nonproductive barking or hacking cough

If the abscess progresses into an air- and fluid-filled cavity and ruptures through a bronchus, the patient may suddenly cough up large amounts of sputum.

**Foul-smelling brown** or **gray sputum** indicates a putrid infection that is caused by *numerous organisms*, including *anaerobes*.

An **odorless green or yellow sputum** indicates a nonputrid infection caused by a *single aerobic organism*.

**Blood-streaked sputum** is common in patients with a lung abscess.

Occasionally, **frank hemoptysis** is seen

**Chest Assessment Findings**

Increased tactile and vocal fremitus

Crackles

The following may be heard directly over the abscess:

Dull percussion note

## CLINICAL DATA OBTAINED AT THE PATIENT'S BEDSIDE (cont)

Bronchial breath sounds

Diminished breath sounds

Whispered pectoriloquy

Pleural friction rub (if abscess is near pleural surface)

## Clinical Data from Lab Tests

**Pulmonary Function Test Findings**
**Severe and Extensive Cases (Restrictive Lung Pathophysiology)**

FORCED EXPIRATORY VOLUME AND FLOW RATE FINDINGS*

| FVC | $FEV_T$ | $FEV_1/FVC$ ratio | $FEF_{25\%-75\%}$ |
|---|---|---|---|
| ↓ | N or ↓ | N or ↑ | N or ↓ |

| $FEF_{50\%}$ | $FEF_{200-1200}$ | PEFR | MVV |
|---|---|---|---|
| N or ↓ | N or ↓ | N or ↓ | N or ↓ |

LUNG VOLUME AND CAPACITY FINDINGS

| $V_T$ | IRV | ERV | RV | |
|---|---|---|---|---|
| N or ↓ | ↓ | ↓ | ↓ | |
| VC | IC | FRC | TLC | RV/TLC ratio |
| ↓ | ↓ | ↓ | ↓ | N |

**Arterial Blood Gases**

MILD TO MODERATE LUNG ABSCESS
**Acute Alveolar Hyperventilation with Hypoxemia†**
**(acute respiratory alkalosis)**

| pH | $PaCO_2$ | $HCO_3^-$ | $PaO_2$ | $SaO_2$ or $SpO_2$ |
|---|---|---|---|---|
| ↑ | ↓ | ↓ | ↓ | ↓ |

(but normal)

SEVERE LUNG ABSCESS
**Acute Ventilatory Failure with Hypoxemia†**
**(acute respiratory acidosis)**

| pH§ | $PaCO_2$ | $HCO_3^-$ § | $PaO_2$ | $SaO_2$ or $SpO_2$ |
|---|---|---|---|---|
| ↓ | ↑ | ↑ | ↓ | ↓ |

(but normal)

**Oxygenation Indices‖**

| $\dot{Q}_S/\dot{Q}_T$ | $Do_2$¶ | $\dot{V}O_2$ | $C(a-\bar{v})O_2$ | $O_2ER$ | $S\bar{v}O_2$ |
|---|---|---|---|---|---|
| ↑ | ↓ | N | N | ↑ | ↓ |

## ABNORMAL LAB TEST AND PROCEDURE RESULTS

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| **Sputum Examination** | Many of these organisms are "slow growers" and may take some time to be identified completely on culture media. |
| Anaerobic Organisms | |
| Anaerobic Gram-Positive Cocci | |
| | Peptostreptococci |
| | Peptococci |
| Anaerobic Gram-Negative Bacilli | |
| | *Bacteroides fragilis* |
| | *Prevotella melaninogenica* |
| | *Fusobacterium species* |

## RADIOLOGIC FINDINGS

**Chest Radiograph**

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|---|---|
| | Increased opacity |
| | Cavity formation |
| | Cavities with air-fluid levels |
| | Fibrosis and calcification |
| | Pleural effusion |
| Early Stages | reveals localized consolidation |

Later, characteristic radiographic appearance of a lung abscess appears after:

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|---|---|
| | (1) the infection ruptures into a bronchus, and/or |
| | (2) tissue destruction and necrosis have occurred, and/or |
| | (3) partial evacuation of the purulent contents has occurred |

The abscess usually appears on the radiograph as a **circular radiolucency** that contains an air-fluid level, surrounded by a dense wall of lung parenchyma.

## General Management of Lung Abscess

**Medications and Procedures Commonly Prescribed by the Physician**

Treatment varies based on the severity of the pneumonia and the lung abscess.

Treatment includes appropriate (usually intravenous) **antimicrobial therapy** coupled with **prompt drainage** and **surgical debridement**.

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| ***Clindamycin*** | standard treatment for a lung abscess caused by an anaerobic pathogen |
| Other drugs that may be used are any combination of: | |
| | ***beta-lactam–beta-lactamase inhibitors*** (e.g., ampicillin-sulbactam) |
| | ***penicillin*** plus ***metronidazole*** |
| | ***carbapenem*** |
| ***Linezolid*** | When the lung abscess is caused by methicillin-resistant Staphylococcus aureus (MRSA) |
| Alternative to linezolid | *vancomycin* |
| | *ceftaroline*, *trimethoprim-sulfamethoxazole*, and *telavancin*. |

**Respiratory Care Treatment Protocols**

## General Management of Lung Abscess (cont)

**Oxygen Therapy Protocol**

treat hypoxemia, decrease the work of breathing, and decrease myocardial work

Hypoxemia caused by capillary shunting is often refractory to oxygen therapy.

**Bronchopulmonary Hygiene Therapy Protocol**

e excessive production and accumulation of mucus associated with a ruptured lung abscess

**Lung Expansion Therapy Protocol**

alveolar consolidation and atelectasis

When treated properly, most patients with a lung abscess show improvement. In acute cases, the size of the abscess quickly decreases and eventually closes altogether. In severe or chronic cases, the patient’s improvement may be slow or insignificant, even with appropriate therapy.