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Lung Abscess 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. **PART 5 Infectious Diseases ■ ■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., bronchial foreign body, tumor) or a systemic process (e.g., HIV infection, another immunocompromising condition). Lung abscesses can also be characterized as acute (<4-6 weeks in duration) or chronic (~40% of cases). **■ ■EPIDEMIOLOGY** The majority of the existing epidemiologic information involves primary lung abscesses. 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. It is widely thought that colonization of the gingival crevices by anaerobic bacteria or microaerophilic streptococci (especially in patients with gingivitis and periodontal disease), combined with a risk of aspiration, is important in the development of lung abscesses. In fact, many physicians consider it extremely rare for lung abscesses to develop in the absence of teeth as a nidus for bacterial colonization. The importance of these risk factors in the development

of lung abscesses is highlighted by a significant reduction in abscess incidence in the late 1940s that coincided with a change in oral surgical technique: beginning at that time, these operations were no longer performed with the patient in the seated position without a cuffed endotracheal tube, and the frequency of perioperative aspiration events was thus decreased. In addition, the introduction of penicillin around

TABLE 132-1 Examples of Microbial Pathogens That Can Cause Lung Abscesses

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

the same time significantly reduced the incidence of and mortality rate from lung abscess.

■ ■ 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 (Table 132-1). Patients who develop primary lung abscesses usually carry an overwhelming burden of aspirated material or are unable to clear the bacterial load. Pneumonitis develops initially (exacerbated in part by tissue damage caused by gastric acid); then, over a period of 7–14 days, the bacteria produce parenchymal necrosis and cavitation whose extent depends on host–pathogen interaction (Fig. 132-1). Anaerobes are thought to produce more extensive tissue necrosis in polymicrobial infections in which virulence factors of the various bacteria can act synergistically to cause more significant tissue destruction.

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

FIGURE 132-1 Representative chest CT scans demonstrating development of lung abscesses. This patient was immunocompromised from underlying lymphoma and developed severe *Pseudomonas aeruginosa* pneumonia, as represented by a left lung infiltrate with concern for central regions of necrosis (panel A, black arrow). Two weeks later, areas of cavitation with air-fluid levels were visible in this region and were consistent with the development of lung abscesses (panel B, white arrow). (Images provided by Dr. Ritu Gill, formerly of the Division of Chest Radiology, Brigham and Women's Hospital, Boston; with permission.)

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 132-1). Lung abscesses also arise from septic emboli, either in tricuspid valve endocarditis (often involving *Staphylococcus aureus*) or in Lemierre's syndrome, in which an infection begins in the pharynx (classically involving *Fusobacterium necrophorum*) and

then spreads to the neck and the carotid sheath (which contains the jugular vein) to cause septic thrombophlebitis. ■ ■ **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. Primary lung abscesses often are polymicrobial, primarily including anaerobic organisms as well as microaerophilic streptococci (Table 132-1). The retrieval and culture of anaerobes can be complicated by the contamination of samples with microbes from the oral cavity, the need for expeditious transport of the cultures to the laboratory, the need for early plating with special culture techniques, the prolonged time required for culture growth, and the need for collection of specimens prior to administration of antibiotics. When attention is paid to these factors, rates of recovery of specific isolates are reportedly as high as 78%. Because it is not clear that knowing the identity of the causative anaerobic isolate alters the approach 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. In addition, a broad array of pathogens can be identified in patients from certain endemic areas and in specific clinical scenarios (e.g., a significant incidence of fungal infections among immunosuppressed patients following bone marrow or solid organ transplantation). Because immunocompromised hosts and patients without the classic presentation of a primary lung abscess can be infected with a wide array of unusual organisms (Table 132-1), it is of special importance to obtain culture material to target therapy. ■ ■ **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.

■ ■ **DIFFERENTIAL DIAGNOSIS** The differential diagnosis of lung abscesses is broad and includes other noninfectious processes that result in cavitary lung lesions, including lung infarction, malignancy, sequestration, cryptogenic organizing pneumonia, sarcoidosis, vasculitides and 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** Lung abscesses are documented by chest imaging. Although a chest radiograph usually detects a thick-walled cavity with an air-fluid level, CT permits better definition and may provide earlier evidence of cavitation. CT may also yield additional information regarding a possible underlying cause of lung abscess, such as malignancy, and may help distinguish a peripheral lung abscess from a pleural infection. This distinction has important implications for treatment because a pleural space infection, such as an empyema, may require urgent drainage. As described earlier (see “Pathology and Microbiology,” above), more invasive diagnostics (such as transtracheal aspiration) were traditionally undertaken for primary lung abscesses, whereas empirical therapy that includes drugs targeting anaerobic organisms currently is used more often. While sputum can be collected noninvasively for Gram’s stain and culture, which may yield a pathogen, the infection is likely to be polymicrobial, and culture results may not reflect the presence of anaerobic organisms. Increasing use of molecular techniques for bacterial detection (e.g., 16S RNA gene amplification) may eventually yield more specific pathogen identification. As stated above, many physicians consider putrid-smelling sputum to be virtually diagnostic of an anaerobic infection.

CHAPTER 132 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). Additional diagnostics, such as bronchoscopy with bronchoalveolar lavage or protected brush specimen collection and CT-guided percutaneous needle aspiration, can be undertaken. Risks posed by these more invasive diagnostics include spillage of abscess contents into the other lung (with bronchoscopy) and pneumothorax and bronchopleural fistula development (especially with CT-guided needle aspiration). However, early diagnostics in secondary abscesses, especially in immunocompromised hosts, are particularly important because the patients involved may be especially fragile, at risk for infection with a broad array of pathogens, and therefore less likely than other patients to respond to empirical therapy.

Lung Abscess TREATMENT Lung Abscess The availability of antibiotics in the 1940s and 1950s established therapy with this drug class as the primary approach to the treatment of lung abscess. Previously, surgery had been relied upon much more frequently. For many decades, penicillin was the antibiotic of choice for primary lung abscesses in light of its anaerobic coverage; however, because oral anaerobes can produce β -lactamases, clindamycin has proved superior to penicillin in clinical trials. 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, with some literature suggesting that a course of at least 6 weeks may be associated with better outcomes. One small

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