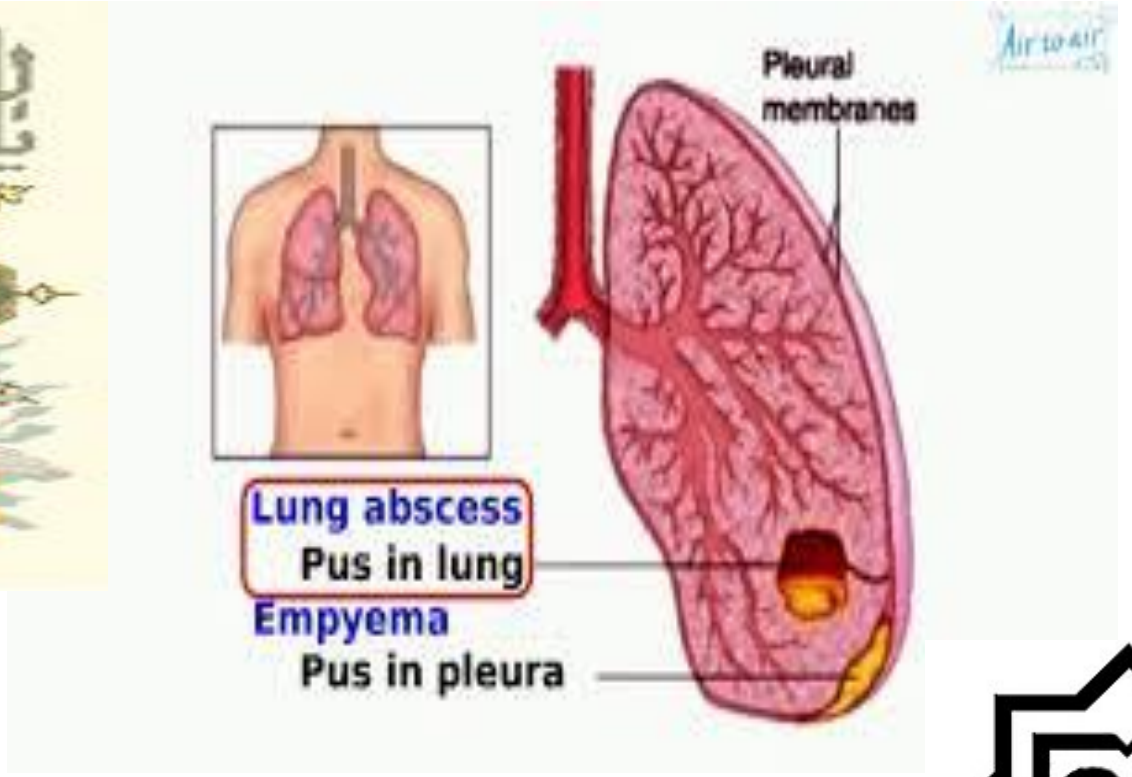


Lung Abscess

DR.MOHSEN SHAFIEPOUR PULMONOLOGIST

دانشگاه علوم پزشکی و خدمات
بهداشتی درمانی کرمان

Lung abscess represents necrosis and cavitation of the lung following microbial infection. Lung abscesses can be single or multiple but usually are marked by a single dominant cavity >2 cm in diameter

ETIOLOGY

The low prevalence of lung abscesses makes them difficult to study in randomized controlled trials. Although the incidence of lung abscesses has decreased in the antibiotic era, they are still a source of significant morbidity and mortality.

Lung abscesses usually are characterized as either primary (~80% of cases) or secondary.

Primary lung abscesses generally arise from aspiration, often are caused principally by anaerobic bacteria, and occur in the absence of an underlying pulmonary or systemic condition.

Secondary lung abscesses arise in the setting of an underlying condition, such as a postobstructive process (e.g., a bronchial foreign body or tumor) or a systemic process (e.g., HIV infection or another immunocompromising condition). Lung abscesses can also be characterized as acute (<4–6 weeks in duration) or chronic (~40%)

EPIDEMIOLOGY

In general, middle-aged men are more commonly affected than middle-aged women.

The major risk factor for primary lung abscesses is aspiration. Patients at particular risk for aspiration, such as those with altered mental status, alcoholism, drug overdose, seizures, bulbar dysfunction, prior cerebrovascular or cardiovascular events, or neuromuscular disease, are most commonly affected. At additional risk are patients with esophageal dysmotility or esophageal lesions (strictures or tumors) and those with gastric distention and/or gastroesophageal reflux, especially those who spend substantial time in the recumbent position

PATHOGENESIS

Primary Lung Abscesses The development of primary lung abscesses is thought to originate when chiefly anaerobic bacteria (as well as microaerophilic streptococci) in the gingival crevices are aspirated into the lung parenchyma in a susceptible host

Pneumonitis develops initially (exacerbated in part by tissue damage caused by gastric acid); then, over a period of 7–14 days, the anaerobic bacteria produce parenchymal necrosis and cavitation whose extent depends on host–pathogen interaction


PATHOPHYSIOLOGY
Gastric content aspiration/entry bacteria into the lung
Inflammatory response
Cavity extend to bronchus
Promotion of abscess into encapsulated
Tissue necrosis
Increased production of sputum
Sputum discharge

Secondary Lung Abscesses

The pathogenesis of secondary abscesses depends on the predisposing factor. For example, in cases of bronchial obstruction from malignancy or a foreign body, the obstructing lesion prevents clearance of oropharyngeal secretions, leading to abscess development. With underlying systemic conditions (e.g., immunosuppression after bone marrow or solid organ transplantation), impaired host defense mechanisms lead to increased susceptibility to the development of lung abscesses caused by a broad range of pathogens, including opportunistic organisms

TABLE 127-1 Examples of Microbial Pathogens That can Cause Lung Abscesses

CLINICAL CONDITION	PATHOGENS
Primary lung abscess (usually with risk factors for aspiration)	Anaerobes (e.g., *Peptostreptococcus* spp., *Prevotella* spp., *Bacteroides* spp., *Streptococcus milleri*), microaerophilic streptococci
Secondary lung abscess (often with underlying immunocompromise)	*Staphylococcus aureus*, gram-negative rods (e.g., *Pseudomonas aeruginosa*, Enterobacteriaceae), *Nocardia* spp., *Aspergillus* spp., Mucorales, *Cryptococcus* spp., *Legionella* spp., *Rhodococcus equi*, *Pneumocystis jirovecii*
Embolic lesions	*Staphylococcus aureus* (often from endocarditis), *Fusobacterium necrophorum* (Lemierre's syndrome; see text for details)
Endemic infections (with or without underlying immunocompromise)	*Mycobacterium tuberculosis* (as well as *Mycobacterium avium* and *Mycobacterium kansasii*), *Coccidioides* spp., *Histoplasma capsulatum*, *Blastomyces* spp., parasites (e.g., *Entamoeba histolytica*, *Paragonimus westermani*, *Strongyloides stercoralis*)
Miscellaneous conditions	Bacterial pathogen (often *S. aureus*) after influenza or another viral infection, *Actinomyces* spp.

PATHOLOGY AND MICROBIOLOGY

Primary Lung Abscesses The dependent segments (posterior upper lobes and superior lower lobes) are the most common locations of primary lung abscesses, given the predisposition of aspirated materials to be deposited in these areas. Generally, the right lung is affected more commonly than the left because the right mainstem bronchus is less angulated.

Because it is not clear that knowing the identity of the causative anaerobic isolate alters the response to treatment of a primary lung abscess, practice has shifted away from the use of specialized techniques to obtain material for culture, such as transtracheal aspiration and bronchoalveolar lavage with protected brush specimens that allow recovery of culture material while avoiding contamination from the oral cavity. When no pathogen is isolated from a primary lung abscess (which occurs as often as 40% of the time), the abscess is termed a nonspecific lung abscess, and the presence of anaerobes is often presumed. A putrid lung abscess refers to cases with foul-smelling breath, sputum, or empyema; these manifestations are essentially diagnostic of an anaerobic lung abscess

Secondary Lung Abscesses The location of secondary abscesses may vary with the underlying cause. The microbiology of secondary lung abscesses can encompass a broad bacterial spectrum, with infection by Pseudomonas aeruginosa and other gram-negative rods the most common

CLINICAL MANIFESTATIONS

Clinical manifestations initially may be similar to those of pneumonia, with fevers, cough, sputum production, and chest pain; a more chronic and indolent presentation that includes night sweats, fatigue, and anemia is often observed with anaerobic lung abscesses. A subset of patients with putrid lung abscesses may report discolored phlegm and foul-tasting or foul-smelling sputum. Patients with lung abscesses due to non-anaerobic organisms, such as S. aureus, may present with a more fulminant course characterized by high fevers and rapid progression.

Findings on physical examination may include fevers, poor dentition, and/or gingival disease as well as amphoric and/or cavernous breath sounds on lung auscultation. Additional findings may include digital clubbing and the absence of a gag reflex.


Signs of a Lung Abscess
Early Signs
Fever and chills
Night sweats
Persistent cough
Later Signs
Pain with deep breathing
Coughing up blood
Fatigue

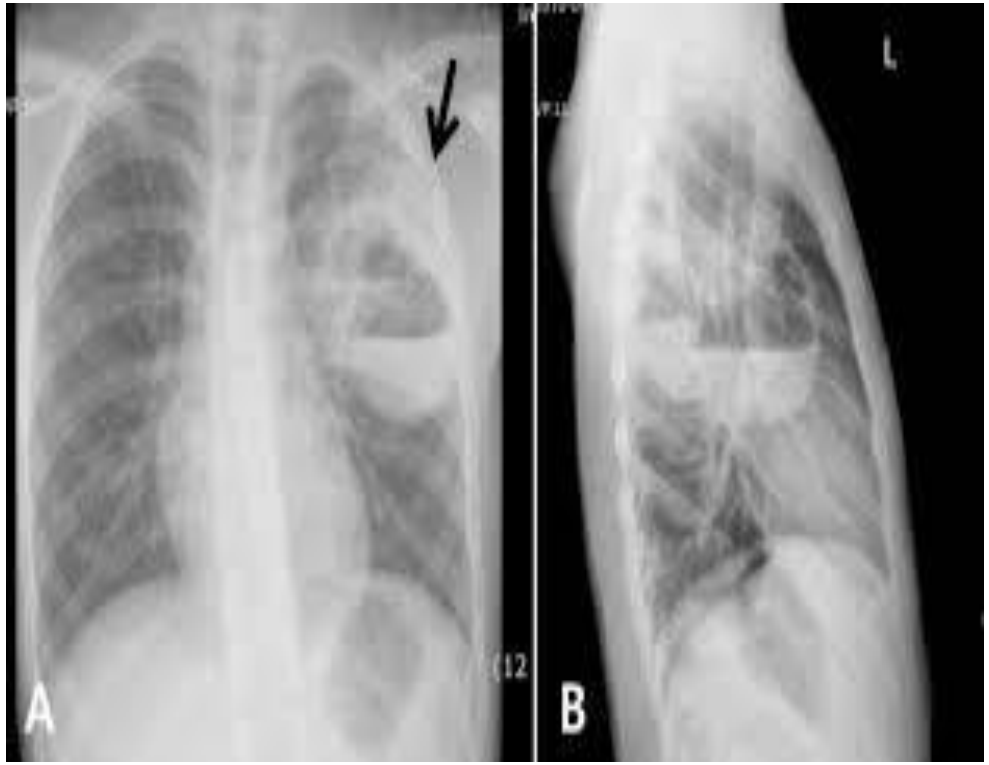
DIFFERENTIAL DIAGNOSIS

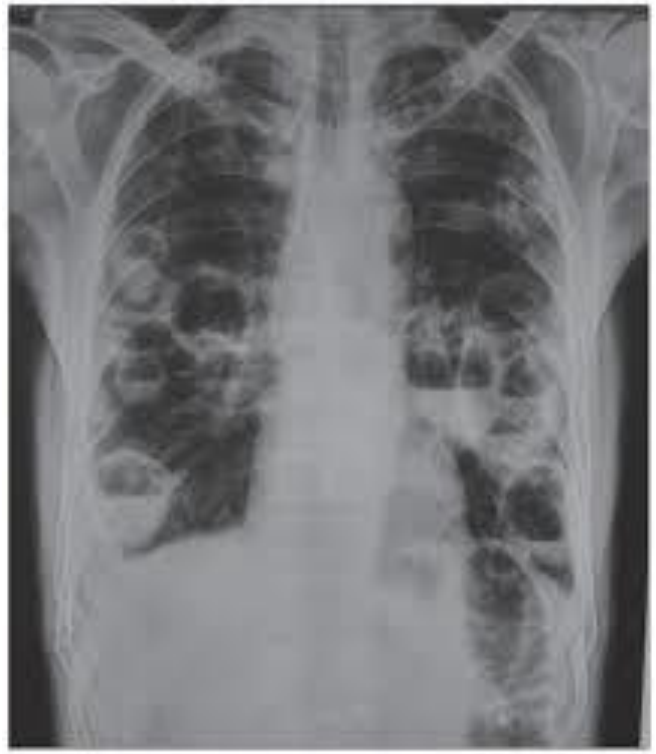
lung infarction,
malignancy,
sequestration,
cryptogenic organizing pneumonia,
sarcoidosis,
Vasculitides
autoimmune diseases (e.g., granulomatosis with polyangiitis),
lung cysts or bullae containing fluid, and septic emboli (e.g., from tricuspid valve endocarditis). Other less common entities can include pulmonary manifestations of diseases that usually present at locations other than the chest (e.g., inflammatory bowel disease, pyoderma gangrenosum).

DIAGNOSIS

CXR

CT scan





many physicians consider putrid-smelling sputum to be virtually diagnostic of an anaerobic infection

When a secondary lung abscess is present or empirical therapy fails to elicit a response, sputum and blood cultures are advised in addition to serologic studies for opportunistic pathogens (e.g., viruses and fungi causing infections in immunocompromised hosts).

Bronchoscopy

CT-guided needle aspiration

TREATMENT Lung Abscess

For primary lung abscesses, the recommended regimens are
(1) clindamycin (600 mg IV three times daily; then, with the disappearance of fever and clinical improvement, 300 mg PO four times daily) or
(2) an IV-administered β-lactam/β-lactamase combination, followed—once the patient's condition is stable—by orally administered amoxicillin-clavulanate. This therapy should be continued until imaging demonstrates that the lung abscess has cleared or regressed to a small scar. Treatment duration may range from 3–4 weeks to as long as 14 weeks. One small study suggested that moxifloxacin (400 mg/d PO) is as effective and well tolerated as ampicillin-sulbactam. Notably, metronidazole is not effective as a single agent: it covers anaerobic organisms but not the microaerophilic streptococci that are often components of the mixed flora of primary lung abscesses.

In secondary lung abscesses, antibiotic coverage should be directed at the identified pathogen, and a prolonged course (until resolution of the abscess is documented) is often required. Treatment regimens and courses vary widely, depending on the immune state of the host and the identified pathogen. Other interventions may be necessary as well, such as relief of an obstructing lesion or treatment directed at the underlying condition predisposing the patient to lung abscess. Similarly, if the condition of patients with presumed primary lung abscess fails to improve, additional studies to rule out an underlying predisposing cause for a secondary lung abscess are indicated.

Although it can take as long as 7 days for patients receiving appropriate therapy to defervesce, as many as 10–20% of patients may not respond at all, with continued fevers and progression of the abscess cavity on imaging.

An abscess >6–8 cm in diameter is less likely to respond to antibiotic therapy without additional interventions. Options for patients who do not respond to antibiotics and whose additional diagnostic studies fail to identify an additional pathogen that can be treated include surgical resection and percutaneous drainage of the abscess, especially when the patient is a poor surgical candidate. Timing of surgical intervention can be challenging; the goal is to balance the morbidity/mortality risk of a procedure with the need for definitively clearing the abscess in the setting of persistent infection that is not responsive to nonsurgical approaches. Possible complications of percutaneous drainage include bacterial contamination of the pleural space as well as pneumothorax and hemothorax

COMPLICATIONS

Larger cavity size on presentation may correlate with the development of persistent cystic changes (pneumatoceles) or bronchiectasis. Additional possible complications include recurrence of abscesses despite appropriate therapy, extension to the pleural space with development of empyema, life-threatening hemoptysis, and massive aspiration of lung abscess contents

PROGNOSIS AND PREVENTION Reported mortality rates for primary abscesses have been as low as 2%, while rates for secondary abscesses are generally higher—as high as 75% in some case series. Other poor prognostic factors include age >60, the presence of aerobic bacteria, sepsis at presentation, symptom duration of >8 weeks, and abscess size >6 cm. Mitigation of underlying risk factors may be the best approach to prevention of lung abscesses, with attention directed toward airway protection, oral hygiene, and minimized sedation with elevation of the head of the bed for patients at risk for aspiration. Prophylaxis against certain pathogens in at-risk patients (e.g., recipients of bone marrow or solid organ transplants or patients whose immune systems are significantly compromised by HIV infection) may be undertaken

APPROACH TO THE PATIENT Lung Abscess For patients with a lung abscess and a low likelihood of malignancy (e.g., smokers <45 years old) and with risk factors for aspiration, it is reasonable to administer empirical treatment and then to pursue further evaluation if therapy does not elicit a response. However, some clinicians may opt for up-front cultures, even in primary lung abscesses. In patients with risk factors for malignancy or other underlying conditions (especially immunocompromised hosts) or with an atypical presentation, earlier diagnostics should be considered, such as bronchoscopy with biopsy or CT-guided needle aspiration. Bronchoscopy should be performed early in patients whose history, symptoms, or imaging findings are consistent with possible bronchial obstruction. In patients from areas endemic for tuberculosis or patients with other risk factors for tuberculosis (e.g., underlying HIV infection), induced sputum samples should be examined early in the workup to rule out this disease