

Anatomic Alterations of the Lungs

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

Lung abscesses	most commonly occur as a complication of aspiration pneumonia
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—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.

Predisposing factors	(1) alcohol abuse,
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(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

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	<i>Bacteroides fragilis</i>
	<i>Prevotella melaninogenica</i>
	<i>Fusobacterium</i> species

Less Common Organisms

<i>Klebsiella</i>
<i>Staphylococci</i>
<i>Mycobacterium tuberculosis</i> (plus atypical organisms <i>Mycobacterium kansasii</i> and <i>Mycobacterium avium</i>)
<i>Histoplasma capsulatum</i>
<i>Coccidioides immitis</i>
<i>Blastomyces</i>
<i>Aspergillus fumigatus</i>

Parasites

<i>Paragonimus westermani</i>

Organisms Known to Cause Lung Abscess (cont)

<i>Echinococcus</i>
<i>Entamoeba histolytica</i>
Rare Causes
<i>Streptococcus pneumoniae</i>
<i>Pseudomonas aeruginosa</i>
<i>Legionella pneumophila</i>

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 ₁	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				
Q _s /Q _t	Do ₂	VO ₂	C(a-v)O ₂	O ₂ ER
↑	↓	N	N	↑

ABNORMAL LAB TEST AND PROCEDURE RESULTS

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

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:

- (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**.

Clinda-mycin 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

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.



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