Chapter 65: Pneumonia and Pulmonary Infiltrates

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PNEUMONIA

EPIDEMIOLOGY

Pneumonia is an infection of the alveoli (the gas-exchanging portion of the lung) emanating from different pathogens, notably bacteria and viruses, but also fungi. Community-acquired pneumonia occurs in 4 million people and results in 1 million hospitalizations per year in the United States.[1,2] Pneumonia is the eighth leading cause of death, particularly among older adults,[3] and is the most common trigger for sepsis. Those who develop hospital- or other healthcare-associated pneumonia (acquired after placement in a care facility) often have infection from resistant organisms (**Table 65-1**).[4,5]

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TABLE 65-1

Acquisition Environment Classification for Pneumonia

Classification	Criteria
Community-acquired pneumonia	Acute pulmonary infection in a patient who is not hospitalized or residing in a long-term care facility 14 or more days before presentation
Hospital-acquired pneumonia	New infection occurring 48 or more hours after hospital admission
Ventilator-acquired pneumonia	New infection occurring 48 or more hours after starting mechanical ventilation
Healthcare-associated pneumonia	Patients hospitalized for 2 or more days within the past 90 days
	Nursing home/long-term care residents
	Patients receiving home IV antibiotic therapy
	Dialysis patients
	Patients receiving chronic wound care
	Patients receiving chemotherapy
	Immunocompromised patients

PATHOPHYSIOLOGY

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Pathogenic lung organisms are usually aspirated, especially in the hospital or healthcare setting (where eating is often not done sitting upright for dubious reasons), although inhalation is another potential route. Staphylococcus aureus and Streptococcus pneumoniae can produce pneumonia from hematogenous seeding. Patients most at risk for pneumonia are those with a predisposition to aspiration, impaired mucociliary clearance, or risk of bacteremia (**Table 65-2**).

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TABLE 65-2

Risk Factors for Pneumonia

Aspiration risk Swallowing and esophageal motility disorders Stroke Nasogastric tube Intubation Seizure and syncope
Bacteremia risk Indwelling vascular devices Intrathoracic devices (e.g., chest tube)
Debilitation Alcoholism Extremes of age Neoplasia Immunosuppression
Chronic diseases Diabetes Renal failure Liver failure Valvular heart disease Congestive heart failure

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Chronic obstructive pulmonary disease Chest wall disorders Skeletal muscle disorders Bronchial obstruction
Bronchoscopy
Viral lung infections

Some forms of pneumonia produce an intense inflammatory response within the alveoli that leads to filling of the air space with exudate and white blood cells. Bacterial pneumonia results in an intense inflammatory response and tends to cause a productive cough. Atypical organisms often trigger a less intense inflammatory response and create a milder or nonproductive cough.

In about half of patients with community-acquired pneumonia, no specific pathogen is identified. When an organism is identified, the pneumococcus is still the most common, followed by viruses and the atypical agents Mycoplasma, Chlamydia, and Legionella. Most patients with severe community-acquired pneumonia who were otherwise healthy have S. pneumoniae and Legionella as pathogens.

In up to 5% of cases, more than one pathogen exists. In nursing home residents, alcoholics, and those with human immunodeficiency virus (HIV) infection and depressed CD4 counts, all the common pathogens exist along with others rarely seen in other patients.

CLINICAL FEATURES

Patients with pneumonia frequently will present with cough (79% to 91%), fatigue (90%), fever (71% to 75%), dyspnea (67% to 75%), sputum production (60% to 65%), and pleuritic chest pain (39% to 49%).[6] Despite described patterns of presentation, the variability in the individual symptoms and physical findings can make clinical diagnosis and differentiation from bronchitis and other upper respiratory tract disease difficult.[7,8] Many types of pneumonia do not have a sudden and characteristic presentation, and many patients with pneumonia have an antecedent viral upper respiratory infection with coryza, low-grade fever, rhinorrhea, or nonproductive cough. Weight loss, malaise, dizziness, and weakness may be associated with pneumonia. Some of the atypical agents are associated with headache or GI illness. Occasionally, pneumonia is associated with extrapulmonary symptoms, including joint pain, hematuria, or skin r[illegible]ogen and clinical feature correlations, all of which can vary or be absent in an individual patient.

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TABLE 65-3

Clinical Characteristics of Common Bacterial Pneumonias

Organism	Symptoms	Sputum	Chest X-Ray
Streptococcus pneumoniae	Sudden onset, fever, rigors, pleuritic chest pain, productive cough, dyspnea	Rust-colored; gram-positive encapsulated diplococci	Lobar infiltrate, occasionally patchy, occasional pleural effusion
Staphylococcus aureus	Gradual onset of productive cough, fever, dyspnea, especially just after viral illness	Purulent; gram-positive cocci in clusters	Patchy, multilobar infiltrate; empyema, lung abscess
Klebsiella pneumoniae	Sudden onset, rigors, dyspnea, chest pain, bloody sputum; especially in alcoholics or nursing home patients	Brown "currant jelly"; thick, short, plump, gram-negative, encapsulated, paired coccobacilli	Upper lobe infiltrate, bulging fissure sign, abscess formation
Pseudomonas aeruginosa	Recently hospitalized, debilitated, or immunocompromised patient with fever, dyspnea, cough	Gram-negative coccobacilli	Patchy infiltrate with frequent abscess formation
Haemophilus influenzae	Gradual onset, fever, dyspnea, pleuritic chest pain; especially in elderly and COPD patients	Short, tiny, gram-negative encapsulated coccobacilli	Patchy, frequently basilar infiltrate, occasional pleural effusion
Legionella pneumophila	Fever, chills, headache, malaise, dry cough, dyspnea, anorexia, diarrhea, nausea, vomiting	Few neutrophils and no predominant bacterial species	Multiple patchy nonsegmented infiltrates, progresses to consolidation, occasional cavitation and pleural effusion

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Organism	Symptoms	Sputum	Chest X-Ray
Moraxella catarrhalis	Indolent course of cough, fever, sputum, and chest pain; more common in COPD patients	Gram-negative diplococci found in sputum	Diffuse infiltrates
Chlamydophila pneumoniae	Gradual onset, fever, dry cough, wheezing, occasionally sinus symptoms	Few neutrophils, organisms not visible	Patchy subsegmental infiltrates
Mycoplasma pneumoniae	Upper and lower respiratory tract symptoms, nonproductive cough, headache, malaise, fever	Few neutrophils, organisms not visible	Interstitial infiltrates, (reticulonodular pattern), patchy densities, occasional consolidation
Anaerobic organisms	Gradual onset, putrid sputum, especially in alcoholics	Purulent; multiple neutrophils and mixed organisms	Consolidation of dependent portion of lung; abscess formation

Abbreviation: COPD = chronic obstructive pulmonary disease.

The physical examination in a patient with acute pneumonia may show evidence of alveolar fluid (rales), consolidation (bronchial breath sounds), pleural effusion (dullness and decreased breath sounds), or bronchial congestion (rhonchi and wheezing).[7,8] Radiologic findings in pneumonia sometimes provide a specific pathogenic diagnosis (Table 65-3).

Pneumococcal Pneumonia

The elderly, children <2 years old, minorities, children who attend group day care centers, and those with immune-depressing comorbid conditions (e.g., previous splenectomy, transplantation, HIV infection, sickle cell disease) are at highest risk for pneumococcal pneumonia. Classically, patients with pneumococcal pneumonia present with sudden onset of disease with rigors, bloody sputum, high fever, and chest pain with lobar infiltrates (**Figure 65-1**); 25% will have parapneumonic pleural effusions. Patients with chronic lung disease, nursing home patients, or otherwise healthy elderly patients tend to have a slower progression of pneumonia, with symptoms of r

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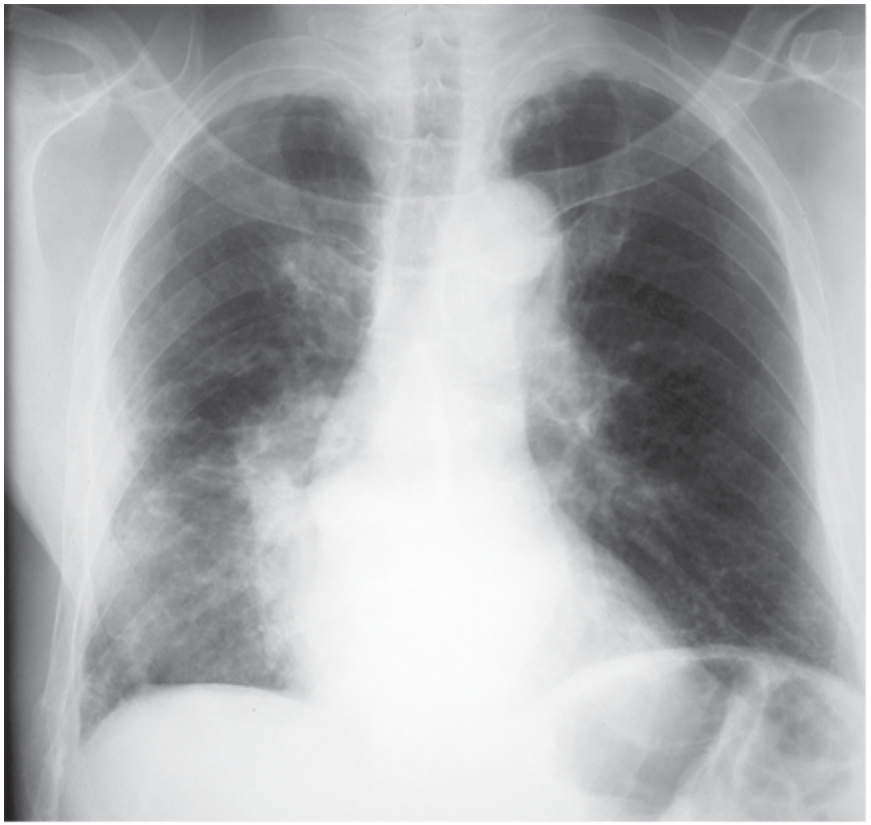
production.

FIGURE 65-1.
Lobar pneumonia.



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Laboratory findings in pneumonia include leukocytosis, elevation of the serum bilirubin or hepatic enzymes, and hyponatremia; none is diagnostic of the infection, but all detail the other organ involvement that can occur.

Pneumococcal pneumonia responds to a variety of antibiotics, although there is an increased incidence of penicillin-, macrolide-, and fluoroquinolone-resistant pneumococci.[9] Penicillin resistance ranges from 5% to 80%, depending on location, with increasing resistance reported in Spain, Italy, and Eastern Europe. Resistance is also increasing to tetracycline and trimethoprim-sulfamethoxazole. Patients with intermediate penicillin-resistant pneumococci may still be effectively treated with routine antibiotics so long as an adequate dose is administered.[10] However, the bacteriologic agent is rarely known to clinicians at the start of therapy.

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Patients with highly penicillin-resistant pneumococci require treatment with vancomycin, imipenem, a newer respiratory fluoroquinolone, or ketolide.

Other Bacterial Pneumonias

S. aureus pneumonia is a consideration in patients with chronic lung disease, patients with laryngeal cancer, immunosuppressed patients, nursing home patients, or others at risk for aspiration pneumonia. S. aureus pneumonia may occur in otherwise healthy patients after viral illness, such as during an influenza epidemic, although pneumococcal pneumonia is still more common. Patients with staphylococcal pneumonia typically have an insidious onset of disease with low-grade fever, sputum production, and dyspnea. The chest radiograph usually demonstrates extensive disease with empyema, pleural effusions, and multiple areas of infiltrate (**Figure 65-2**). If leucocidin excretion by the organism accompanies this infection (rare and not predictable), rapid progression and death are common even with adequate therapy. Patients with healthcare-acquired pneumonia are at risk for infection with methicillin-resistant S. aureus.[5,11]

FIGURE 65-2.
Staphylococcal pneumonia with extensive infiltration and effusion or empyema.

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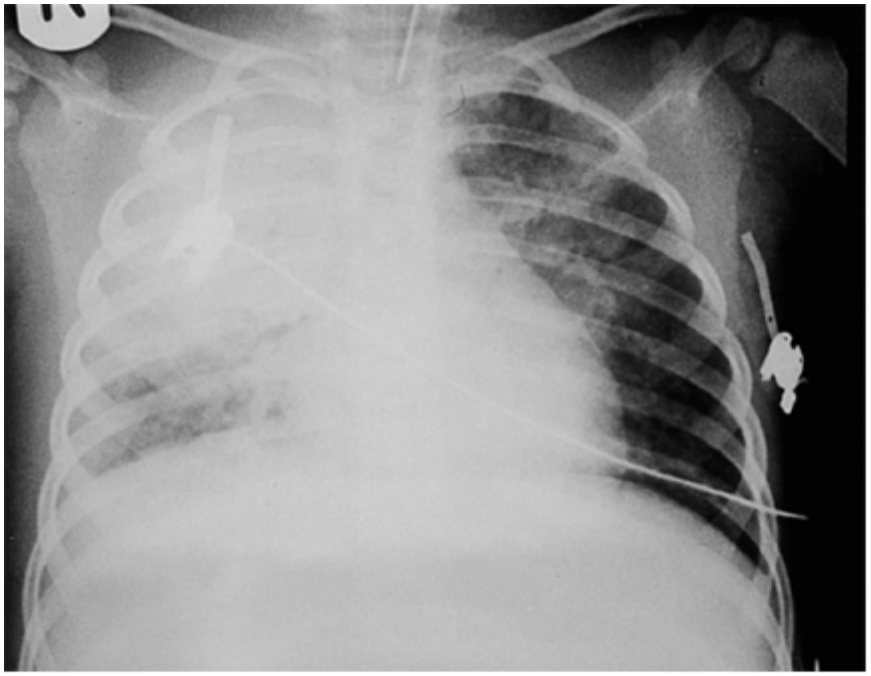

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Klebsiella pneumonia often occurs in compromised patients: those at risk of aspiration, alcoholics, the elderly, and those with chronic lung disease. In contrast to S. aureus, patients with Klebsiella have acute onset of severe disease with fever, rigors, and chest pain. Patients with Klebsiella may develop pulmonary abscesses, although more commonly radiographs show a lobar infiltrate.

Pseudomonas causes a severe pneumonia with cyanosis, confusion, and other signs of systemic illness. The chest radiograph may show bilateral lower lobe infiltrates, occasionally associated with empyema. Pseudomonas is not a typical cause of community-acquired pneumonia, more often seen in patients with a prolonged hospitalization, who have received broad-spectrum antibiotics or high-dose steroid therapy, who have structural lung disease (including cystic fibrosis), or who are nursing home residents.

Haemophilus influenzae pneumonia occurs in any age, although it most commonly occurs in the elderly, or in those with chronic lung disease, sickle cell disease, or immunocompromised disorders and in alcoholics and diabetics. Since the onset of routine vaccination of children, the incidence of H. influenzae pneumonia in children has markedly dropped. Patients with this type of community-acquired pneumonia may either have a gradual progression of disease with low-grade fever and sputum production or occasionally the sudden onset of chest pain, dyspnea, and sputum production. Bacteremia may be seen in older adults. Pleural effusions and multilobar infiltrates are common findings in H. influenzae pneumonia.

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Moraxella catarrhalis pneumonia has clinical features similar in spectrum to those of H. influenzae. Typically, patients with M. catarrhalis present with an indolent course of cough and sputum production, with fever and pleuritic chest pain are common. The chest radiograph usually shows diffuse infiltrates.

Pneumonia from Atypical Bacteria and Viruses

The atypical bacteria are Legionella, Chlamydophila, and Mycoplasma. Because these agents lack a cell wall, they do not respond to β-lactam antibiotics but respond to macrolides or a respiratory fluoroquinolone.

Legionella can cause a range of illness from benign self-limited disease to multisystem organ failure with acute respiratory distress syndrome. Patients at particular risk include cigarette smokers, patients with chronic lung disease, transplant patients, and the immunosuppressed. **There is no seasonality to** Legionella **pneumonia, making it a more prominent cause of pneumonia in the summer when other pathogens decline in frequency.** Legionella pneumonia is commonly complicated by GI symptoms, including abdominal pain, vomiting, and diarrhea. In addition, Legionella can affect other organ systems, causing sinusitis, pancreatitis, myocarditis, and pyelonephritis. The chest radiograph frequently shows a patchy infiltrate, with the occasional appearance of hilar adenopathy and pleural effusions (**Figure 65-3**).

FIGURE 65-3.
Classic diffuse, patchy infiltrates seen with Legionella pneumonia.

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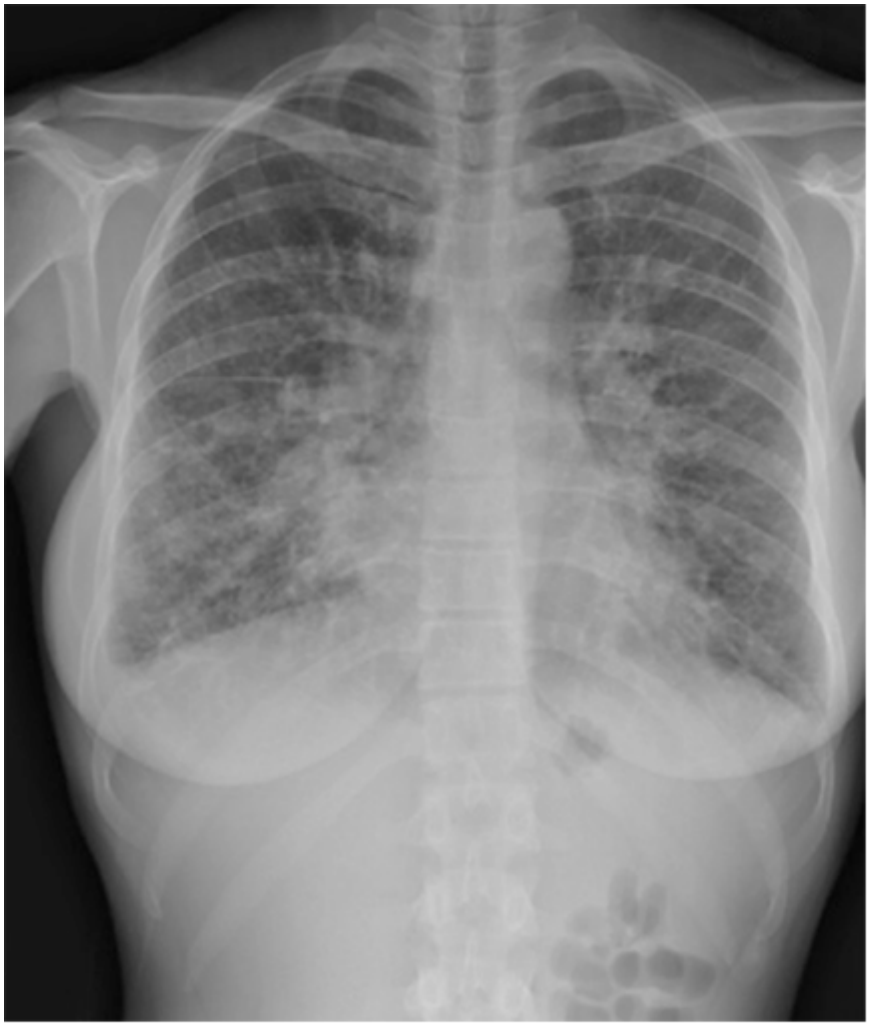

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Infection with Chlamydophila usually causes a mild illness with sore throat, low-grade fever, and nonproductive cough, although occasionally patients have a more severe course. Patients with Chlamydophila pneumonia frequently have rales or rhonchi. The chest radiograph usually shows a patchy subsegmental infiltrate, overlapping with the appearance of Legionella pneumonia. Chlamydial infection is linked to adult-onset asthma.

Mycoplasma pneumonia also occurs year-round, although it tends to cluster in epidemics every 4 to 8 years. Mycoplasma may cause a s[illegible], sore throat, and headache. Mycoplasma pneumonia is also frequently associated with

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retrosternal chest pain. Unlike Legionella, Mycoplasma usually is not associated with GI symptoms. Like the other atypical pathogens, chest radiograph often shows patchy infiltrates, commonly with hilar adenopathy or pleural effusions. Mycoplasma occasionally causes extrapulmonary symptoms, including rash, neurologic symptoms, arthralgia, hematologic abnormalities, or rarely acute kidney injury. Bullous myringitis is not at all specific for Mycoplasma infection but is associated with many other causes of otitis media.

Viruses cause pneumonia, often severe; influenza is the most common viral pneumonia and is seasonal. The outbreak of severe acute respiratory syndrome and the Middle East respiratory syndrome, both from coronaviruses, demonstrates how a local infection can be rapidly transmitted worldwide.[12] The most recent pandemic viral infection has been H1N1 in 2009. Varicella, typically benign in most childhood infections, can lead to a virulent pneumonia in pregnant patients. Further discussion of life-threatening viral infections is covered elsewhere (see chapter 153, "Serious Viral Infections").

DIAGNOSIS

Suspect pneumonia from symptoms and signs (often fever, cough, dyspnea, or weakness with rales or rhonchi), while recognizing that each individual symptom or finding lacks high accuracy. When symptoms suggest a possibility, order a chest radiograph; if clinical findings suggest pneumonia (with or without an infiltrate on chest x-ray), treat emprically.[13] No single set of recommendations for diagnostic testing applies to all patients, requiring clinical judgment. In otherwise healthy, mildly ill, ambulatory patients, no further ancillary testing may be necessary. To optimally risk-stratify anyone over 50 years old or more than mildly ill, seek evidence of other organ affliction; this is done by including CBC, serum electrolytes, BUN, creatinine, and glucose levels. Pulse oximetry is needed in all cases because a saturation on room air of <91% is associated with more complications. An arterial blood gas analysis is reserved for those appearing ill, with underlying lung disease, with oxygen desaturation, or in respiratory distress.

Most patients do not require identification of a specific organism through blood or sputum analysis to direct antibiotic treatment. The incidence of positive blood cultures in nonhospitalized patients with community-acquired pneumonia is low, pathogen identification usually does not alter treatment, and the majority of patients respond to empiric antibiotic treatment. The value of sputum culture is similar to the value of blood cultures and often limited by poor sampling, with less than 15% being adequate and helpful.[14] Atypical agents may be detected by evaluation of titers from acute and convalescence sera or by direct fluorescent antibody testing.

In hospitalized community-acquired pneumonia patients, the incidence of positive blood cultures increases along with increasing disease severity.[15] For this reason, **obtain blood cultures in those admitted to the intensive care unit and in those with leukopenia, cavitary lesions, severe liver disease, alcohol abuse, asplenia, or pleural effusions.**[16] In any admitted patient, a **sputum culture**

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and Gram stain are options if an adequate sample can be obtained. Legionella urine antigen tests are useful in intensive care unit patients, alcoholics, those with classic findings, and those with a recent (within the past 2 weeks) travel history.

The differential diagnosis of patients with cough and radiographic abnormality includes lung cancer, tuberculosis, pulmonary embolism, chemical or hypersensitivity pneumonitis, connective tissue disorders, granulomatous disease, and fungal infections. Because radiographic signs of pneumonia vary, it is difficult to predict the causative microorganism by its radiographic appearance. In general, patients with bacterial pneumonia are more likely to have unilobar or focal infiltrates than patients with viral or atypical pneumonia. Hilar adenopathy is more common in patients with atypical pneumonia. Pleural effusions can accompany bacterial, viral, or atypical pneumonia. Cavitary lesions occur in patients with bacterial pneumonia or tuberculosis. Lung abscesses are rare complications of pneumonia in the antibiotic era, usually due to S. aureus or Klebsiella. Pneumococcal and staphylococcal pneumonia may mimic a lung mass, along with other atypical pneumonias, such as Q fever and tularemia.

PNEUMONIA IN SPECIAL POPULATIONS

Pneumonia in Alcoholics

Alcoholics have a higher risk than the normal population for many lung diseases, including pneumonia, tuberculosis, pleurisy, bronchitis, empyema, and chronic obstructive pulmonary disease. Alcoholics are more likely than the general population to be undernourished, to develop aspiration pneumonitis, to be heavy smokers, and to have sequelae of alcoholic cirrhosis and portal hypertension. Compared with the nonalcoholic, the alcoholic has greater oropharyngeal colonization with gram-negative bacteria, and also has depressed granulocyte and lymphocyte counts with impaired neutrophil delivery.

S. pneumoniae **is the most common pathogen causing pneumonia in alcoholics, but** Klebsiella **species and** Haemophilus **species are also important agents of infection.** In general, rates of pneumonia and subsequent mortality are higher in alcoholics compared with nonalcoholic patients.

Pneumonia in Diabetics

Diabetic patients between the ages of 25 and 64 years old are four times more likely to have pneumonia and influenza, and diabetics are two to three times more likely than nondiabetics to die with pneumonia and influenza as an underlying cause of death. **Pathogens that occur with increased frequency in diabetic patients include** S. aureus**, gram-negative bacteria, mucormycosis, and** M[illegible]ons due to S. pneumoniae, Legionella pneumophila, and influenza are associated with increased morbidity and mortality in diabetic patients.

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Pneumonia in Pregnant Women

Community-acquired pneumonia in pregnancy is one of the most serious nonobstetric infections, with maternal mortality of approximately 3%. Pregnancy does not alter the course of bacterial pneumonia, but the prognosis of viral pneumonia during pregnancy is more serious than in the nonpregnant patient. Pregnant woman are at risk for developing severe influenza-associated pneumonia, and antivirals are often used in this patient group (see chapter 153, "Serious Viral Infections").

Pregnant women who develop varicella pneumonia are more often smokers and have skin lesions suggestive of the disease on exam. **Obtain a chest radiograph and pulse oximetry measure in any pregnant woman with symptoms of respiratory tract infection and varicella exposure.** Empiric IV acyclovir is often used, although there is little evidence that the timing of administration affects outcome.

Pneumocystis jiroveci pneumonia is the most common cause of acquired immunodeficiency syndrome–related death in pregnant women in the United States, with a mortality of approximately 50%; over half receive mechanical ventilation during hospitalization. Combination treatment with pentamidine, steroids, and eflornithine improves survival compared with patients treated with trimethoprim-sulfamethoxazole alone.

Pneumonia in the Elderly

Pneumonia is the most common serious elderly infection, representing the fifth leading cause of death.[17] The incidence of lower respiratory tract infection in the elderly ranges from 25 to 44 cases per 1000 in the general population, with a mortality rate approaching 40%. Chronic obstructive pulmonary disease, congestive heart failure, cardiovascular and cerebrovascular disease, lung cancer, dementia, diminished gag reflex, and other aspiration risks make the elderly susceptible to infection.

Those over age 65 years are three times more likely to have pneumococcal bacteremia than younger patients. Atypical pathogens are still more common in younger populations but occur in the elderly. Legionella is the most common atypical agent in the elderly and is responsible for up to 10% of cases of community-acquired pneumonia. **Influenza is the most common serious viral infection in the elderly. Postinfluenza bacterial pneumonia, whether following H1N1 or other seasonal influenza, is most commonly caused by** S. pneumoniae**,** S. aureus**, or** H. influenzae**.** This usually presents as a worsening of respiratory symptoms after days of improvement.

Elderly patients with pneumonia may present with nonpulmonary symptoms like falls, weakness, tremulousness, functional decline, a[illegible]fusion. Elderly patients are more likely to be afebrile on presentation but are more likely than younger adults to have a serious bacterial infection when the temperature is higher than 38.3°C (100.9°F).

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Age alone does not confer a poor prognosis until extremes (over 85 years), but age does interact with other organ dysfunction to increase mortality and morbidity. Up to one third of elderly patients with community-acquired pneumonia will not manifest leukocytosis. Poor prognostic indicators for pneumonia in the elderly include hypothermia or a temperature >38.3°C (100.9°F), leukopenia, immunosuppression, gram-negative or staphylococcal infection, cardiac disease, bilateral infiltrates, and extrapulmonary disease. Elderly pneumonia patients frequently require hospitalization, and 10% receive intensive care.

Pneumonia in Nursing Home Patients

Pneumonia is a major cause of morbidity, mortality, and hospitalization among nursing home residents.[17,18] Nursing home patients are less likely than those living independently to have a productive cough or pleuritic chest pain, but more likely to be confused and have poorer functional status and more severe disease.[18] Eight findings are independent predictors of pneumonia in nursing home patients: increased pulse rate, respiratory rate ≥30 breaths/min, temperature ≥38°C (100.4°F), somnolence or decreased alertness, presence of acute confusion, lung crackles on auscultation, the absence of wheezes, and an increased leukocyte count.[19] A patient with one of these features has a 33% chance of having pneumonia, whereas three or more features suggest a 50% likelihood of pneumonia.[19] Fewer than 10% of nursing home patients with pneumonia will have no respiratory symptoms. Fever, although nonspecific, is present in approximately 40% of cases of nursing home–acquired pneumonia.

The most frequently reported pathogens among patients with nursing home–acquired pneumonia are S. pneumoniae**, gram-negative bacilli, and** H. influenzae**.** Because nursing home patients live in close proximity to each other, residents are subject to outbreaks of influenza. Vaccination against influenza is 33% to 55% effective in preventing postinfluenzal pneumonia in nursing home patients. M. pneumoniae and Legionella are uncommon causes of pneumonia in nursing home patients.

Nursing home–acquired pneumonia is often treated in the hospital, but some patients can be treated in nursing homes with either intramuscular or oral antibiotics.[20] **Nursing home patients are at risk for the organisms linked to health care–associated pneumonia, so therapy should include coverage for gram-negative bacteria and methicillin-resistant** S. aureus**.**[4]

Pneumonia in Human Immunodeficiency Virus Patients

Community-acquired pneumonia accounts for roughly three fourths of bacterial pneumonia diagnosed in patients hospitalized with HIV infection. Compared with HIV-seropositive patients hospitalized without pneumonia, those admitted with pneumonia generally have a

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lower CD4+ T-cell count, a higher Acute Physiology and Chronic Health Evaluation II score, a longer length of hospital stay, a greater chance of intensive care unit admission, and a higher case fatality rate.

S. pneumoniae **is the most common cause of bacterial pneumonia in patients with HIV.** Pseudomonas aeruginosa **is also a common cause of bacterial pneumonia in HIV-positive patients**. HIV-positive patients with P. aeruginosa pneumonia, compared with HIV-negative patients, are more likely to have a lower leukocyte and CD4+ T-cell count and a longer hospital stay but a similar case fatality rate. (See chapter 154, "Human Immunodeficiency Virus Infection.")

Pneumonia in Transplant Patients

Bacterial pneumonia is more common in patients receiving liver, heart, or lung transplants during the first 3 months after surgery, compared with other transplant and surgical patients. Gram-negative bacilli (especially P. aeruginosa associated with mechanical ventilation), S. aureus, and Legionella predominate in the first 3 months posttransplantation. K. pneumoniae, Escherichia coli, and fungi may also cause pneumonia in this time period. These early-onset nosocomial bacterial pneumonias carry a substantial mortality rate, approximately 33%. Cytomegalovirus, P. jiroveci, and fungal infections, especially Aspergillus species, are opportunistic infections, which may be seen in the first 6 months after surgery. **After 6 months posttransplantation, typical community-acquired pneumonia bacteria (**S. pneumoniae**,** H. influenzae**) are common and less often fatal than earlier infections** (see chapter 297, "The Transplant Patient").

TREATMENT

Emergency physicians start community-acquired pneumonia therapy usually based on bedside features rather than culture data; the Infectious Diseases Society of America and the American Thoracic Society guidelines, along with the American College of Emergency Physicians (ACEP) Clinical Policy, help care decisions.[21] Pediatric recommendations are provided in the chapter 125, "Pneumonia in Infants and Children."

The drugs listed in Tables 65-4, 65-5, 65-6, 65-7, 65-8, 65-9[4,16,22] are based on site acquisition and comorbidities but do not represent a comprehensive list. Other antibiotic regimens are effective and guided by local resistance patterns and availbility.[16] Outpatient treatments are listed in **Tables 65-4** and **65-5**. Inpatient treatments are listed in **Tables 65-6, 65-7, 65-8, 65-9**.

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TABLE 65-4

Therapy for Outpatient Treatment of Uncomplicated Patients*

Class	Examples	Comments
Macrolide	Azithromycin, 500 milligrams PO on day 1 and 250 milligrams on days 2–5	Respiratory fluoroquinolones reserved for those who cannot tolerate or have failed other therapy
	or	
	Clarithromycin XL, 1000 milligrams PO each day for 7 d	
Tetracycline-like macrolide	Doxycycline, 100 milligrams twice a day for 10–14 d	Second-line choice

*Other drugs may also be effective.

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TABLE 65-5

Therapy for Outpatient Management of Patients with Significant Comorbidities* without Criteria for Healthcare-Associated Pneumonia

Class	Examples	Comments
Fluoroquinolone	Levofloxacin, 750 milligrams daily for 5 d	Other respiratory fluoroquinolones may also be used.
	or	
	Moxifloxacin, 400 milligrams daily for 7–14 d	
β-Lactamase inhibitor penicillin derivative	Amoxicillin-clavulanate, 2 grams twice daily	A third-generation cephalosporin may be used instead of the aminopenicillin.
plus	plus	
Macrolide	Azithromycin, 500 milligrams PO on day 1 and 250 milligrams on days 2–5	

*Significant comorbidities include chronic heart, lung, liver, or renal disease; diabetes mellitus; alcoholism; malignancies; and asplenia. See text. Dosing may need adjustment for patients with renal insufficiency. Other therapies may also be effective.

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TABLE 65-6

Inpatient Therapy for Non–Intensive Care Unit Patients* with Community-Acquired Pneumonia

Class	Examples	Comments
Fluoroquinolone	Levofloxacin, 750 milligrams IV	Other respiratory fluoroquinolones may also be used.
	or	
	Moxifloxacin, 400 milligrams IV	
Cephalosporin or penicillin class with β-lactamase inhibitor	Ceftriaxone, 1 gram IV or ampicillin/sulbactam 1.5 gram IV	Other third-generation cephalosporins may also be used in combination with other macrolides or doxycycline.
plus	plus	
Macrolide	Azithromycin, 500 milligrams IV	

*Other drugs may also be effective; dosing may need adjustment for patients with renal insufficiency. Oral therapy with selected drugs may be acceptable for non–intensive care unit patients.

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TABLE 65-7

Empiric Therapy for Patients with Suspected Healthcare-Associated Pneumonia

Three-Drug Regimen Recommended		
Class	**Examples**	**Comments**
Antipseudomonal cephalosporin	Cefepime, 1–2 grams every 8–12 h	An aminoglycoside may be substituted in place of the fluoroquinolone. Levofloxacin, 750 milligrams every 8 h, may be substituted for ciprofloxacin. Linezolid, 600 milligrams every 12 h, may be substituted for vancomycin.
	or	
	Ceftazidime, 2 grams every 8 h	
plus	plus	
Fluoroquinolone	Ciprofloxacin, 400 milligrams every 8 h*	
plus	plus	
Anti-MRSA drug	Vancomycin, 15 milligrams/kg every 12 h	
Antipseudomonal carbapenem	Imipenem, 500 milligrams every 6	An aminoglycoside may be substituted in place of the fluoroquinolone. Levofloxacin, 750 milligrams every 8 h, may be substituted for ciprofloxacin. Linezolid, 600 milligrams every 12 h, may be substituted for vancomycin.

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Three-Drug Regimen Recommended		
Class	**Examples**	**Comments**
	or	
	Meropenem, 1 gram every 8 h	
plus	plus	
Fluoroquinolone	Ciprofloxacin, 400 milligrams every 8 h*	
plus	plus	
Anti-MRSA drug	Vancomycin, 15 milligrams/kg every12 h	
β-Lactam/β-lactamase inhibitor	Piperacillin-tazobactam, 4.5 grams every 6 h	An aminoglycoside may be substituted in place of the fluoroquinolone. Levofloxacin, 750 milligrams every 8 h, may be substituted for ciprofloxacin. Linezolid, 600 milligrams every 12 h, may be substituted for vancomycin.
plus	plus	
Antipseudomonal fluoroquinolone	Ciprofloxacin, 400 milligrams every 8 h*	

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Three-Drug Regimen Recommended		
Class	**Examples**	**Comments**
plus	plus	
Anti-MRSA drug	Vancomycin, 15 milligrams/kg every 12 h	

Note: Healthcare-associated pneumonia risks include (1) patients hospitalized for 2 or more days within the past 90 days, (2) nursing home/long-term care residents, (3) patients receiving home IV antibiotic therapy, (4) dialysis patients, (5) patients receiving chronic wound care, (6) patients receiving chemotherapy, and (7) immunocompromised patients.

Abbreviation: MRSA = methicillin-resistant Staphylococcus aureus.

*See text. Dosing may need adjustment for patients with renal insufficiency.

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TABLE 65-8

Inpatient Therapy for Intensive Care Unit Patients*

Class	Example	Comments
Cephalosporin	Ceftriaxone, 1 gram IV	Other β-lactams may also be used in place of ceftriaxone. See Table 65-9 for additional recommendations.
plus	plus	
Macrolide	Azithromycin, 500 milligrams IV	
Cephalosporin	Ceftriaxone, 1 gram	Other β-lactams may also be used in place of ceftriaxone. See Table 65-9 for additional recommendations.
plus	plus	
Fluoroquinolone	Either moxifloxacin, 400 milligrams IV	
	or	
	Levofloxacin, 750 milligrams IV	
Fluoroquinolone	Moxifloxacin, 400 milligrams IV	Aztreonam is generally well tolerated in penicillin-allergic patients.
plus	or	
Either a monobactam	Levofloxacin, 750 illigrams IV	

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Class	Example	Comments
or	plus	
Lincosamide	Either aztreonam, 1–2 grams IV	
	or	
	Clindamycin, 600 milligrams IV	
Anti-MRSA drug (add if HCAP or MRSA risk)	Vancomycin, 10–15 milligrams/kg IV	To be added to one of the above regimens for patients with MRSA or HCAP risk.
	or	
	Linezolid, 600 milligrams IV	

Abbreviations: HCAP = healthcare-associated pneumonia; MRSA = methicillin-resistant Staphylococcus aureus.

*Other combinations may also be used. Dosing may need adjustment for patients with renal insufficiency.

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TABLE 65-9

Inpatient Therapy for Patients with Higher Pseudomonas Risk*

Class	Example	Comments
β-Lactam/β-lactamase inhibitor	Piperacillin-tazobactam, 3.375 milligrams IV	Other antipseudomonal cephalosporins or quinolones may be used. Carbapenems are also appropriate. Consider adding an aminoglycoside if substituting a macrolide.
plus	plus	
Fluoroquinolone	Ciprofloxacin, 400 milligrams IV	
Monobactam	Aztreonam, 1 gram IV	May be used for patients with penicillin allergy. Carbapenems and aminoglycosides may also be appropriate.
plus	plus	
Fluoroquinolone	either Moxifloxacin, 400 milligrams IV	
	or	
	Levofloxacin, 750 milligrams IV	
Anti-MRSA drug (add if HCAP or MRSA risk)	Vancomycin, 10–15 milligrams/kg IV	To be added to one of the above regimens for patients with MRSA or HCAP risk.
	or	

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Class	Example	Comments
	Linezolid, 600 milligrams IV	

Abbreviations: HCAP = healthcare-associated pneumonia; MRSA = methicillin-resistant Staphylococcus aureus.

*Other combinations may also be used. Dosing may need adjustment for patients with renal insufficiency.

In **outpatients,** single-drug therapy is the common first choice, using a macrolide or a respiratory fluoroquinolone, with doxycycline as an alternative. Erythromycin is a cost-effective agent for community-acquired pneumonia but is associated with GI side effects in about 25% of adult patients and also causes photosensitivity. Clarithromycin has fewer GI side effects, although some patients may complain about a metallic taste. Azithromycin has the advantage of once-a-day dosing or single-dose therapy with the newer formulations. The newer fluoroquinolone agents, including moxifloxacin, levofloxacin, and gemifloxacin, extend coverage to both common bacterial agents and atypical agents, along with the advantage of once-a-day dosing. However, given concerns about resistance developing, **the Centers for Disease Control and Prevention recommends that fluoroquinolones be reserved for patients who cannot tolerate other agents, have documented pneumococcal resistance, or have failed other therapies. Fluoroquinolones should not be used in patients with myasthenia gravis.**

Patients who received broad-spectrum antibiotics within the previous 3 months are at risk for drug-resistant infection. In these patients, consider a respiratory fluoroquinolone or combination therapy using an aminopenicillin or a third-generation cephalosporin with a macrolide (including doxycycline). Patients with chronic cardiac, pulmonary, renal, or hepatic disease, severe diabetics, chronic alcoholics, patients on immunosuppressive therapy, or patients with asplenia may require therapy with more than one agent.

Inpatients not admitted to the intensive care unit benefit from coverage for both atypical and common organisms. Often, monotherapy using a respiratory fluoroquinolone is an option, although single-agent therapy with a macrolide or doxycycline is avoided. An aminopenicillin/β-lactamase or cephalosporin in combination with a macrolide or with a respiratory fluoroquinolone is common.

Treat patients admitted to the intensive care unit with a combination of agents, including an aminopenicillin or cephalosporin with either a respiratory fluoroquinolone or a macrolide. Penicillin-allergic patients could be treated with a respiratory fluoroquinolone with either a[illegible]s at risk for Pseudomonas infection, add at least two agents active against the organism. This may

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include an antipseudomonal β-lactam such as piperacillin-tazobactam or cefepime with a respiratory fluoroquinolone. Alternatively, a carbapenem, such as imipenem, along with either a fluoroquinolone or aminoglycoside is appropriate. In penicillin-allergic patients, use a monobactam along with a fluoroquinolone. If the combination does not include two drugs with antipseudomonal activity, consider adding an aminoglycoside. Patients admitted to the intensive care unit with healthcare-associated pneumonia should have coverage for methicillin-resistant S. aureus with drugs such as vancomycin or linezolid.[22]

Emergency physicians play a prominent role in the initiation of treatment for patients being hospitalized with community-acquired pneumonia, although the effect of delays in care are debated.[16,23,24] The Joint Commission currently recommends starting antibiotics within 6 hours of diagnosis. Although it is reasonable to not delay therapy, there is no single best time frame to optimize outcomes without overuse.

DISPOSITION AND FOLLOW-UP

Most patients with community-acquired pneumonia do not require hospitalization.[25] In general, physicians tend to overestimate the risk of pneumonia mortality. **It is best to use an illness severity or prognostic tool to better aid care site decisions**.

The best-tested tool is the **Pneumonia Severity Index (PSI),** which estimates the risk of short-term death and intensive care unit need in those with community-acquired pneumonia.[25,26,27] With the Pneumonia Severity Index, patients are assigned to one of five risk categories (class I to V) based on points (starting with age, adjusted for sex) and bedside features; **the lowest risk patients (<50 years old with minimal x-ray and vital sign/concomitant condition threats) need no further testing;** all others have labs tested to better assess prognosis and organ function. Nonhypoxemic (room air saturation of 91% or higher) Pneumonia Severity Index class I to III patients have <4% mortality and are candidates for outpatient therapy. The Pneumonia Severity Index was not designed to determine prognosis in patients with severe HIV, patients with other forms of pneumonia (ventilator or hospital-acquired), or pregnant patients. Other factors, such as social situation or unusual medical conditions, play a role in the admission decision. Also, one profound derangement should drive care (e.g., blood pressure <60 mm Hg systolic or presence of coma), irrespective of the class assigned. A brief hospitalization or observation is an alternative for class III patients with mild hypoxemia, and class IV and V patients are usually treated as inpatients. A free online Pneumonia Severity Index calculator is available at http://pda.ahrq.gov/clinic/psi/psicalc.asp, and a version that can be downloaded to a hand-held computer is obtainable from http://pda.ahrq.gov/clinic/psi/psi.htm.

The **CURB-65 rule** looks at the presence of confusion, uremia >7 mmol/L, respiratory rate ≥30 breaths/min, age ≥65 years old, or abnormal blood pressure (systolic <90 mm Hg or diastolic <60 mm Hg), with 1 point assigned for each factor.[20] The CRB-65 uses the

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same variables but eliminates the uremia measurement. Patients with a CURB-65 or CRB-65 score of <2 have a low mortality rate and are candidates for outpatient therapy.

The Pneumonia Severity Index and CURB scoring systems inform but do not determine care location. Some patients are better served based on social or medical factors not assessed by these scores. Nonetheless, the validated scoring tools help safely increase the number of appropriate patients treated on an outpatient basis and to limit unnecessary admissions.[26] Absent a structured approach, admission decision patterns for patients with community-acquired pneumonia among emergency physicians can vary widely, often unrelated to disease severity and socioeconomic states.

Once the decision to admit the patient is made, the next decision is to determine which patients require admission to the intensive care unit. Patients in septic shock or those requiring mechanical ventilation will be placed in an intensive care unit setting. Other criteria for intensive care unit admission include a markedly elevated respiratory rate, a partial pressure of arterial oxygen/fraction of inspired oxygen ratio ≤250, multilobar infiltrates, confusion, uremia with a BUN >20 milligrams/dL, leukopenia, thrombocytopenia, hypothermia, hyponatremia, lactic acidosis, and asplenia. No single criterion will mandate intensive care unit admission; consider intensive care unit or intermediate-care admission for patients with three or more of the criteria in **Tables 65-10, 65-11, 65-12**. Those with a PSI class of V or a CURB-65 of ≥3 often require intensive care.[20,25]

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TABLE 65-10

Step 1 of Pneumonia Severity Index (PSI)

Step 1: Assess initial factors

Age (if <50 y old, move on to next features; if ≥50 y, go to Step 2)

Comorbid conditions—ask about:

Neoplastic disease

Cerebrovascular disease

Congestive heart failure

Renal disease

Liver disease

Physical examination

No altered mental status

Pulse <125 beats/min

Respiratory rate <30 breaths/min

Systolic blood pressure >90 mm Hg

Temperature >35°C (95°F) or <40°C (104°F)

If all negative, then assign to risk class I (lowest risk); if over 50 y or any abnormality present, go on to Step 2 testing and classification in Table 65-11.

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TABLE 65-11

Step 2 of Pneumonia Severity Index: Assignment to Risk Classes II to V

Criteria	Points Given
Demographics	
Age	Men: Age (in years) Women: Age (in years) – 10
Nursing home resident	10
Coexistent illness (same as Step 1)	
Neoplastic disease	30
Congestive heart failure	10
Cerebrovascular accident	10
Renal disease	10
Liver disease	20
Physical examination (same as Step 1)	
Abnormal mental status	20
Pulse ≥125 beats/min	10
Respiratory rate >30 breaths/min	20
Systolic blood pressure (<90 mm Hg)	20
Temperature <35°C (95°F) or >40°C (104°F)	15

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Criteria	Points Given
Ancillary studies	
Arterial pH <7.35 (may assume normal if clinical condition suggests)	30
BUN ≥30 milligrams/dL (11 mmol/liter)	20
Na <130 mEq/L	20
Glucose >250 milligrams/dL (14 mmol/liter)	10
Hematocrit <30%	10
Pa_{O_2} <60 mm Hg or O_2 saturation on room air <91%	10
Pleural effusion	10
Summary points risk assignment	
Sum of points <70 = risk class II Sum of points 71–90 = risk class III Sum of points 91–130 = risk class IV Sum of points >130 = risk class V	

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TABLE 65-12

Prediction of Mortality from Pneumonia

Class	Points	Mortality (%)	Treatment Recommendation
I	No predictors	0.1	Outpatient
II	<70	0.6	Outpatient
III	71–90	2.8	Individualized; nonhypoxemic may be candidate for home therapy
IV	91–130	8.2	Inpatient
V	>130	29.2	Inpatient (often intensive care unit)

Most patients will achieve some resolution within 3 to 5 days after the initiation of antibiotics. Many hospitalized patients can be switched to oral antibiotics at approximately 3 days and then subsequently discharged to complete a course of therapy. After ED or hospital discharge, contact within 3 to 5 days may help avoid a need for repeat care. However, up to half of patients are still symptomatic at 30 days, with a significant minority of patients experiencing chest pain, malaise, or mild dyspnea even 2 to 3 months after treatment. Educate patients about smoking cessation and moderation of alcohol use, and provide information about rest, nutrition, hydration, follow-up, and the importance of pneumococcal and influenza vaccination.

Finally, not all radiographic infiltrates result from an infection. Congestive heart failure may present a radiographic picture that overlaps with pneumonia, and pulmonary embolism can be associated with segmental or lobar densities. A variety of cancers may mimic pneumonia, best detected by CT scan or repeat radiographs after therapy. Eosinophilic or fungal diseases often have transient or recurring infiltrates. Finally, obtain an occupational history to identify patients with hypersensitivity disorders or chemical pneumonitis.

ASPIRATION PNEUMONIA

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Aspiration pneumonia results from the swallowing of colonized oropharyngeal contents into the lower respiratory tract with subsequent inflammation and infection. Aspiration pneumonitis is from exposure of sterile gastric contents into the lower respiratory tract. This results in a rapid chemical pneumonitis due to irritation of the pulmonary tissues from the acidic material. Aspiration pneumonia—infection occurring from the exposure—is often unwitnessed, especially in the elderly. Other aspiration syndromes include drowning, solid foreign body aspiration with or without asphyxia, and lipoid pneumonia. Sterile pneumonitis and aspiration pneumonia are difficult to distinguish from one another, even with bronchial lavage. When certain, the treatment of aspiration pneumonitis is largely supportive.[28]

Aspiration of small amounts of oropharyngeal content is common. Approximately half of healthy adults aspirate small amounts oropharyngeal secretions during sleep.[28] Silent aspiration is more common in patients with community-acquired pneumonia. Aspiration occurred in 71% of patients with pneumonia compared to 10% of control subjects.[29] Approximately 5% to 15% of community-acquired pneumonia cases result from aspiration,[30,31,32] and 30% of those in continuing care facilities with pneumonia have aspiration pneumonia.[30,33] Mortality rates differ for aspiration based on location, with mortality higher in nursing home patients with aspiration pneumonia (28.2%) compared with those with community-acquired aspiration pneumonia (19.4%).[30,33] The incidence of aspiration pneumonia in those who aspirate with acute stroke or chronic degenerative neurologic conditions is higher than in patients without those conditions.[30]

Risks for aspiration pneumonia include conditions that promote oropharyngeal colonization with pathogenic bacteria or conditions that impair the swallowing or gag mechanism (**Table 65-13**). The incidence of aspiration is highest in patients with dementia or stroke. The risk of infection is compounded by poor oral care, which leads to oropharyngeal colonization of the oral cavity. Placement of nasogastric or gastric feeding tubes and the use of sedative and neuroleptic drugs also increase the risk of aspiration.[34,35,36,37,38] Aspiration pneumonia is the most common cause of death in gastric tube–fed patients.[39]

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TABLE 65-13

Risk Factors for Aspiration Pneumonia

Intoxicants
Alcohol and illicit drugs
Therapeutic drug overdose
Sedative drug use
Procedural sedation
General anesthesia
Neurologic
Stroke, especially brainstem involvement with dysphagia
Seizure
Head trauma
Chronic debilitating neurologic condition, especially dementia
Oropharyngeal
Impaired glottic functions
Emergent intubation
Periodontal disease and poor oral hygiene
GI
High gastric pressures: prior meal, bag-mask ventilation
Gastroesophageal reflux
Esophageal dysmotility or obstruction
Nasogastric, orogastric, percutaneous gastric tube
Tracheobronchial fistula
Other
Chronic supine position
Rapid sequence intubation
Advanced age
Chronic debility
Extension neck contractures

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Although many patients have clinical evidence of aspiration, along with dysphagia, emesis, or coughing while eating, up to one third of those who aspirate have "silent aspiration" without evidence of cough or gagging. Small bowel obstruction, gastroesophageal reflux, esophageal dysmotility, esophageal obstruction, and tracheoesophageal fistula are GI risk factors for aspiration. Chronic degenerative neurologic conditions such as Parkinson's disease, myasthenia gravis, amyotrophic lateral sclerosis, acute stroke, encephalopathy, seizures, and alteration of consciousness increase the risk of aspiration.

PATHOPHYSIOLOGY

The development of pneumonitis depends on the volume and pH of the aspirate, with consensus that gastric contents with pH <2.5 and an aspirated volume of 0.3 to 0.4 mL/kg (20 to 30 mL in adults) are required to develop aspiration pneumonitis.[32] The injury produced by acid aspiration is initially a direct caustic effect followed by an inflammatory response that peaks in 4 to 6 hours. Proinflammatory cytokines increase capillary permeability and cause fluid and inflammatory cells to enter the area of irritation. These reactions may manifest clinically as cough, pleuritic chest pain, fever, and radiographic findings. Aspiration of solid or viscous material blocking the airway may result in precipitous asphyxiation.

Typical bacterial species involved in aspiration pneumonia include S. pneumoniae, S. aureus, H. influenzae, and Enterobacteriaceae in community-acquired aspiration pneumonia.[32] Common bacterial species in hospital-acquired aspiration pneumonia include P. aeruginosa and gram-negative organisms.[32] Antibiotic therapy for typical aspiration syndromes should include coverage for anaerobic organisms.[40,41]

The posterior portions of the upper lobes and the upper portions of the lower lobes are most commonly involved in recumbent aspiration. In upright patients, the most dependent portions of the lungs are the basal segments of the lower lobes. The inflammatory injury may include bilateral patchy, interstitial or alveolar infiltrates, particularly in aspiration of large volumes seen with near drowning.[42]

CLINICAL FEATURES

Witnessed aspiration is a key feature in the diagnosis of aspiration pneumonitis or pneumonia. Typically those with noninfectious aspiration are younger, and the aspiration is witnessed. These patients will present giving a history of aspiration and coughing immediately afterward. "Silent aspirators" are typically older and have a chronic neurologic disorder and will present with a cough or fever or general malaise. Silent aspirators are more likely to be from a chronic care facility and have a history of prior pneumonia

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episodes. Historical features that suggest silent aspiration include general debility, recurrent cough, hoarseness, or dysphagia. History may be difficult to obtain in chronically debilitated or otherwise noncommunicative patients.

The clinical symptoms of aspiration pneumonia include fever, dyspnea, and productive cough. Other symptoms of systemic infection in the elderly and debilitated may be present, including a change in mental status, lethargy, and nausea or vomiting.[43] The physical examination may reveal signs classic for pneumonia, including tachycardia, tachypnea, fever, rales, or decreased breath sounds in an ill-appearing patient. Patients with underlying pulmonary disease may decompensate rapidly and have more symptoms and signs of respiratory distress.

DIAGNOSIS

Chest radiographs in aspiration pneumonia usually show unilateral focal or patchy consolidations in the dependent lung segments (**Figure 65-4**). Occasionally, a bilateral or interstitial pattern can be seen. The right lower lobe is the most common area of consolidation if the aspiration occurs when the patient is upright.

FIGURE 65-4.
Aspiration pneumonia of the right lower lobe.

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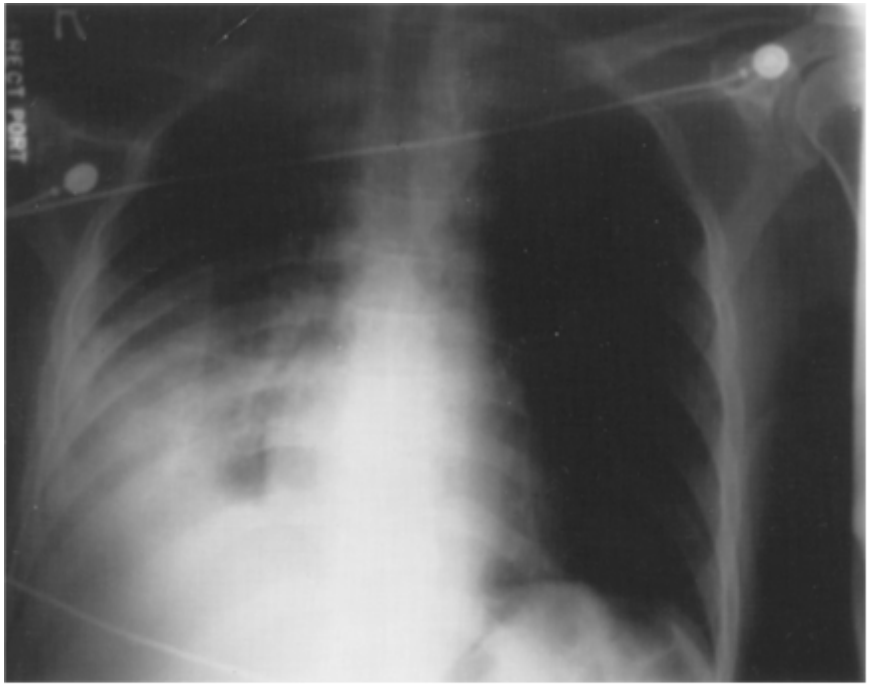

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Early in the course, the white blood cell count may not be elevated. Arterial blood gases to identify hypoxemia or hypoventilation aid care and are best compared to previous values if chronic lung disease exists.

TREATMENT

Aspiration of large volumes of solid material, foods, nonfood objects, or very tenacious liquids may require suctioning of the tracheobronchial tree or bronchoalveolar lavage to clear the airway. Bronchodilators aid aspiration-induced bronchospasm.

Choice of antibiotics depends on the circumstances of the aspiration and the suspected bacterial etiology of the infection (**Table 65-14**).[32,44,45] Most patients with aspiration pneumonia are infected by gram-negative organisms and require broad-spectrum antibiotics, such as third-generation cephalosporins, fluoroquinolones, piperacillin-tazobactam, or carbapenems.[28] When methicillin-resistant S. aureus is suspected, consider the addition of vancomycin or linezolid.[28] In community-acquired aspiration pneumonia where the usual organisms are S. aureus, S. pneumoniae, and H. influenzae, levofloxacin and ceftriaxone are recommended.[46] Patients with severe

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periodontal disease, putrid sputum, or lung abscess require anaerobic coverage, such as piperacillin-tazobactam or imipenem or a combination of two drugs (levofloxacin, ciprofloxacin, or ceftriaxone plus clindamycin or metronidazole).[46]

TABLE 65-14

Initial Treatment for Presumed Aspiration Pneumonia

Acquisition Site	Empiric Therapy	Empiric Therapy for Penicillin Allergy
Community acquired	Ampicillin/sublactam or Amoxicillin/clavulanate or Levofloxacin or Moxifloxacin	Clindamycin
Hospital acquired or Severe periodontal disease, putrid sputum or alcoholism	Pipracillin-tazobactam +/– Vancomycin +/– gentamicin or Cefepime or ceftazidime plus Clindamycin or metronidazole or Levofloxacin + clindamycin	Ciprofloxacin + vancomycin

DISPOSITION

Healthy persons who aspirate small volumes of nontoxic material may be observed and, if stable and reliable, discharged to return for

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ent is generally not needed for witnessed aspiration of a small amount of nontoxic liquid provided

the patient's symptoms (cough, low-grade fever) resolve within 24 to 48 hours.[28] Patients should be able to identify worsening symptoms and have the ability to follow up if symptoms worsen.

Stable patients at risk for worsening (e.g., diabetes, advanced age, renal dialysis, recent stroke, chronic pulmonary disease, active cancer and HIV) are usually admitted to the hospital or an observation unit. Start antibiotics in the ED in those who have definite evidence of infection. Deliver supplemental oxygen as needed, and treat cardiopulmonary compromise. Noninvasive positive-pressure ventilation and endotracheal intubation are options if gas exchange is impaired (see chapters 28 "Noninvasive Airway Management" and 29 "Intubation and Mechanical Ventilation"). Admit all unstable patients to an intensive care unit.

NONINFECTIOUS PULMONARY INFILTRATES

Commonly, a noninfectious cause is suspected by the appearance of the chest radiograph or after antibiotics fail to improve the patient's symptoms. Noninfectious infiltrates occur in response to a wide variety of pathophysiologic processes involving the respiratory, cardiovascular, and immune systems or may be due to the infiltration of malignant cells.

CLINICAL FEATURES

The most important symptom of noninfectious pulmonary infiltrates is dyspnea, but some disorders present with hemoptysis, cough, chest pain, or fatigue. Fever can also be a symptom of autoimmune disease exacerbation; however, it may be impossible to clinically differentiate a noninfectious source from an infectious source of fever in the ED.

Table 65-15 lists the most common causes of noninfectious pulmonary infiltrates, their pathophysiology, chest radiograph findings, and symptoms.[45,47,48,49,50,51,52] Typical radiographic appearances help to prioritize the differential diagnosis. An interstitial infiltrate is classically described as fine, diffuse, linear density representing fluid or the accumulation of cells in the interstitial spaces. An alveolar infiltrate is a small ill-defined or reticular density representing fluid or abnormal cells in the alveoli. A ground-glass appearance is defined as multiple finely granular densities. Table 65-15 lists diseases that produce acute radiographic change, not diseases that produce chronic densities, such as fibrosis or scarring.

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TABLE 65-15

Noninfectious Causes of Pulmonary Infiltrates

Disease	Pathophysiology	Chest Radiography Findings	Symptoms
Congestive heart failure	As pulmonary capillary hydrostatic pressure rises, fluid crosses into the interstitium. When the capacity of lymphatic drainage is exceeded, fluid accumulates in the interstitium and eventually collects in the alveoli.	With increasing atrial pressures, chest x-ray reveals cephalization, Kerley B lines (thickening of the interlobular septa), interstitial edema, thickening of fissures, and, ultimately, alveolar edema and pleural effusions.	See chapter 53, Acute Heart Failure.
Pulmonary embolism	Pulmonary artery occlusion, typically at multiple sites, with its secondary effects, including infarction.	Chest x-ray nonspecific or normal: cardiac enlargement (27%), normal (24%), pleural effusion (23%), elevated hemidiaphragm (20%).	See chapter 56, "Venous Thromboembolism."
Aspiration pneumonitis	After aspiration of gastric contents, an intense inflammatory reaction occurs; biphasic pattern, at 1–2 h, caustic effect of low pH of the aspirate on alveolar cells; at 4–6 h, infiltration of neutrophils into the alveoli and lung interstitium.	Alveolar infiltrates in the posterior segments of the upper lobes if aspiration occurs while the patient is in the supine position; infiltrates in the basal segments of the lower lobes if aspiration occurs while the patient is upright.	See "Aspiration Pneumonia" section earlier in this chapter.
Allergic bronchopulmonary aspergillosis	Allergic lung reaction to Aspergillus fumigatus that occurs most commonly in patients with asthma or cystic fibrosis. Eosinophils fill small airways and alveolar spaces due to Bronchiectasis.	Branching band-like opacities may be seen, or may see alveolar infiltrates, peripheral and migratory.	Dyspnea, wheezing, productive cough; may have hemoptysis and occasionally fever.

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Disease	Pathophysiology	Chest Radiography Findings	Symptoms
Eosinophilic lung disease; chronic (Löffler's syndrome) and acute forms exist	The acute form leads to rapidly progressive respiratory failure. This is a reaction to the presence of infection, such as ascariasis, or in association with chronic conditions, such as asthma or atopic disease. Eosinophils accumulate in distal airways and alveolar and interstitial spaces.	Alveolar and interstitial infiltrates; classically, these are peripheral, but findings are proximal in equal numbers.	Mild or severe symptoms; dyspnea, fever, cough, and wheezing. In acute forms, hypoxia and potential for progression to respiratory failure.
Hypersensitivity pneumonitis (also called extrinsic allergic alveolitis)	Inflammation of the alveoli secondary to hypersensitivity in response to inhaled organic dust.	Diffuse micronodular interstitial infiltrates, may see ground-glass densities in the lower or mid-lung fields.	Fever, chills, malaise, cough, chest tightness, dyspnea, and headache.
Acute interstitial pneumonitis	Idiopathic.	Bilateral interstitial infiltrates, sometimes patchy alveolar densities and ground-glass appearance.	Progressive dyspnea over days to weeks.
Acute respiratory distress syndrome	Reaction of the lung to a number of precipitating causes, including sepsis, trauma, surgeries, transfusions, and therapeutically induced immunosuppression.	Classically, patchy peripheral infiltrates that extend to the lateral lung margins suggest the diagnosis.	Hypoxia, tachypnea, rales.
Drug-induced pneumonitis	Causes include chemotherapeutic, immunosuppressive, antimicrobial, and herbal agents.	Typically, bilateral interstitial infiltrates.	Cough, mild fever, dyspnea, and, potentially, hypoxia.

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Disease	Pathophysiology	Chest Radiography Findings	Symptoms
Sarcoidosis	Systemic granulomatous disease of unknown etiology.	Hilar lymph node enlargement and/or diffuse parenchymal interstitial pulmonary infiltrates. Occasionally may be peripheral.	Dyspnea, cough, weight loss; skin lesions may also be found. May be asymptomatic.
Bronchiolitis obliterans with organizing pneumonia	Inflammation of the bronchioles and surrounding tissues, leading to loss of the integrity of the bronchioles and organizing pneumonia without infection. Occurs in association with immuno-compromise and connective tissue diseases, such as SLE.	Patchy alveolar infiltrates and, occasionally, cavitation.	Cough, dyspnea, fever.
Wegener's granulomatosis (granulomatosis with polyangiitis)	Inflammatory and granulomatosis disease involving the blood vessels of unknown etiology. The upper respiratory tract, lung parenchyma, and kidneys are typically affected.	Alveolar infiltrates, nodules, or cavities.	Cough and dyspnea; may see epistaxis and sinusitis.
Goodpasture's syndrome (antiglomerular basement antibody disease)	Autoimmune disease affecting the lungs and kidney.	Diffuse, bilateral, predominately alveolar densities.	Fatigue, dyspnea, cough with hemoptysis; may have simultaneous hematuria.
Churg-Strauss vasculitis (eosinophilic granulomatosis	Systemic vasculitis of unknown cause primarily affecting the lung and may eventually involve the skin, nervous system, kidney, GI tract, and heart.	Bilateral peripheral, patchy, alveolar infiltrates. May see nodules.	Cough, dyspnea, allergic rhinitis, may see symptoms related to skin, coronary, or intestinal involvement.

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Disease	Pathophysiology	Chest Radiography Findings	Symptoms
Radiation pneumonitis	Interstitial pulmonary inflammation seen in 5%–15% of patients with thoracic radiation treatment.	Subtle hazy ground-glass densities to marked patchy infiltrates or homogenous consolidation. Air bronchograms are commonly present.	Symptoms occur 1–6 mo after treatment: low-grade fever, cough, fullness in the chest.
Chemical pneumonitis	Inflammatory reaction to the presence of foreign substance, such as barium, petroleum distillates, pesticide, or irritating gases.	Diffuse, alveolar, and interstitial infiltrates.	History of exposure or aspiration of substance; acute dyspnea, cough, and, possibly, wheezing.
Alveolar cell carcinoma, often called bronchiolar carcinoma	Malignant pulmonary cancer originating in a bronchiole and spreading across the alveolar walls.	Classically, a butterfly distribution of alveolar infiltrates, but may be unilateral.	Often severe coughing, dyspnea, and copious sputum production.
Bronchoalveolar cell carcinoma	Adenocarcinoma that typically originates in the lung periphery and grows along alveolar walls.	Peripheral alveolar infiltrates that do not respond to antibiotics; may see a peripheral nodule or mass.	Symptoms like pneumonia but fails to respond to antibiotics. May lack fever or leukocytosis.
Fat emboli	ED presentations are typically after trauma, associated with long-bone fracture.	Interstitial prominence, suggesting interstitial edema. Radiographic findings may be delayed 1–2 d after trauma.	Dyspnea, cough, hemoptysis, and pleural pain. May be associated with confusion, stupor, delirium, and a petechial skin rash, most commonly on the chest.

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Disease	Pathophysiology	Chest Radiography Findings	Symptoms
Alveolar hemorrhage	In the setting of chemotherapy induction for leukemia and thrombocytopenia (<20,000/mL), hemorrhage filling alveoli is common from endobrachial and interstitial sources. Also seen with SLE.	Focal or diffuse alveolar infiltrates.	Dyspnea, hemoptysis; symptoms are frequently less severe than radiographic appearance would predict.
Leukemic infiltrates	Most common in myeloid leukemia when peripheral blast cell counts exceed 100,000/mL. Primitive myeloid leukemic cells invade through the endothelium of the lung capillary beds, yielding hemorrhage.	Interstitial or alveolar infiltrates. Diffuse infiltrates associated with hypoxia and need for intubation; focal infiltrates associated with coexistent pneumonia.	Respiratory distress, hypoxemia, and may progress to respiratory failure.

Abbreviation: SLE = systemic lupus erythematosus.

Patients with connective tissue diseases or on immunosuppression are at risk for bronchiolitis obliterans, organizing pneumonia, pneumonitis, and alveolar hemorrhage, often from therapy. Patients with atopic disease or asthma are at risk for eosinophilic lung disease.

DIAGNOSIS

Assess critically ill patients with CBC, renal function assessment, electrolytes, chest radiograph, and liver function tests, and add specimen cultures to look for complications of the acute disease process rather than establishing a new disease diagnosis. Newer high-sensitivity procalcitonin assays help differentiate infection from an exacerbation of a systemic inflammatory condition (very low values suggest a nonbacterial cause).[53] In many patients, an infectious cause of pulmonary infiltration cannot be excluded until bronchoscopy or lung biopsy. Therefore, the diagnostic plan can rarely be completed in the ED.

TREATMENT

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Assess gas exchange with pulse oximetry in all, and use selective arterial blood gas analysis in those ill, hypoxemic, or with underlying chronic lung disease; use noninvasive or mechanical ventilation as needed. Prepare for possible subglottic stenosis with difficult airway equipment in patients with systemic inflammatory disease.[52] Broad-spectrum antibiotics, such as piperacillin-tazobactam (3.375 to 4.5 grams IV) and vancomycin (1 gram IV), or similar coverage, are recommended for critically ill patients with pneumonia and systemic inflammatory disease or immunocompromise.[48] Definitive treatment for noninfectious pulmonary diseases will occur after ED stabilization. Many of the disorders listed in Table 65-15 are treated acutely with corticosteroids such as methylprednisolone (0.5 to 1 gram IV).[52] Additional immunosuppressive drugs may be initiated by the admitting physician.

DISPOSITION AND FOLLOW-UP

Patients suspected of having a noninfectious cause of pulmonary infiltrate require testing beyond the capabilities of the ED. Hospitalization should be based on the severity of the medical illness, with attention to hypoxemia, hypercapnia, and work of breathing. In stable patients with mild symptoms, outpatient referral to a pulmonologist or another specialist is best.

PRACTICE GUIDELINES

Guidelines concerning the management of community-acquired pneumonia[16] and healthcare-associated pneumonia[4] are undergoing revision, but will be available online at the following address: http://www.thoracic.org/professionals/clinical-resources/disease-related-resources/pneumonia.php.

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USEFUL WEB RESOURCES

American Thoracic Society, Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia—http://www.thoracic.org/professionals/clinical-resources/disease-related-resources/pneumonia.php

Current information concerning severe acute respiratory syndrome from the World Health Organization—http://www.who.int.une.idm.oclc.org/csr/don/archive/disease/severe_acute_respiratory_syndrome/en

Infectious Diseases Society/American Thoracic Society Guidelines for the treatment of pneumonia—http://www.thoracic.org/statements/resources/mtpi/idsaats-cap.pdf

Pneumonia Severity Index calculator—http://pda.ahrq.gov/clinic/psi/psicalc.asp

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