Management of Pediatric Community-acquired Bacterial Pneumonia

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Practice Gaps

Management of pediatric community-acquired pneumonia should focus on judicious use of antimicrobial medications, bacterial diagnostics, and surgical drainage when complicated by large effusion and empyema. Treatment in adherence to national guidelines produces favorable outcomes.

Objectives

After completing this article, readers should be able to:

1. Reinforce rational antibiotic use for bacterial community-acquired pneumonia (CAP) in outpatient and inpatient settings.
2. Review and update techniques for microbial diagnosis of CAP.
3. Review medical and surgical management of complicated pneumonia.
4. Present specific considerations for CAP in patients with neuromuscular disease.

INTRODUCTION

Community-acquired pneumonia (CAP) is the most common cause of death in children worldwide, accounting for 15% of deaths in children younger than 5 years of age. (1) Nearly 1 in 500 children will be hospitalized for CAP, which creates a substantial economic burden. CAP is thus important to diagnose and appropriately treat. While viral causes of CAP are most common, differentiating viral versus bacterial etiologies can be difficult. This leads to excessive use of antimicrobial medications or susceptibility to feeling a pressure to prescribe. (2) Overall, in the United States, 11.4 million antimicrobial prescriptions for pediatric respiratory tract infections per year are avoidable. (3) Furthermore, broad-spectrum but less effective antimicrobial agents are often prescribed when pharmacokinetically favorable narrow-spectrum agents are available. (4) Arguably, the untoward effects of overtreatment of CAP in those in whom treatment is unwarranted compounds the morbidity of this disease process. Because of mounting knowledge of antimicrobial side effects, resistance, and microbiome effects, practitioners must adhere to the principles of judicious use when treating CAP. In this regard, CAP, its epidemiology, various etiologic origins, clinical

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ABBREVIATIONS

CAP	community-acquired pneumonia
CT	computed tomography
IV	intravenous
MIC	minimum inhibitory concentration
MRSA	methicillin-resistant *Staphylococcus aureus*
PCR	polymerase chain reaction
VATS	video-assisted thorascopic surgery

presentations, and general diagnosis and treatment were thoroughly reviewed in this journal (5) and are also discussed at length in national guidelines. (6) The intent of this review is to supplement this excellent work by focusing on specific treatment once a provider has weighed the risks and benefits and decided that a child's condition warrants treatment of bacterial CAP and management of its complications. Salient details from both sources are summarized throughout.

REVIEW OF INITIAL DIAGNOSIS

No standard of reference for diagnosis or single definition of pneumonia exists. In this review, CAP is defined as an acute lower respiratory tract infection acquired in a previously healthy individual. Associated symptoms include fever, cough, dyspnea, and tachypnea with supporting evidence of parenchymal infection and inflammation, diagnosed according to findings at chest auscultation or the presence of focal opacity seen on chest radiographs. (5)(7) Focal opacity on chest radiographs is often held as a standard of reference; however, some viral processes and atelectasis can cause focal radiographic findings (though atelectasis traditionally resolves in 48–72 hours). In addition, findings on radiographs can lag behind clinical symptoms. Viral pneumonitis accounts for most respiratory infections, particularly in children younger than 5 years of age. Unfortunately, no constellation of clinical symptoms or signs (fever, tachypnea, hypoxemia, work of breathing) displays good specificity or sensitivity for radiographic findings of pneumonia, except that symptom severity and ill appearance do correlate with focal infiltrates. To exclude pneumonia, investigators in 1 study assessed the absence of cough, crackles (rales), rhonchi, retractions, and nasal flaring in young infants and found it useful in its negative predictive value; but again, the presence of these findings was insensitive in the prediction of pneumonia at radiography. (8) Left with few alternatives, pediatricians typically use a combination of radiographic and physical findings to decide to treat a patient for bacterial disease. Decisions on whether to hospitalize a patient are made by weighing the criteria presented in Table 1, which were adapted from national guidelines and prior review articles. (5)(6)

When to perform imaging in cases of acute pneumonia is not well delineated, although some rules of thumb apply. Chest radiographs are indicated in patients with more severe respiratory distress, particularly those who meet criteria for hospitalization. This imaging modality is used to assess the presence of focal parenchymal opacities, as well as screening for the presence of complications such as effusion or empyema in patients who have not responded to antibiotic treatment. Per Infectious Disease Society of America guidelines, other indications for chest radiography include inconclusive clinical findings and ruling out other possible causes of respiratory distress that can be diagnosed at radiography (foreign body, pneumothorax, pleural disease, or cardiac disease, including pulmonary edema and cardiomegaly). Imaging is also indicated in febrile infants without a source who are younger than 12 months of age, if there is evidence of leukocytosis. Conversely, in patients with mild evidence of lower respiratory tract infection (fever, cough) without hypoxemia or a focal lung examination who are stable for outpatient treatment, radiographs of the chest are not typically indicated. (9)(10)

Laboratory examinations are considered for all patients ill enough to be hospitalized with suspected bacterial pneumonia. These may commonly include blood cultures, inflammatory markers, complete blood cell count, and nasopharyngeal swab polymerase chain reaction (PCR) for viruses. Blood cultures rarely yield positive findings in CAP, and they should not be performed in patients treated on an outpatient basis or in hospitalized patients with uncomplicated disease. However, in patients with severe disease, the 10% to 18% yield is arguably worthwhile. (11) Inflammatory markers (erythrocyte sedimentation rate, C-reactive protein level, or procalcitonin) may aid in clinical decision-making if measured longitudinally, particularly in those with complicated CAP (discussed later). (12) The complete blood cell count may provide information on further complications, such as thrombocytopenia or anemia from hemolytic uremic syndrome. (6) Nasopharyngeal swabs for viral PCR should only be performed if the results will change management. The number and types of examinations performed depends on the severity and trajectory of the illness. A thorough history may lead one to consider testing for other unusual causes of lobar pneumonia. These causes and their historical cues are listed in Table 2.

In the remainder of this review, it is assumed that a practitioner has weighed the clinical, radiologic, and laboratory evidence for their patient as discussed earlier, reviewed national guidelines, and made a judicious decision to treat bacterial causes of CAP. With this context in mind, we will discuss pathogenesis, outpatient and inpatient management with respect to routine and novel diagnostics, antimicrobial choices and length of therapy, diagnosis and management of complications, and recurrent lobar pneumonia and special CAP considerations for patients with neuromuscular disease.

PATHOGENESIS AND BASIC DEFINITIONS

Pneumonia occurs as result of invasion of the lower respiratory tract by a pathogenic organism. Bacterial infection

TABLE 1. **Criteria to Consider Hospitalization for Pediatric Pneumonia**

• Hypoxemia (oxygen saturations <90% to 92% at sea level)
• Infants <3 to 6 months of age with suspected bacterial community-acquired pneumonia
• Tachypnea:
◦ Infants <12 months of age: respiratory rate >70 breaths per min
◦ Children: respiratory rate >50 breaths per min
• Respiratory distress: apnea, grunting, difficulty breathing, and poor feeding
• Signs of dehydration or inability to maintain hydration or oral intake
• Capillary refill time >2 s
• Infants and children with toxic appearance
◦ Suspected or confirmed to have infection with a virulent organism (community-acquired methicillin-resistant *Staphylococcus aureus* or group A *Streptococcus*)
• Underlying conditions/comorbidities that:
◦ May predispose patients to a more serious course (eg, cardiopulmonary disease, genetic syndromes, neurocognitive disorders, neuromuscular disorders)
◦ May be worsened by pneumonia (eg, metabolic disorder)
◦ May adversely affect response to treatment (eg, immunocompromised host, sickle cell disease)
• Complications (eg, effusion and/or empyema)
• Failure of outpatient therapy (48–72 h with no clinical response)
• Caretaker unable to provide appropriate observation or to comply with prescribed home therapy
Indications for intensive care unit admission include:
• Severe respiratory distress or impending respiratory failure that requires:
◦ Intubation and mechanical ventilation
◦ Positive pressure ventilation
• Recurrent apnea or slow irregular respirations
• Cardiopulmonary monitoring due to cardiovascular compromise secondary to:
◦ Sustained tachycardia
◦ Inadequate blood pressure
◦ Requirement of pharmacological support for blood pressure or perfusion
◦ Altered mental status due to hypercarbia or hypoxemia
◦ Pulse oximetry measurement of <92% on fractional inspired oxygen concentration of >0.50
• Pediatric Early Warning Score >6

Reproduced with permission from Gereige RS, Laufer PM. Pneumonia. Pediatr Rev. 2013;(34):19. (5)

represents a failure of many layers of extrinsic and intrinsic defense. Physical barriers to infection include upper respiratory tract nasal hairs and turbinate architecture, as well as complex respiratory airway branching that inhibits access to distal airways. In the large airways, cough and mucociliary clearance of secretions and humoral and cell-mediated defenses work to defend the lower respiratory tract from invasion. Secreted and humoral immunoglobulins, as well as intrinsic antimicrobial properties of alveolar fluid, work with the phagocytic alveolar macrophages to eradicate bacteria. When these defenses are overwhelmed in some capacity, bacterial pathogens penetrate and cause disease. (13)

Many factors may contribute to overwhelming of these defenses and subsequent pneumonia, but the influence of

TABLE 2. **Unusual Causes of Pneumonia in Children: A Summary of Historical Clues**

AGENT	HISTORICAL CLUES	SUMMARY OF INCUBATION, DIAGNOSTICS, AND TREATMENT (CONSULT REFERENCES FOR DETAILS)[a]
Bacillus anthracis	Exposure to contaminated hides (including drum covers); often will have skin manifestation (eschar), as well	Incubation: 2–43 d Diagnostic: culture[b] and PCR Treatment: ciprofloxacin, doxycycline
Blastomyces dermatitidis	Travel to Central United States	Incubation: 2 wk to 3 mo Diagnostic: culture[b], serologic analysis Treatment: amphotericin
Chlamydophila psittaci	Exposure to sick birds	Incubation: 5–14 d Diagnostic: serologic analysis Treatment: doxycycline, azithromycin second line
Coccidioides immitis	Travel to endemic area (Arizona, Nevada, California, Texas, Utah, Mexico, Central and South America)	Incubation: 1–4 wk for primary infection, disseminated disease weeks to years Diagnostic: culture[b], serologic analysis Treatment: not always needed, but fluconazole, itraconazole, amphotericin B
Coxiella burnetti	Exposure to infected birthing fluids or excreta (including unpasteurized milk) from sheep, cattle, and goats	Incubation: 14–22 d Diagnostic: PCR and serologic analysis, best if acute and convalescent Treatment: doxycycline best, second-line TMP-sulfa
Cryptococcus gatii	Travel to endemic area (Pacific Northwest)	Incubation: 8 wk to 13 mo Diagnostic: culture[b] Treatment: amphotericin
Entamoeba histolytica	Exposure to contaminated food, most commonly in resource-limited settings, institutionalized settings, or men who have sex with men; occurs in conjunction with liver abscess or triad of liver abscess, parapneumonic effusion, pericardial effusion	Incubation: days to years, most commonly 2–4 wk Diagnostic: identification of organisms in sample, serology Treatment: metronidazole plus luminal amebicide
Francisella tularensis	Exposure to ticks and potentially horseflies or sick animals (most notoriously rabbits); history of lawn-mowing over carcasses	Incubation: 1–21 d (typically 3–5 d) Diagnostic: culture[b], PCR of blood or source, serologic analysis Treatment: aminoglycoside, ciprofloxacin
Hantavirus	Exposure to mice feces and/or urine in endemic area (Colorado, Utah, New Mexico, Arizona); often hemoconcentration with thrombocytopenia	Incubation: 1–6 wk Diagnostic: serologic analysis Treatment: supportive
Histoplasmosis	Travel to endemic area (Central United States), exposure to birds and/or bird excrement	Incubation: 1–3 wk for primary infection, disseminated disease weeks to years Diagnostic: culture[b], serologic analysis, urine antigen Treatment: not always needed, but if so amphotericin B, itraconazole
Legionella pneumophila	Exposure to contaminated water supply	Incubation: 2–10 d Diagnostic: culture, antigen in urine, serologic analysis Treatment: azithromycin, levofloxacin
Leptospira spp	Exposure to urine (or water contaminated with urine) of infected animals; usually some liver involvement, as well	Incubation: 2–30 d, usually 5–14 d Diagnostic: serologic analysis Treatment: penicillin
Mycobacterium tuberculosis	Exposure to infected persons or high-risk settings or to persons with chronic cough with such exposures	Incubation: highest risk for disease first 2 y after infection, but can be years Diagnostic: culture, rapid diagnostics, clinical Treatment: 4 drugs, see references
Mycoplasma pneumoniae	Exposure to infected person 1–4 weeks ago	Incubation: 1–4 wk (usually 2–3 wk) Diagnostic: PCR (preferred), serum immunoglobulin M Treatment: azithromycin

Continued

TABLE 2. (Continued)

AGENT	HISTORICAL CLUES	SUMMARY OF INCUBATION, DIAGNOSTICS, AND TREATMENT (CONSULT REFERENCES FOR DETAILS)[a]
Yersinia pestis	Exposure to infected animals, including prairie dogs, squirrels, ill cats and dogs, fleas 85% of US cases are in New Mexico, Colorado, Arizona, and California —	Incubation: 1–8 days Diagnostic: culture[b], PCR, serologic analysis Treatment: doxycycline, ciprofloxacin second-line TMP-sulfa

TMP-sulfa=trimethoprim/sulfamethoxazole.

[a] *Information for consideration of differential only; practitioners should refer to the AAP* Red Book *and national guidelines. Timing of positive serologic findings varies, and some diseases require acute and convalescent sera. Some organisms require specific culture conditions. Treatment regimens may depend on location and severity of disease.*

[2] *Alert the laboratory if a specimen will be sent for culture that has a high risk of infection for laboratory personnel.*

viral coinfection on bacterial pneumonia is an important concept. Animal models suggest that respiratory viruses destroy the respiratory epithelium and change the landscape of the cell surface to exhibit more antigen receptors. These changes impair the cough reflex and mucociliary clearance. In addition, viruses may inhibit normal macrophage function. Influenza is most commonly associated with subsequent bacterial superinfection, but suspicion for this entity should be high in any child with a viral prodrome who exhibits abrupt worsening of clinical status in a time frame in which a viral infection should be resolving. (14) A public health example of this viral-bacterial interplay is readily available, in that pneumococcal vaccines decrease the morbidity of influenza infections, while some viral vaccines decrease the incidence of radiographic findings of pneumonia. (15)

Bacterial pneumonia can be classified according to several pathophysiological definitions based primarily on radiologic and physical findings. Lobar pneumonia involves a single discrete lobe or lung segment of parenchymal inflammation, a discrete opacity on chest radiographs, and focal findings of crackles, bronchial breath sounds, and diminished aeration at auscultation. This classic pattern is typical of pneumococcal infection. Bronchopneumonia involves inflammation of the airways and interstitium and appears more diffuse on images, with scattered crackles, rhonchi, and asymmetrical aeration at examination, commonly associated with *Streptococcus pyogenes* or *Staphylococcus aureus*. Mixed peribronchial and interstitial disease with focal parenchymal inflammation is observed in cases of viral pneumonia that become subsequently bacterial (in patients with influenza, for example). Cavitary pneumonia is a result of tissue necrosis associated with *Mycobacterium tuberculosis*, although it can occur with other pathogens. (13) Complicated pneumonia includes parapneumonic effusions, pulmonary abscesses, bronchopleural fistulas, and necrotizing pneumonia.

CAUSATIVE PATHOGENS AND THEIR IDENTIFICATION

Definitive identification of bacterial etiologic origins in CAP is limited by lack of a primary sample for culture or PCR from the lower respiratory tract. This in turn limits our ability to describe with confidence the microbial and epidemiological patterns of bacterial pneumonia. That said, bacterial causes of CAP continue to include *Streptococcus pneumoniae*, *S aureus*, and *S pyogenes*. Overall, with the advent of *S pneumoniae* vaccines, the incidence of unequivocal bacterial CAP is decreasing, although of those who develop CAP, *S pneumoniae* remains the most common cause. Multiple studies in which antigen detection and nucleic acid PCR were used on culture-negative empyemas demonstrated that most culture-negative empyemas are caused by penicillin-susceptible, nonvaccine serotypes of *S pneumoniae*. (13)(16)(17) For *S aureus*, there is some evidence that pediatric lung infections from methicillin-resistant *S aureus* (MRSA) are increasing. (18)(19)(20) Because of immunization, herd immunity, and partial immune responses to even 1 dose of vaccine, invasive disease due to *Haemophilus influenzae* type B is now exceedingly uncommon. Nontypeable *H influenzae* strains are now responsible for most cases of invasive *Haemophilus* disease, including pneumonia. (21) Between 2003 and 2012, the annual incidence of invasive, nontypeable *H influenzae* disease was 1.6 cases per 100,000 children younger than 5 years of age. Invasive disease with *Moraxella catarrhalis* is similar. Studies on the evaluation of the role of these organisms are marred by easy contamination from the upper airway, and results are difficult to interpret. It is likely

that these organisms play a small role in unequivocal bacterial CAP, and that when they do, disease is likely to be less severe. *Mycoplasma* undoubtedly causes CAP and can cause lobar disease and effusions, although the role of treatment remains controversial (as discussed later). A list of less usual causes for pneumonia and when to consider them is presented in Table 2.

Inexpensive, reliable, noninvasive methods for establishing a bacterial etiologic origin in pediatric pneumonia are highly desirable to promote selection of the most narrow and effective antimicrobial treatment. Options for pathogen discovery include upper and lower airway samples, coupled with traditional culture methods, targeted PCR, and targeted relative quantitative PCR (although these PCR methods are not yet available except in research settings).

Upper-airway samples include nasopharyngeal washes and swabs and throat samples. These samples are useful in the detection of various bacteria, including *Mycoplasma pneumoniae, Chlamydophila pneumoniae, Bordetella pertussis,* and *Bordatella parapertussis*. Sputum expectoration is commonly used in adults but has been a challenge in pediatrics. In theory, sputum includes a lower airway sample. Sputum samples are informative in adults and have been studied with success in children as young as 1 month of age. (22) Coordination with a respiratory therapist to use specialized techniques to improve sputum expectoration and/or nasal aspiration may be required in children younger than 6 years of age to obtain a successful, high-quality specimen (fewer than 10 squamous epithelial cells per low-power field). While sputum collection is not necessary for evaluation in a patient treated on an outpatient basis, attempts should be made to obtain sputum in children with moderate to severe pneumonia who are hospitalized. (6) Low-quality sputum specimens are not meaningful, can be misleading, and should not be cultured. (13)(23) Studies of PCR of sputum and upper-airway samples are plagued by the bias of pretreatment and lack of comparison to the lower airway. In theory, these are improved by using quantitative PCR but are still problematic and are not yet readily available. (24)

Sampling techniques for the lower respiratory tract include direct pleural sampling, pleural fluid aspiration after placement of a chest tube or video-assisted thorascopic surgery (VATS), bronchoalveolar lavage via flexible bronchoscopy, or (rarely performed) direct biopsy of the parenchyma or open thoracotomy.

Sampling of pleural fluid without placement of a chest tube (thoracentesis) is the most direct way of obtaining more information but has become unfamiliar for many practitioners. For a period of time, it was believed that identifying the microorganism did not change the treatment, and amid concerns about surgical complications such as pneumothorax, bleeding, and pain, the practice has become much less common. In the current era of antimicrobial resistance, this should be reconsidered. Ideally, sampling should occur before treatment with antibiotics. Procedural instructions for thoracentesis and recommendations for radiologic assistance are available. (25) Commonly, pleural fluid is obtained with therapeutic procedures such as chest tube placement or VATS, which almost always occur after some period of antimicrobial therapy, often resulting in negative culture findings.

When pleural fluid is obtained, it should be sent for Gram stain and bacterial culture, as well as cell count and differential, to allow differentiation of bacteria from other causes of effusion (ie, mycobacterial, oncologic). Modified criteria, originally proposed by Light et al, (26) in 1972, allow differentiation of exudate from transudate on the basis of fluid pH level, presence of leukocytes, protein, glucose, and lactate dehydrogenase ratios. However, international guidelines do not recommend these tests because they rarely change management in pediatric cases with a high pretest probability of bacterial pneumonia. Culture of the fluid is crucial; however, if the patient is pretreated, 70% of findings are negative. (10) *S pneumoniae* is a particularly difficult bacterium to culture because of its propensity for autolysis and relative fragility during sample transport. (27) Other cultures should be based on unusual exposure history or clinical situations (Table 2).

Efforts to better characterize the bacterial components of pleural fluid with culture-independent methods are underway. Many studies focus on identification of *S pneumoniae* in culture-negative pleural samples via PCR-based identification. Antigen testing and PCR testing of pleural fluid greatly increase diagnostic yield, although these are not yet readily available. (15) A study in which uniplex PCR was used in pleural fluid demonstrated a causative organism in 82% of 56 children. (28) In addition to targeted PCR, relative quantitation of PCR, PCR for the gene-encoding bacterial 16S ribosomal RNA subunit, and deep sequencing are all up-and-coming techniques. (29) Urinary antigen tests for *S pneumoniae* are not recommended for children because of the high rate of false-positive results. (6)

In patients receiving mechanical ventilation, 2 additional options exist for obtaining lower respiratory tract specimens: bronchoscopy and tracheal aspirate. Tracheal aspirates are likely of similar utility to sputum and are most useful if obtained early, prior to colonization of the endotracheal tube with patient or hospital flora. The use of bronchoscopy to obtain a bronchoalveolar lavage sample is an option if other sources of microbial diagnosis cannot be obtained and should be particularly considered in complicated and/or immunosuppressed

hosts who may have unusual pathogens or in children who are not improving despite receiving adequate therapy for usual pathogens. In pediatrics, nonbronchoscopic bronchoalveolar lavage or mini–bronchoalveolar lavage has been studied for safety in older children (30) with ventilator-associated pneumonia but is not commonly used because of the size of the pediatric airway. (31)

OUTPATIENT MANAGEMENT

Uncomplicated bacterial pneumonia is an entity that can be effectively treated in most children on an outpatient basis with oral antibiotics and supportive care. Cough and fever are often present in any patient in whom pneumonia is being considered, but the degree of respiratory distress is important to assess for each patient. (32)

For management of outpatient mild to moderate bacterial pneumonia, treatment with antibiotics is empirical; it is not recommended to pursue tests to assess for a cause if the patient does not meet criteria for inpatient treatment. Criteria for hospitalization include hypoxemia, moderate respiratory distress, age younger than 12 months, and presence of a moderate to large pleural effusion, in addition to the criteria in Table 1. (5)(6) A child who meets the criteria for outpatient management will thus be relatively well and amenable to oral therapy but at risk for progression; thus, close follow-up is warranted.

A key to success in the outpatient realm is an appropriate choice and dose of an antimicrobial agent. Selection of appropriate oral antibiotics is based on assessment of presumed pathogens, patient age, exposures, prior medical history, medication allergies, and community bacterial resistance patterns. The key organism to cover in this setting is *S pneumoniae* because it remains the most common cause, despite vaccination. (15) The cornerstone of oral antimicrobial treatment for *S pneumoniae* is amoxicillin. Practitioners commonly presume that oral cephalosporins are superior to amoxicillin for *S pneumoniae*; this likely stems from knowledge that some *S pneumoniae* penicillin nonsusceptible isolates are susceptible to ceftriaxone and assume that oral cephalosporins are superior to amoxicillin. Indeed, the opposite is true. Oral cephalosporins have short half-lives, are poorly absorbed, are highly protein bound, and are often dosed at long intervals. This results in serum concentrations that do not provide enough killing time (serum concentration over minimum inhibitory concentration [MIC]) to treat, except for organisms with a low MIC to a selected drug. Amoxicillin reaches higher levels and is less protein bound, thus giving it more time with a drug concentration over the MIC for many pathogens, provided the MIC is in the susceptible or intermediate drug level range. Because the pharmacokinetics of the oral cephalosporins are far inferior to amoxicillin, their use in CAP should be reserved for patients who are allergic to penicillin or patients with a cause known to be resistant to amoxicillin but susceptible to cephalosporins (ie, *M catarrhalis* or β-lactamase–positive *H influenzae)*. (6)(33)

Another consideration for treatment with β-lactam antibiotics is the dosing interval. Many practitioners are unaware that more frequent dosing will provide more killing time and have the potential to treat organisms with slightly higher MICs. For example, for *S pneumoniae* with a penicillin MIC of 2.0 μg/mL, 90 mg per kilogram of body weight divided into doses administered twice daily will achieve cure in approximately 65% of patients, while if divided into doses administered 3 times daily, it is estimated to provide cure in 90%. (34) Thus, when amoxicillin is used, pharmacokinetics are superior if used in high doses (90–100 mg/kg per day) divided into doses administered 3 times a day, instead of twice daily. This dosing strategy should be selected where higher rates of nonsusceptible *S pneumoniae* exist or arguably in all patients with lobar CAP in whom room for error with outpatient treatment should be minimized. (35) Although twice-daily dosing is successful in otitis media because of the prolonged half-life of the drug in the ear fluid (and thus creating more time with a drug level over the MIC of the offending organism) when compared to serum (4 vs 1.2 hours, respectively), this cannot be safely extrapolated to true bacterial pneumonia. (36)

Although empirical coverage of *H influenzae* and *M catarrhalis* is not warranted in most patients, it is important to note that 30% of *H influenzae* and 100% of *M catarrhalis* produce a β-lactamase, rendering those isolates resistant to amoxicillin. They are routinely susceptible to amoxicillin–clavulanic acid and cephalosporins. Other microbial causes of CAP include *S aureus* and *S pyogenes,* although these bacteria do not usually cause disease mild enough to be treated in an outpatient setting. Oral antimicrobial selections are discussed in Fig 1.

M pneumoniae is known to cause diffuse or lobar CAP, but the benefits of treatment remain controversial. (37)(38) The ability of a practitioner to differentiate *Mycoplasma* from other etiologic origins by using clinical history and examination findings is not reliable and can lead to overtreatment of this pathogen. Although national guidelines recommend consideration of treatment in patients older than 5 years of age, (6) this may lead to undue pressure to treat, given the lack of proven benefit. The judicious practitioner should be allowed room to align with national reviews (38) and not routinely treat this entity empirically, particularly if symptoms

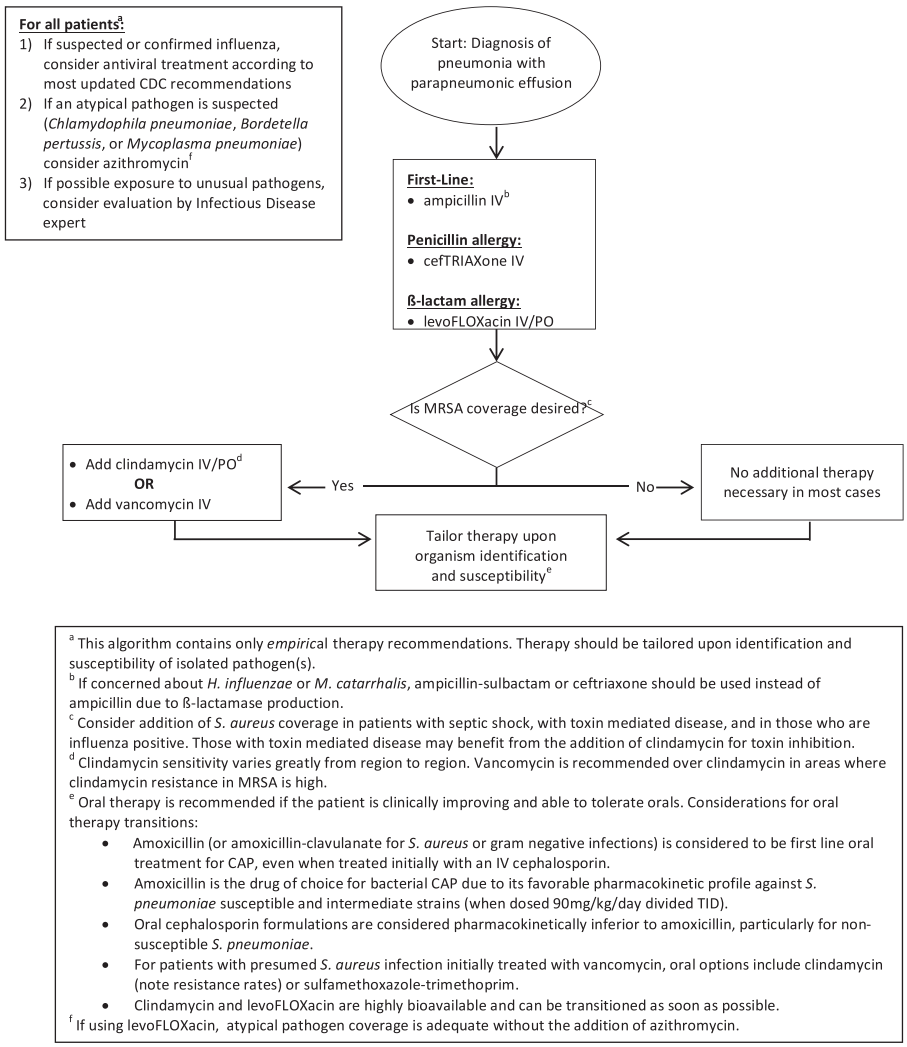

Figure 1. Complicated CAP empirical antibiotic therapy algorithm. Adapted from Complicated Community Acquired Pneumonia, Clinical Care Guidelines, Children's Hospital Colorado, updated October 11, 2016. (71) CAP=community-acquired pneumonia, CDC=Centers for Disease Control and Prevention, IV=intravenous, MRSA=methicillin-resistant *Staphylococcus aureus,* PO=per os, TID=3 times daily.

are also consistent with viral disease or if providers are already treating the patient for other bacterial causes. In adult populations, the desire to cover both *Mycoplasma* and bacterial causes has led to a crisis in the overuse of fluoroquinolones, a practice the Food and Drug Administration has strongly discouraged. (39) Though azithromycin is largely ineffective against the traditional CAP pathogens mentioned earlier, it is often used in an attempt to treat both typical and atypical infections, which contributes to the fact that it is the second most commonly prescribed antimicrobial agent in outpatient pediatrics. (40) Despite a recent publication in which investigators suggest that azithromycin may decrease subsequent wheezing when used in early childhood, (41) the difficulties of this research make the results inconclusive, and any potential benefit must be weighed against the need for dual therapy, side effects, development of resistance, and detrimental effects on the microbiome. (42)(43) Many centers now have rapid diagnostics to target *M pneumoniae*, so treatment might logically be reserved for hospitalized patients with positive PCR test findings.

Length of therapy for uncomplicated bacterial CAP should not exceed 7 days, and there are data to support 3 days for nonsevere CAP. (44) Studies have demonstrated similar success rates of 7 days when compared with 10 days and 5 days. (45)(46) Although all studies involving CAP are subject to the Pollyanna phenomenon (positivity bias), (47) the number and consistency of the shorter therapy studies increase the quality of the evidence such that the benefits (in terms of mitigating resistance, decreased side effects, and compliance) of 5 or 7 days should make these lengths standard.

A patient is considered to have failed outpatient antimicrobial therapy for CAP when clinical worsening occurs,

despite 48 hours of properly chosen and dosed antimicrobial agents. Notably, fever may persist (for an average of 48 hours), (48) but if a patient is improving in other ways (better oral intake, lower respiratory rate, increased normal activities), this would not be deemed a failure. If failure occurs, repeat chest radiography and consideration of hospitalization are in order. If the patient is hospitalized, it is not necessary to expand coverage unless resistant organisms are suspected (ie, rapid progression suggestive of *S aureus* or *S pyogenes*), since intravenous (IV) ampicillin reaches much higher serum levels than amoxicillin and provides extended killing time for *S pneumoniae*. One may suspect highly resistant *S pneumoniae* in children who have not received the pneumococcal conjugate vaccine PCV13, since they are not immunized against serotype 19A.

INPATIENT MANAGEMENT

Inpatient management of CAP can be separated into 2 patient scenarios: those who are admitted with viral pneumonitis and who might have superimposed CAP and those with a clear need-to-treat bacterial CAP, with or without a parapneumonic process. For the first category, the discussion of diagnosis, outpatient management, and hospitalization criteria in Table 1 was addressed earlier. For those with an undisputed need-to-treat bacterial CAP as the primary diagnosis, a few management principles apply. These include diligence in solidifying a microbial diagnosis, thoughtful antimicrobial therapy, and management of complicated disease.

Although inpatient bacterial CAP is still most likely to be *S pneumoniae*, other causes should be considered in certain inpatient settings. *S aureus* should be particularly considered in patients with influenza and superimposed CAP. *S aureus* and *S pyogenes* should be considered in those with rapidly progressive disease or signs and/or symptoms of sepsis or toxic shock. While ampicillin provides adequate coverage of *S pyogenes*, coverage for inpatients may need to be expanded for *S aureus* with consideration of MRSA coverage, depending on severity of disease and local resistance patterns. The need to cover *H influenzae* or *M catarrhalis* specifically in the inpatient setting in normal hosts is debatable, and hospitals that choose to prioritize the use of ampicillin and/or amoxicillin (which lack coverage for 30% of *H influenzae* and all *M catarrhalis*) demonstrate similar outcomes when compared to the historical use of more expanded regimens. (49) *S pneumoniae, S aureus,* and *S pyogenes* can all cause parapneumonic processes. For antibiotic treatment guidelines for inpatient CAP, please see Fig 1 on antibiotic choice (IV and oral step-down).

Studies support the use of standardized inpatient CAP guidelines. Per the Centers for Disease Control and Prevention Study of Etiology of Pneumonia in the Community on pneumonia causes, the use of inpatient clinical care guidelines improved the use of ampicillin and decreased use of cephalosporins and macrolides without negatively affecting outcomes. (50) In this study, investigators also looked at combined clinical physiological parameters (respiratory rate, oxygen saturation) for "time to clinical stability" and reported that guidelines were also helpful in determining timing of discharge. (51)

Given that moderate to severe CAP involves bacteria-triggered inflammation, investigations into adjunctive anti-inflammatory therapies have included macrolides and corticosteroids. Adjunctive corticosteroid use is supported in adults with severe CAP, with studies showing shorter time to clinical stability, shorter hospital lengths of stay, and possible decreased mortality. There were no clinically significant side effects identified in relation to corticosteroids in these patients. (52)(53) The use of corticosteroids has not yet been studied in pediatric CAP and should thus be approached with caution. A short steroid course (5–7 days) should be strongly considered in patients with CAP who received a diagnosis of asthma if they exhibit signs of reversible airway obstruction. Use of azithromycin as an anti-inflammatory agent in acute CAP remains controversial and is not recommended at this time. (54)(55)

COMPLICATED PNEUMONIA

There is substantial variability in admission rates for children seen in the emergency department for CAP independent of illness severity, and there is little to help clinicians predict which patients will go on to develop moderate to severe complications. In a recent study in *Pediatrics*, in which the Centers for Disease Control and Prevention Study of Etiology of Pneumonia in the Community data were also used, predictive analytics were used to develop 3 prognostic models to estimate risk for severe pneumonia outcomes in children. A simple electronic health record model in which 9 predictors were aggregated, including data on age, race, temperature, vital signs, and partial pressure of arterial oxygen to fractional index of oxygen ratio, was used to accurately identify risk for intensive care unit admission and severe outcomes, including the need for invasive mechanical ventilation and death. This work, despite requiring more validation, may provide an important tool for clinicians in determining those at highest risk for complications from CAP. (56)

Despite decreasing incidence of bacterial pneumonia and invasive pneumococcal disease attributed to vaccination

against *H influenzae* and *S pneumoniae*, studies indicate that the rate of empyema and other complications of bacterial CAP are increasing, particularly in preschool-aged patients. (18)(57) This is possibly due to pneumococcal serotype replacement and/or antibiotic resistance. (58) Complications of CAP include parapneumonic effusion, empyema, pulmonary abscess, bronchopleural fistula, necrotizing pneumonia, acute or impending respiratory failure, and sepsis. Please refer to Fig 2 for radiographic examples of some of these complications. *Parapneumonic effusion* refers to an exudative process that results in a pleural fluid collection due to pneumonia. Parapneumonic effusions develop in stages on the basis of duration. In the first several days, effusions are exudative and free flowing. By the second week, they become fibropurulent with fibrin deposition over the pleurae. Fluid can become septated. By 10 to 14 days, the effusion becomes organized, with a stiff pleural membrane. Empyema is a purulent effusion, with leukocytosis and/or bacteria in the pleural space. Effusions are categorized by size to aid in clinical decision-making (Fig 3). A bronchopleural fistula occurs when an erosion in the airway or parenchyma communicates directly with the pleura, such that air enters the pleural space. Necrotizing pneumonia occurs as a complication of both lobar and bronchopneumonia and is defined by a combination of parapneumonic effusion, loculation, and septation of the effusion and abscesses. These patients have a combination of the findings discussed earlier, exhibit diminished or absent aeration and crackles at auscultation, and typically appear ill and even toxic, with high fevers, hypoxemia, and malaise. Complicated CAP should be suspected in cases of previously healthy children with prolonged and persistent fever or deteriorating clinical status, despite receiving appropriate antibiotic treatment. Other clinical scenarios—such as rapid progression to impending or fulminant respiratory failure—or the presence of chronic comorbid illness—such as immunodeficiency, chronic lung disease, or anatomic abnormalities—should prompt earlier consideration and evaluation with imaging and laboratory diagnostics.

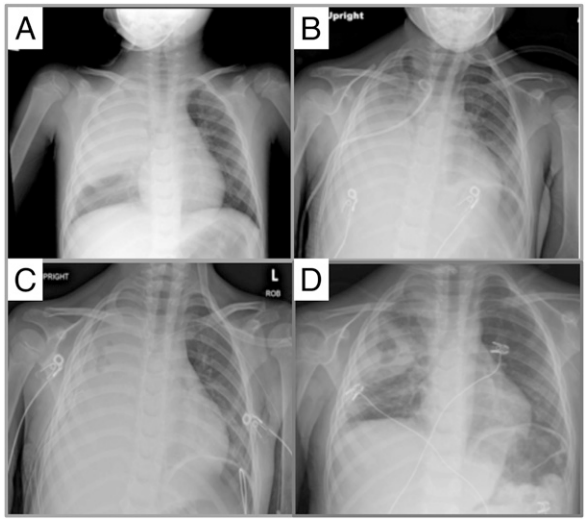

Figure 2. Radiologic progression of complicated pneumonia. A. Radiograph obtained on August 24 shows right upper lobe opacity and small right pleural effusion. B. Radiograph obtained on August 29 shows right middle and lower lobe consolidation and right-sided pleural effusion with chest tube. C. Radiograph obtained on August 30 shows worsening consolidation of the right upper lobe with minimal central lucencies, likely an underlying component of pleural effusion. A chest tube is in place. D. Radiograph obtained on September 3 shows improved aeration with lucencies within the consolidation of the right upper lobe, which suggests cavitation with small right effusion.

Indications for chest computed tomography (CT) in complicated CAP include concern for abscess or other parenchymal abnormality. Identification of pleural septations as evidence of organized pleural effusion or empyema is not reliable and does not correlate with outcomes of specific interventions (ie, chest tube drainage, intrapleural fibrinolytics, or VATS). Therefore, chest CT is not indicated on a routine basis for evaluation of mild to moderate pneumonia or even in cases of simple pleural effusion. When indicated, CT should be performed with IV contrast material to allow differentiation of thoracic structures.

The utility of lung ultrasonography in diagnosing complicated pneumonia is controversial, in both the literature and clinical practice. Lung ultrasonography in combination with initial chest radiography can demonstrate small pneumonic consolidations and allow early diagnosis of pleural effusion. (59) Evaluation of pleural effusion with chest ultrasonography may *(a)* allow localization, *(b)* demonstrate the presence of loculations or septations to further characterize empyema, and/or *(c)* guide thoracentesis and drain placement. However, the presence or absence of septations on ultrasonography does not enable prediction of a response to specific therapies or indicate a need for surgical intervention over medical management. Ultrasonography is highly user dependent, and images should be acquired and interpreted by experienced personnel.

Management of parapneumonic effusions may be solely medical or may involve a range of procedures to drain fluid and physically disrupt fibrosis and inflammation. The decision of type and timing for percutaneous drainage or surgical intervention often depends on local expertise and the individual clinical scenario. Long-term outcomes of children with pleural empyema are good, regardless of the treatment approach used during the acute phase of illness, (58) although drainage procedures that meet the criteria as outlined in national recommendations may shorten hospital stays. (13) An example of an algorithm based on these guidelines is provided in Fig 3 and is used at our institution. The goal of

drainage is to debulk disease to provide symptomatic relief and to allow antimicrobial agents to penetrate poorly perfused areas.

Criteria for surgical intervention in cases of parapneumonic effusion and empyema involve consideration of the size and duration of the effusion on images and the degree of respiratory compromise and illness severity. Patients with clinically significant hypoxemia, hypercapnia, and positive pressure requirements or with complete respiratory failure that requires intubation and ventilation will likely benefit from drainage. Other indications for drainage of the pleural space include the finding of thick pus at diagnostic thoracentesis, presence of fever and systemic illness after 5 to 7 days of therapy, presence of effusion for more than 10 days, and, in cases of toxic shock with *S aureus*, debulking disease and thus toxins.

Data are limited regarding an optimal drainage procedure; therefore, the procedure type is often based on institutional expertise, availability, and provider comfort. Options include thoracentesis, chest tube placement with or without fibrinolytics, VATS, and thoracotomy for open decortication. Randomized controlled trials in which VATS was compared to the use of a chest tube with fibrinolytics indicated that the therapies are equivalent in terms of length of stay but favored the chest tube with fibrinolytics in terms of cost and favored VATS in terms of rates of need for

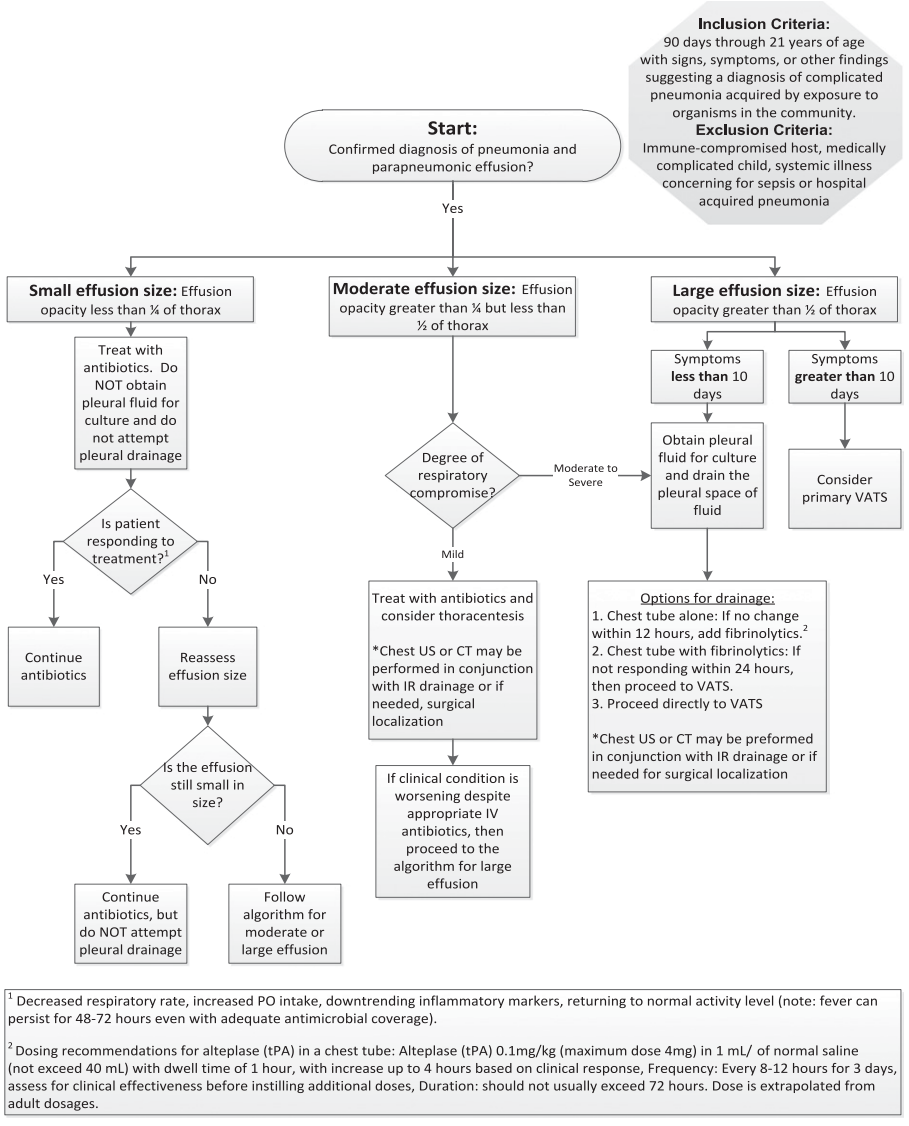

Figure 3. Management of pneumonia with parapneumonic effusion. Adapted from Bradley et al. Clinical Infectious Disease 2011 and from Complicated Community Acquired Pneumonia, Clinical Care Guidelines, Children's Hospital Colorado, updated October 11, 2016. (71) CT=computed tomography, IR=interventional radiology, IV=intravenous, PO=per os, tPA=tissue plasminogen activator, US=ultrasonography, VATS=video-assisted thoracoscopic surgery.

additional drainage procedures. (60)(61) Fibrinolytics should be considered with chest tube placement to address loculated infection and to facilitate effluent drainage. Choice of fibrinolytic is dictated in part by availability, with tissue plasminogen activator being the most commonly used in the United States. Dosages have not been fully validated in children and are variable in the literature. (60)(62) By extrapolating the adult dosages, our center uses tissue plasminogen activator of 0.1 mg/kg, given in 3 total doses (every 24 hours) with a dwell time of 1 hour (Fig 3). (6)

Lung abscesses are increasingly rare in pediatric complicated pneumonia because of increased access to care and treatment with antibiotics. They may occur as a complication within necrotizing pneumonia but can also occur in cases of more subacute scenarios. Lung abscesses are characterized by the presence of a well-defined "rim" of fibrosis around liquefaction necrosis, with or without air fluid levels. The primary parenchymal infection that leads to a lung abscess may originate hematogenously, via aspiration of oral flora, or secondary to an inhaled foreign body. Owing to their rarity in previously healthy children, lung abscesses should trigger further consideration of underlying conditions or predisposing risk factors, including aspiration (acute or chronic), foreign body, and structural abnormality, such as a pulmonary sequestration or congenital pulmonary airway malformation (formerly known as *congenital cystic adenomatoid malformation*). Immunodeficiency or chronic infection should be considered when *M tuberculosis* or endemic fungi are identified. It is difficult to differentiate lung abscess from other structurally similar complications, such as loculated pneumothorax, pneumatocele, or cavitary necrosis. Chest CT is the imaging modality of choice.

There is not strong evidence that surgical drainage of small to moderately sized abscesses improves outcomes over prolonged medical therapy alone. (63) Aspiration and culture of fluid from lung abscesses are typically reserved for patients who do not respond to appropriate antibiotics within 5 to 7 days because drainage carries risk, and most abscesses will drain spontaneously via the bronchial tree. Antibiotic treatment must often be initiated without a specimen or before identification of specific bacteria. Parenteral therapy should reflect the empirical recommendations in Fig 1, with care given to considering additional anaerobes and gram-negative organisms if aspiration is suspected as the etiologic origin of the abscess. Duration of treatment is typically 4 to 6 weeks, with at least 1 to 2 weeks of therapy after resolution of fever and until normalization of inflammatory markers. Performing repeat imaging to follow up resolution is indicated.

When lung necrosis and abscess develop near the pleural boundary, inflammation and infection can erode from the airway and enter the pleural space, creating an air leak known as a *bronchopleural fistula*. This complication is rare in pediatric pneumonia, but some single-center retrospective case reviews have indicated increasing rates associated with specific pneumococcal serotypes. (64) Care of this complication is not straightforward and requires a multispecialty approach, including surgical consultation.

Round pneumonia is a separate complication from an abscess, although also very rare. It has a distinctive radiologic pattern, described as an opaque round shape, and a study of children with round pneumonia typically demonstrated the consolidations to have well-defined borders, to be located posteriorly, and to be solitary in their distribution. Round lesions on chest radiographs should trigger consideration of a broader differential diagnosis, including fungal infection, lung abscess, and congenital or acquired pulmonary malformations such as cysts, congenital pulmonary airway malformation, or pulmonary sequestration, as well as a range of neoplasms, including lymphoma and neuroblastoma. (65) Primary pulmonary malignancies are rare in pediatrics.

Necrotizing pneumonia is an inexact term used to denote evidence of parenchymal necrosis in the lung. It is commonly a precursor for a range of complications, including lung abscess and pneumatocele. Radiologically, these changes appear as focal lucencies on chest radiographs, often with an accompanying parapneumonic effusion. CT scans, when obtained, show areas of low attenuation within the parenchyma that are attributed to liquefaction; these areas can be patchy or continuous, with an area of consolidation. Importantly, the term *necrotizing pneumonia* conveys an assessment of clinical severity out of proportion to traditional symptoms of severe pneumonia, since some of these patients progress rapidly to septic shock and respiratory failure. Necrotizing pneumonia is thought to be caused by particularly virulent bacterial strains of *S pyogenes, S pneumoniae,* or *S aureus,* particularly *S aureus* harboring Panton-Valentine leukocidin, a toxin associated with neutrophil lysis. (66) Similar to patients with lung abscess discussed earlier, most cases of necrotizing pneumonia resolve with medical treatment alone.

Antimicrobial therapy of complicated pneumonia should include coverage for *S pneumoniae* for all patients, and for very ill patients or those with influenza, therapy should include coverage for *S pyogenes* and *S aureus,* with consideration for empirical MRSA coverage. Antimicrobial agents should be narrowed as soon as possible, (67) as directed by culture results and local epidemiology (Fig 1). Two to 4 weeks of antibiotic therapy is typical for treatment of complicated pneumonia; however, there is a lack of data to support a definitive length of treatment. Length of therapy

should be determined by the clinical course and response to therapy. Parenteral antibiotics are recommended for initial therapy to optimize antimicrobial concentrations in the lung tissue and pleural fluid. The decision to transition a patient with complicated CAP to oral antibiotics is best guided by clinical response, including improved respiratory status, decreasing fever, and decreasing inflammatory markers. In a recent study, there were no differences in complications related to infection in patients treated with IV versus oral antibiotics. (61)

Severe pneumonias have other well-described associations worth mentioning that may complicate care in the acute setting. These include syndrome of inappropriate antidiuretic hormone and hemolytic-uremic syndrome (particularly with *S pneumoniae*). Another rare complication of pneumonia is empyema necessitans. This occurs when infected fluid in the chest erodes into the chest wall, causing local symptoms where the erosion occurs. Empyema necessitans is seen in more indolent infection that has gone undetected, such as with actinomycetes or mycobacteria or, rarely, inadequately treated traditional bacterial complicated pneumonia.

USE OF FOLLOW-UP RADIOLOGY

Repeat chest radiography is indicated in cases of clinical deterioration or instability after 24 to 48 hours of antibiotics. Identification of effusion or worsening infiltrate can inform decisions about broadening antibiotic coverage or consideration of pleural drainage.

In patients with a chest tube in place, standards vary from institution to institution about serial chest imaging, with some supporting daily radiographs to be acquired to be able to monitor chest tube placement, while others only repeat chest radiography in cases of clinical deterioration or possible tube malfunction.

In the outpatient realm, repeat chest radiographs are not indicated in most cases of mild, moderate, or even severe pneumonia. Because recurrent pneumonia in a specific location may suggest underlying anatomic abnormalities, chest imaging should be repeated 6 to 8 weeks after clinical resolution of pneumonia. Complete radiologic resolution of acute pneumonia occurs by 2 months in more than 90% of cases. (68) Radiographic resolution of a lung abscess and round pneumonia should be documented.

RECURRENT BACTERIAL PNEUMONIAS

Recurrent pneumonia is defined by more than 2 episodes of pneumonia in 1 year or more than 3 episodes in a lifetime. Approximately 8% of patients hospitalized for pneumonia meet these criteria. (68) Regardless of the age of the patient, recurrent pneumonia should trigger further evaluation for underlying microbiological, functional, anatomic, and chronic disease factors. (69)(70) Conducting radiologic follow-up at 2 months to distinguish persistent from recurrent pneumonias is reasonable to assist in determination of a differential diagnosis and to direct further imaging, laboratory testing, and procedural evaluation. (68)

Diagnosis of recurrent or persistent pneumonia is confirmed by means of persistent findings of opacification on chest radiographs. Further evaluation of persistent localized consolidation starts with direct visualization and sampling of the affected region by means of flexible bronchoscopy and bronchoalveolar lavage. Cytologic analysis and culture of the fluid can demonstrate persistent pathogens or suggest aspiration if a high burden of lipid-laden macrophages is seen. CT with contrast material is used to evaluate the parenchyma and distal airways. Pathologic changes such as airway bronchiectasis, cystic changes, and congenital sequestrations and infectious complications, such as cavitary abscesses or pleural effusion with loculations, can direct further evaluation and therapies.

Consolidations that persist and/or recur in the same area suggest a persistent pathogen or a focal anatomic abnormality. Persistent infectious causes include less common pathogens, such as tuberculosis, endemic fungal infections, *Actinomyces*, and nocardiosis. Focal anatomic abnormalities include obstructing lesions of the airway, including a retained foreign body, compressing lymph node or tracheal growth, compressing vascular rings or slings, and dynamic obstruction due to compressive tracheal tracheomalacia or bronchomalacia. Other focal abnormalities lead to poor clearance, such as segmental bronchiectasis, tracheal bronchus, pulmonary sequestration, or airway cyst, can lead to poor mucociliary clearance. *Right middle lobe syndrome* describes the presence of recurrent right middle lobe consolidations that likely occur because of poor collateral ventilation due to the acute angle to that lobe from the right mainstem bronchus. This lobe is more susceptible to atelectasis, aspiration, and lymph node compression.

Infiltrates that recur, but in anatomically distinct areas, invoke concern for immune defects or difficulties with airway clearance. Asthma causes recurrent atelectasis and infiltrates due to airway inflammation and mucus plugging. Associated symptoms of airway reactivity, including nighttime cough and wheeze that is worse with activity, can suggest uncontrolled asthma. Other defects and/or diseases to consider include cystic fibrosis, ciliary defects, surfactant protein defects, HIV, and congenital immune deficiencies.

A negative newborn screening test finding for cystic fibrosis should not deter the provider from pursuing a sweat chloride test. If results are positive, the provider should contact the nearest cystic fibrosis center for further evaluation. Another unusual cause to consider is recurrent bacterial seeding of the lungs in patients with cardiac defects, especially valvular disease and septal defects.

The tempo of the evaluation is dictated by the severity of the illness. For example, chronic hypoxemia, hypoventilation, weight loss, persistent fevers, anemia, leukopenia or leukocytosis, or digital clubbing would all trigger a more aggressive approach. Recurrent pneumonia evaluation can be aided by referral to specialists, including an infectious disease specialist, pulmonologist, and otolaryngologist.

DIAGNOSIS AND MANAGEMENT OF PNEUMONIA IN PATIENTS WITH NEUROMUSCULAR DISEASE

Pneumonia in patients with neuromuscular disease (including spinal muscular atrophy and Duchenne muscular dystrophy) requires additional diagnostic considerations and management recommendations. Because of persistent muscle weakness that leads to restrictive lung physiology, most patients with neuromuscular disease have decreased total lung capacity. Compounding this physiology, these patients often have impaired cough function at baseline, and bulbar weakness may increase the risk for aspiration into the lungs. Thus, these patients are at high risk for developing pneumonia in general, and it can be community acquired, hospital acquired, or health care associated. Once infected, the decreased pulmonary reserve accelerates deterioration to respiratory failure in these patients. (71)

Initial diagnosis and stabilization should include early consideration of chest radiography, electrocardiography, blood gas analysis, and assessment of electrolyte levels. Patients with spinal muscular atrophy are at risk for hypoglycemia if they receive nothing by mouth for 4 to 6 hours and should be started on IV dextrose, regardless of hydration status, if they receive nothing by mouth. Empirical antibiotic coverage includes traditional CAP coverage, but based on clinical history, risk factors, and past microbiology, may need to be expanded to cover anaerobes, gram-negative findings (including resistant gram-negative findings), and/or MRSA. A diagnostic bronchoalveolar lavage should be considered in intubated patients. Supportive care with oxygen and biphasic noninvasive ventilation is often essential, being mindful that increasing fractional index of oxygen alone or instituting continuous positive airway pressure may blunt the hypoxic respiratory drive response in the setting of chronic hypercapnia. Airway clearance regimens that include mechanical insufflation-exsufflation are important in aiding mucus clearance and airway recruitment. Admission criteria for patients with neuromuscular disease are more conservative, requiring assessment of each patient in relationship to his or her pulmonary baseline. If the patient requires continuous nasal intermittent positive pressure ventilation, new or increased oxygen requirement, or new or worsened hypercapnia or if suctioning and cough augmentation requirements are frequent, the patient should be admitted with consideration of critical care services. Consider cardiac dysfunction as either primary or secondary to respiratory distress, since many of these patients develop cardiomyopathy (particularly with Duchenne muscular dystrophy).

Summary

- On the basis of some research evidence, as well as consensus, treatment of uncomplicated community-acquired pneumonia can reasonably be achieved in 7 days or less. (43)(44)(45)
- On the basis of strong evidence, narrow-spectrum treatment is the preferred therapy in almost all settings. (6)(33)(35)
- On the basis of consensus and moderate evidence, in this era of antimicrobial resistance, efforts to obtain a specimen for pathogen identification may be beneficial.
- On the basis of strong evidence, consideration of surgical and/or procedural management of complicated pneumonia should be based on size of effusion and clinical severity. (6)(13)
- On the basis of moderate evidence, patients with recurrent pneumonia require further evaluation.
- On the basis of expert consensus, patients with a neuromuscular disorder are particularly susceptible to severe disease and more resistant pathogens and may require broader antibiotic coverage and aggressive airway clearance.

References for this article are at http://pedsinreview.aappublications.org/content/38/9/394.

PIR QUIZ

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1. A previously healthy 13-month-old girl who lives in Arizona is brought to the office with a 2-day history of fever and increasing cough. Her mother states that the child has continued to breastfeed and has a normal number of wet diapers. Her immunizations are up to date. She is alert and mildly ill appearing. Her temperature is 102.1°F (38.9°C), heart rate is 142 beats/min, respiratory rate is 50 breaths/min, and oxygen saturation is 95% on room air. At physical examination, there is no grunting or chest retractions. There are crackles heard over the right lung base. The remainder of the examination findings are normal. She has no known allergies. Which of the following is the most likely pathogen?
    A. *Bordetella pertussis.*
    B. *Haemophilus influenzae* type B.
    C. *Histoplasma capsulatum.*
    D. *Mycoplasma pneumoniae.*
    E. *Streptococcus pneumoniae.*

2. For the same 13-month-old girl in the previous question, which of the following is the most appropriate next step in treatment?
    A. Admit her to the hospital for intravenous (IV) ceftriaxone and vancomycin.
    B. Admit her to the hospital for IV ceftriaxone and levofloxacin.
    C. Outpatient amoxicillin.
    D. Outpatient azithromycin.
    E. Outpatient cefdinir.

3. A previously healthy 18-month-old boy is admitted to the hospital after presenting to the emergency department with a 3-day history of fever and cough. His oral intake is decreased. His immunizations are up to date. He has no known allergies. At examination, he is moderately ill appearing. His temperature is 102.3°F (39.0°C), his heart rate is 148 beats/min, his respiratory rate is 48 breaths/min, and his oxygen saturation is 88% on room air with subcostal retractions. Supplemental oxygen is administered, and his oxygen saturation increases to 98%. There are crackles at the left lung base. A chest radiograph shows a focal left lower lobe consolidation with a small parapneumonic effusion. Blood cultures are pending, and a viral respiratory screen yields negative results for viral pathogens. Which of the following is the most appropriate next step in management?
    A. Chest tube placement and IV ceftriaxone and vancomycin.
    B. IV ampicillin.
    C. IV ceftriaxone and oral azithromycin.
    D. IV ceftriaxone and vancomycin.
    E. Oral levofloxacin.

4. A 4-year-old boy is admitted to the hospital with an 8-day history of increasing cough and 5 days of fever. He has global developmental delay and spastic quadriplegia. A chest radiograph shows an oval cystic lesion in the right middle lobe with an air-fluid level. Blood cultures are pending. In addition to empirical antibiotics, which of the following is the most appropriate next step in management?
    A. Chest computed tomography with contrast material.
    B. Lateral decubitus chest radiography.
    C. Swallow study.
    D. Sweat chloride assay.
    E. Thoracotomy.

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5. A 2-year-old girl is admitted to the hospital with fever for 3 days with a right middle lobe consolidation. She has had a cough for the past 3 months with 2 prior admissions for pneumonia, with the same location of the infiltrate noted on chest radiographs. She clinically improved with a course of antibiotics. Prior to 3 months ago, she was healthy. She has been growing at the 75th percentile for height and weight. She has not been noted to wheeze. Which of the following is the most likely diagnosis?

   A. α_1-antitrypsin deficiency.
   B. Chronic granulomatous disease.
   C. Cystic fibrosis.
   D. Retained foreign body.
   E. Wiskott-Aldrich syndrome.

Additional Resources for Pediatricians

AAP Textbook of Pediatric Care, 2nd Edition

- Chapter 315: Pneumonia - https://pediatriccare.solutions.aap.org/chapter.aspx?sectionid=124995320&bookid=1626

Point-of-Care Quick Reference

- Pneumonia - https://pediatriccare.solutions.aap.org/content.aspx?gbosid=165559

Parent Resources from the AAP at HealthyChildren.org

- Pneumonia: https://www.healthychildren.org/English/health-issues/conditions/chest-lungs/Pages/Pneumonia.aspx

For a comprehensive library of AAP parent handouts, please go to the *Pediatric Patient Education* site at http://patiented.aap.org.