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**
	—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,
	(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	*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

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**.

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.

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