Community-Acquired Pneumonia in Children

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Abstract: Community-acquired pneumonia (CAP) is the most common cause of childhood mortality globally. In the United States, CAP is a leading cause of pediatric hospitalization and antibiotic use and is associated with substantial morbidity. There has been a dramatic shift in microbiological etiologies for CAP in children over time as pneumococcal pneumonia has become less common and viral etiologies have become predominant. There is no commonly agreed on approach to the diagnosis of CAP in children. When indicated, antimicrobial treatment should consist of narrow-spectrum antibiotics. In this article, we will describe the current understanding of the microbiological etiologies, clinical presentation, diagnostic approach, risk factors, treatment, and future directions in the diagnosis and management of pediatric CAP.

Key Words: community-acquired pneumonia, hypoxia, diagnosis

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TARGET AUDIENCE

This continuing medical education (CME) activity is intended for physicians, advanced practice providers, and emergency medical service providers who provide emergency clinical care for children.

LEARNING OBJECTIVES

After participating in this activity, the learner should be better able to:

1. Describe the epidemiology, common pathogens, and risk factors for community-acquired pneumonia (CAP) in children.
2. Explain the diagnostic approach to children with possible CAP, including the limitations of clinical features and radiographic imaging.
3. Discuss appropriate treatment strategies for children with CAP and future directions related to the diagnostic and prognostic approach for children with CAP.

INTRODUCTION AND EPIDEMIOLOGY

Community-acquired pneumonia (CAP) is the most common cause of mortality among infants and children aged 1 to 59 months worldwide.[1] Each year, CAP is implicated in the deaths of more than 800,000 infants and children.[1] Owing to limited diagnostic and therapeutic resources and higher rates of childhood malnutrition, most of these deaths occur in low- and middle-income countries.[2,3] The CAP also leads to substantial morbidity among children in high-income countries. In the United States, CAP accounts for approximately 2 million outpatient visits, 2.2% of all pediatric emergency department visits, and more than 100,000 hospitalizations among children each year.[4–6]

Varied definitions of CAP exist,[7–9] but it is broadly defined as an infection of the lower respiratory tract that was acquired through contact with another individual outside of health care settings.[10] This broad definition has considerable overlap with other lower respiratory tract disorders, leading to overdiagnosis of CAP, and does not differentiate bacterial from viral etiologies. One potential result of overdiagnosis of CAP is excess antibiotic use. A more specific definition of CAP is "the presence of fever, acute respiratory symptoms, or both, plus evidence of parenchymal infiltrates on chest radiography" that was acquired through community spread, and not through contact in a health care facility.[11] In addition to varied definitions, there is also variation in the diagnostic approach to CAP in children. Clinicians typically rely on clinical examination findings and chest radiography to establish the diagnosis.[12–14]

Historically (before the COVID-19 pandemic), CAP was a seasonal disease among children, with the annual peak in the winter months and the nadir in summer months in the United States.[15] Although not entirely understood, this seasonality is thought to be due to indoor crowding that occurs in winter months, leading to more exposure to respiratory pathogens.[15] However, during the COVID-19 pandemic, after social distancing and masking recommendations were lifted, there was a dramatic recurrence of CAP among children in the United States during summer months that historically have been characterized by few cases of CAP. It remains unclear if the seasonality of CAP will return as the COVID-19 pandemic wanes.

In this article, we will describe the current understanding of the microbiological etiologies, clinical presentation, diagnostic approach, risk factors, treatment, and future directions in the diagnosis and management of pediatric CAP. Most of our discussion focuses on CAP presentation in high-income countries. However, given the global importance of pediatric pneumonia, we address issues relevant to the global population of children with pneumonia where noted.

Microbiological Etiologies of CAP

The most common pathogens causing CAP vary by age (Fig. 1) and the presence of underlying medical conditions. In the United States and other areas where the pneumococcal conjugate vaccine (PCV) is widely available, there has been a dramatic shift in microbiological etiologies for CAP since the introduction of the vaccine. Pneumococcal pneumonia has become less common and viral etiologies have become predominant.[16] The PCV13 was introduced in the United States in 2011 and is recommended for children at ages 2, 4, 6, and 12 to 15 months.[17] The PCV13 covers only 13 of the more than 100 serotypes of pneumococcus; trials are underway to assess the efficacy of pneumococcal vaccines with broader serotype coverage.[18,19]

Since the introduction of the PCV7 and PCV13 vaccines, large surveillance studies conducted in the United States and

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A

Among children aged 0-4 years with pathogens detected in the United States, The Gambia, Mali, Kenya, Zambia, South Africa, Bangladesh, and Thailand (N=3,308)

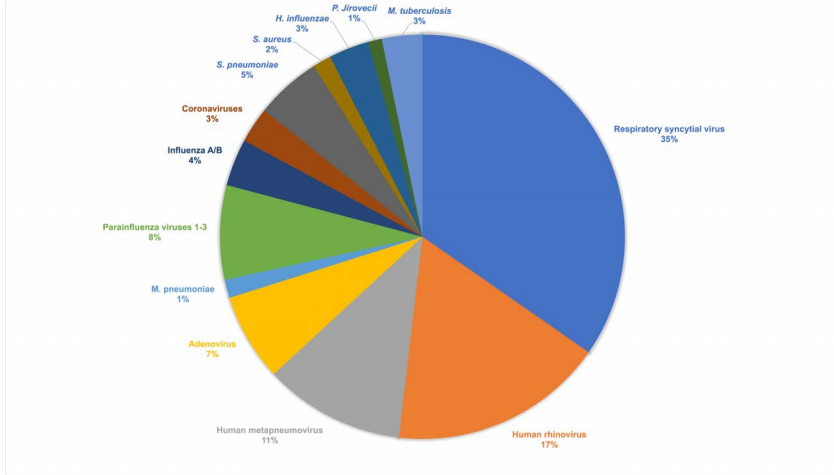

Figure adapted from the PERCH Study Group. *Lancet*. 2019. and Jain S, et al. *N Eng J Med*. 2015.

B

Among children aged 5-17 years hospitalized with CAP in the US (N=683)

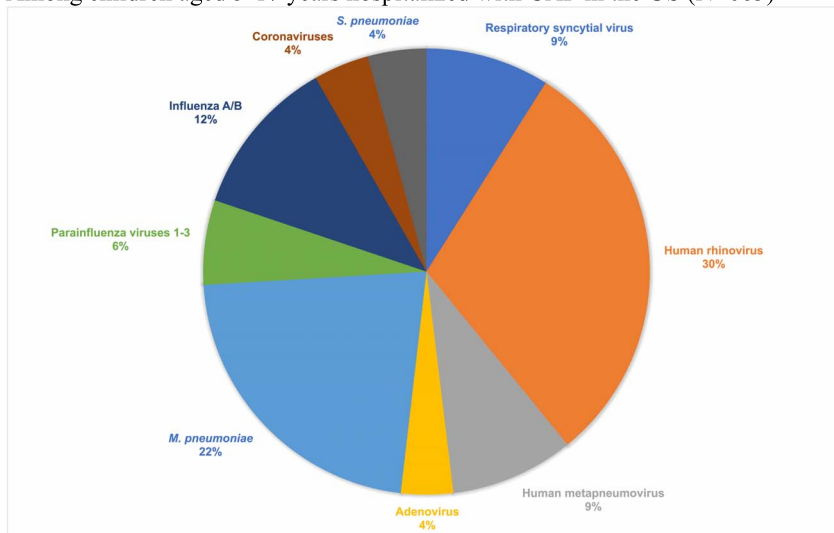

Figure adapted from the Jain S, et al. *N Eng J Med*. 2015.

FIGURE 1. Distribution of detected pathogens in children with CAP in the United States and globally (A) among children aged 0 to 4 years with pathogens detected in the United States, The Gambia, Mali, Kenya, Zambia, South Africa, Bangladesh, and Thailand (N = 3308). Figure adapted from the PERCH Study Group. *Lancet*. 2019. and Jain S, et al. *N Eng J Med*. 2015. B, Among children aged 5 to 17 years hospitalized with CAP in the United States (N = 683). Figure adapted from the Jain S, et al. *N Eng J Med*. 2015.

globally suggest that viral etiologies of CAP predominate in young children.[5,20–24] Respiratory syncytial virus, human rhinovirus, and human metapneumovirus are the most common viral etiologies of CAP in infants and children younger than 5 years both in the United States and globally. Similarly, in children 5 to 12 years, viruses including respiratory syncytial virus, human rhinovirus, and human metapneumovirus account for approximately half of cases of CAP in which a pathogen is detected. *Mycoplasma pneumoniae* is the most commonly detected bacterial pathogen leading to CAP in school-aged children in the United States,[5,25] but viruses also predominate in adolescents. When bacteria are detected in children with CAP, *Mycoplasma*, *Streptococcus pneumoniae*, *Staphylococcus aureus*, streptococcus, and *Haemophilus influenzae* are common pathogens in the United States.[5] Although it is likely that these studies underestimate the prevalence of typical bacterial pathogens given the diagnostic challenges of detecting these organisms, the importance of viral etiologies in children with CAP is substnatial.

Children with underlying conditions and resulting immunosuppression are at greater risk of bacterial and fungal etiologies for CAP. Special consideration should be made for children with

sickle cell disease who are predisposed to infections with encapsulated bacterial organisms such as *S. pneumoniae* and children undergoing chemotherapy who may be at risk for invasive *S. pneumoniae*, *S. aureus*, *Pseudomonas aeruginosa*, mycobacteria, *Pneumocystis jirovecii*, and fungal disease. Children with cystic fibrosis are more commonly infected with *H. influenzae* type B, *S. aureus*, *P. aeruginosa*, and *Stenotrophomonas maltophilia*.

Clinical Presentation of Children With CAP

There is no uniform clinical presentation of children with CAP. Common signs and symptoms include hypoxemia, increased work of breathing, cough, fever, and tachypnea. Other presenting symptoms may include abdominal pain and, occasionally, emesis. Hypothermia and altered mental status may be seen in severe CAP. On physical examination, children with CAP may exhibit rales/crackles on examination of the chest, focal diminished breath sounds, retractions, nasal flaring, grunting, and head bobbing. The presence of wheezing has been negatively associated with radiographic CAP in several studies.[26–28] A substantial challenge is the varied use of terminology to describe, and limited reliability of, physical findings associated with, pediatric CAP.[29]

Diagnosis of CAP in Children

A commonly agreed-on approach to diagnose children with CAP is lacking; accordingly, there is debate on whether CAP is a clinical or radiographic diagnosis.[30] The only true etiologic reference standard for CAP is lung biopsy, which is invasive, rarely necessary, and should be reserved only for children with severe CAP in whom traditional therapies have failed.[7]

Current recommendations from the Infectious Diseases Society of America (IDSA) and the World Health Organization (WHO) encourage the use of clinical impression, presence of tachypnea or retractions, and auscultatory findings to establish the diagnosis of CAP in children.[7–9] However, recent studies have raised questions about the diagnostic accuracy of commonly used clinical examination findings such as tachypnea, retractions, and fever when used in isolation to diagnose CAP.[12–14]

Several systematic reviews including US and global studies, including a study that pooled data from large populations of children, concluded that no single sign or symptom is sufficiently diagnostic of radiographic CAP (Table 1).[12–14] Hypoxemia is most associated with the presence of radiographic CAP, but does not discriminate sufficiently to be used in isolation. Clinical prediction rules that combine signs and symptoms likely improve precision in the diagnosis of children who are evaluated for CAP (Table 2).[26,27,32–36] In an external validation study, only 1 clinical prediction rule had fair discriminatory value in identifying children at risk of radiographic CAP.[31] This rule uses a combination of age, oxygen saturation level, presence of fever, rales, and wheezing. However, no clinical prediction rule for radiographic CAP has been implemented, making their impact on clinical care unclear.

Chest Radiography for CAP in Children

Current IDSA and WHO guidelines do not recommend routine use of chest radiography for the diagnosis of CAP in children who are well enough to be managed as outpatients.[7–9] However, current IDSA guidelines recommend that chest radiography be performed on children with hypoxemia, significant respiratory distress, failure of empiric antibiotic therapy, and those hospitalized for CAP.[7] Due to limited accessibility in many settings, WHO guidelines only recommend the use of chest radiography for CAP if available.

Although chest radiography is not routinely recommended, rational use may improve antibiotic stewardship given its high negative predictive value,[37] particularly among children with moderate clinician suspicion for CAP.[38] Chest radiography may also be useful when the diagnosis is uncertain. A recent study demonstrated a decline in overall use of chest radiography for CAP in US pediatric emergency departments, although it was still used in 80% of encounters in which children were diagnosed with CAP.[39]

Chest radiography has important limitations. With the exception of the presence of focal alveolar infiltrates, there is poor-to-fair interrater agreement among pediatric radiologists for findings that are commonly used to diagnose radiographic CAP.[40] Moreover, chest radiography cannot be used to distinguish viral from bacterial etiologies in children with CAP.[5] Although it is often taught that radiographic CAP trails clinical findings, one prospective study suggests that negative chest radiography for CAP has excellent negative predictive value.[37]

Lung Ultrasound for CAP in Children

Lung ultrasound may prove useful in the diagnosis of CAP. The use of lung ultrasound may be more efficient than chest radiography. Lung ultrasound findings consistent with CAP include 3 or more B-lines (ie, well-defined, hyperechoic lines that move in concert with the lungs), air bronchograms that are dynamic (ie, move with respiration), and hypoechoic, liver-like (ie, hepatization) patterns in the lungs that suggest a focal consolidation (Fig. 2).[41] The findings from a meta-analysis of 5 studies evaluating the test characteristics of lung ultrasound for CAP in children in the hands of skilled sonographers demonstrated a sensitivity of 96% and specificity of 93% with chest radiograph used as the reference standard.[42] However, most studies evaluating point-of-care ultrasound use in the emergency department only included pediatric emergency medicine providers with special training in ultrasonography.[43,44] The accuracy of lung ultrasound in the hands of unskilled sonographers remains unknown. Moreover, studies assessing clinical outcomes for children with CAP identified with lung ultrasound are lacking, but may help inform its potential utility in the evaluation of children with suspected CAP.

Laboratory Evaluation of Children for CAP

Routine laboratory testing is not indicated for children being evaluated for CAP. Blood cultures should not be obtained in healthy, immunized children who are treated in the outpatient setting.[7] The IDSA strongly recommends the collection of blood cultures in children requiring hospitalization for "presumed moderate-to-severe bacterial CAP."[7] However, blood cultures obtained in children who are hospitalized demonstrate low pathogen yields (ie, 2.5%–7%),[45,46] infrequent identification of bacteria resistant to commonly used penicllins,[46] and may be low-yield among children admitted to non-intensive care unit settings or those with less severe disease.[47] Several studies have demonstrated the utility of an algorithm to guide blood culture use in those at higher risk for bacteremia.[48,49] In these algorithms, children were considered high-risk if they were younger than 6 months old or not fully immunized, had central venous catheters, were immunosuppressed, toxic-appearing or admitted to an intensive care unit, had chronic medical problems, or had effusions or empyemas identified on chest radiography.

Elevated and low white blood cell counts are neither sensitive nor specific for the diagnosis,[50,51] do not differentiate bacterial from viral etiologies,[52] nor predict outcomes of children with CAP.[53] The acute phase reactant C-reactive protein has some

TABLE 1. Summary of Test Characteristics of Symptoms and Signs for Radiographic CAP Both in the United States and Globally

	Rambaud-Althaus C, et al. *Lancet Infect Dis.* 2015.[14]		Shah SN, et al. *JAMA*. 2017.[12]		Rees CA, et al. *BMJ Glob Health*. 2020.[13]	
	Sensitivity	**Specificity**	**Sensitivity**	**Specificity**	**Sensitivity**	**Specificity**
Symptoms						
Fever (caregiver-reported)	0.94	0.12	0.92	0.14	0.82	0.17
Cough	0.96	0.14	0.88	0.25	0.77	0.08
Difficulty breathing	0.60	0.52	0.37	0.69	0.70	0.08
Rapid breathing	0.79	0.31	—	—	0.79	0.22
Poor feeding	0.64	0.52	0.58	0.60	0.29	0.76
Vomiting	0.36	0.70	0.27	0.76	0.12	0.87
Signs						
Nasal flaring	0.47	0.73	0.36	0.84	0.18	0.85
Grunting	0.24	0.87	0.13	0.95	0.06	0.96
Temperature >38°C	0.56	0.55	0.48	0.67	0.54	0.60
Respiratory rate >40 breaths/min	0.78	0.51	0.79	0.51	—	—
Rales	0.49	0.45	0.43	0.64	0.82	0.26
Ronchi	0.19	0.67	0.16	0.83	—	—
Decreased breath sounds	0.22	0.76	0.25	0.72	—	—
Wheezing	0.22	0.75	0.16	0.83	0.29	0.73
Lower chest indrawing	0.48	0.72	0.38	0.80	0.74	0.15

discriminatory value in differentiating bacterial from viral etiologies of CAP in children[51,54] and may be used to follow response to treatment.[7] Erythrocyte sedimentation rate has poor sensitivity and specificity in the diagnosis of CAP in children.[55] Procalcitonin may aid in the decision to treat a child with suspected CAP with antibiotics. A low serum procalcitonin level in children (ie, <0.1–0.25 ng/mL) is highly sensitive and has demonstrated high negative predictive value in determining typical bacterial etiology in children evaluated for CAP.[5] Trials assessing the efficacy of the integration of serum procalcitonin into decision-to-treat algorithms in adults with CAP have demonstrated lower rates of antibiotic administration without differences in treatment failure rates, length of hospitalization, or need for intensive care unit admission.[56] In children, several small clinical trials have similarly demonstrated lower rates of antibiotic administration to children with suspected CAP in those using a procalcitonin-guided algorithm without differences in hospitalization duration.[57,58]

The current IDSA guidelines recommend against the routine use of white blood cell count, C-reactive protein, and procalcitonin in the diagnosis of CAP.[7] However, the guideline suggests that C-reactive protein may be helpful to trend response to therapy for children hospitalized with CAP. Further research is needed to better understand the role of C-reactive protein, procalcitonin, and other serum biomarkers in the management of pediatric CAP. No biomarker should be used in isolation without considering clinical assessment of the child.

Risk Factors for Developing CAP and Disease Severity

Children who have underlying pulmonary diseases, including chronic lung disease and bronchopulmonary dysplasia, are at higher risk for developing CAP due to difficulty clearing pulmonary secretions, which can lead to hypoxemic respiratory failure. In addition, children with defects in humoral immunity may have greater risk of developing severe CAP due to inability to clear pathogens.

Definitions of "severe CAP" vary based on geography and intended use. In high-income countries, several definitions have been proposed, with most including measures of substantial morbidity such as use of positive-pressure ventilation, chest drainage procedures, or vasoactive infusions.[59,60] The 2011 IDSA CAP guidelines define respiratory distress as the presence of age-adjusted tachypnea, dyspnea, retractions, grunting, nasal flaring, apnea, altered mental status, or pulse oximetry less than 90% in room air.[7] Most relevant to low- and middle-income countries, the WHO defines severe CAP as cases in which a child with CAP cannot drink, has persistent vomiting, convulsions, lethargy, stridor, or severe malnutrition.[61]

Several prediction models have been developed to identify children with CAP who are at risk of developing severe disease in the United States and globally. The results of a systematic review published in 2018 that included studies from multiple settings (ie, high-income and low- and middle-income countries) suggest that hypoxemia, altered mental status, age 3 to 6 months, and dyspnea are clinical factors most associated with severe CAP in children.[62] In addition, children with multilobar infiltrates or moderate-to-large pleural effusions identified radiographically had greater risk of developing severe CAP. An important limitation of this review is the varied definitions for severe CAP across studies. A more recent clinical prediction rule from a cohort of more than 1100 children aged 3 months to 18 years who presented to the emergency department with clinically suspected CAP identified the following risk factors for severe CAP[59]: respiratory rate greater than 95th percentile for age, blood pressure less than 5th percentile for age, impaired oxygenation, presence of retractions, prolonged capillary refill (ie, ≥3 seconds), chest radiograph findings suggestive of atelectasis or pneumonia, and pleural effusion. Further research is ongoing to validate these more recent prediction rules. In low- and middle-income countries, several clinical prediction rules have been developed to identify infants and children with CAP who are at risk of death.[63–66] However, although most have performed suboptimally when externally validated,[67] one clinical prediction rule using data from children in 20 low- and

TABLE 2. Summary of Variables Included in Existing Clinical Prediction Rules to Identify Children at Risk of Radiographic CAP in the United States and Globally

Reference	Parameters	Original Derivation Area Under the Curve (AUC) (95% CI)	External Validation AUC[31] (95% CI)
Ramgopal S, et al. *Pediatrics*. 2022.[26]	Age in years Fever duration Age-adjusted tachypnea Focal decreased breath sounds Rhinorrhea Wheezing	0.85 (0.82–0.88)	—
Lipsett SC, et al. *Pediatr Infect Dis J*. 2022.[27]	Age in years Triage oxygen saturation Presence of fever Presence of rales Presence of wheeze	0.71 (0.68–0.75)	0.72 (0.68–0.75)
Oostenbrink R, et al. *Eur J Emerg Med*. 2013.[32]	Ill-appearance Tachypnea Decreased oxygen saturation Elevated C-reactive protein	0.79 (0.69–0.89)	0.55 (0.49–0.60)
Lynch T, et al. *Pediatrics*. 2004.[33]	Fever Decreased breath sounds Crackles Tachypnea	0.67 (95% CI not reported)	0.54 (0.50–0.58)
Mahabee-Gittens EM, et al. *Clin Pediatr*. 2005.[34]	Age in months Respiratory rate ≥50 Oxygen saturation ≤96% Nasal flaring	0.81 (0.75–0.87)	0.49 (0.42–0.55)
Bilkis MD, et al. *Pediatr Emerg Care*. 2010.[35]	Grunting Cough Rales Decreased breath sounds Vomiting	0.77 (0.72–0.84)	-
Chan FYY, et al. *Am J Emerg Med2*. 2020.[36]	Fever duration Chills Nasal symptoms Abnormal chest examination oxygen saturation ≤ 96% or tachypnea	0.73 (0.65–0.81)	-

CI indicates confidence interval.

middle-income countries demonstrated good discriminatory value in external validation.[68] To date, none of these have been implemented or used widely in clinical practice.

Disposition

Although rates of hospitalization for CAP in children vary substantially across hospitals in the United States,[69] there are several indications for hospitalization recommended by the IDSA.[7] These include age younger than 3 months, oxygen saturation of less than 90%, age-adjusted tachypnea, respiratory distress (ie, retractions, nasal flaring, or dyspnea), apnea, signs of dehydration, concern for virulent organisms such as methicillin-resistant *S. aureus*, or if there are inadequate resources to observe a child for deterioration at home. Once validated, CAP risk prediction models are likely to help improve disposition decision-making.

Treatment

In general, antibiotics should be administered to children with suspected bacterial CAP. However, there are currently no available tests in routine clinical practice to distinguish bacterial from viral etiologies. If there is a high suspicion for viral CAP in a well-appearing child, IDSA guidelines suggest that clinicians may consider withholding antibiotics.[7] This includes preschool-aged children well enough to be managed as outpatients without any clinical red flags (eg, hypoxemia, significant tachypnea, or dyspnea).

For children in whom bacterial etiologies are suspected, antibiotic therapy should be narrow spectrum and targeted toward common bacterial pathogens. For fully immunized children without severe CAP who do not require inpatient treatment high-dose amoxicillin (ie, 90 mg/kg/day divided twice daily) is the recommended treatment regimen.[7] Amoxicillin monotherapy provides adequate coverage for the most common bacterial pathogen,

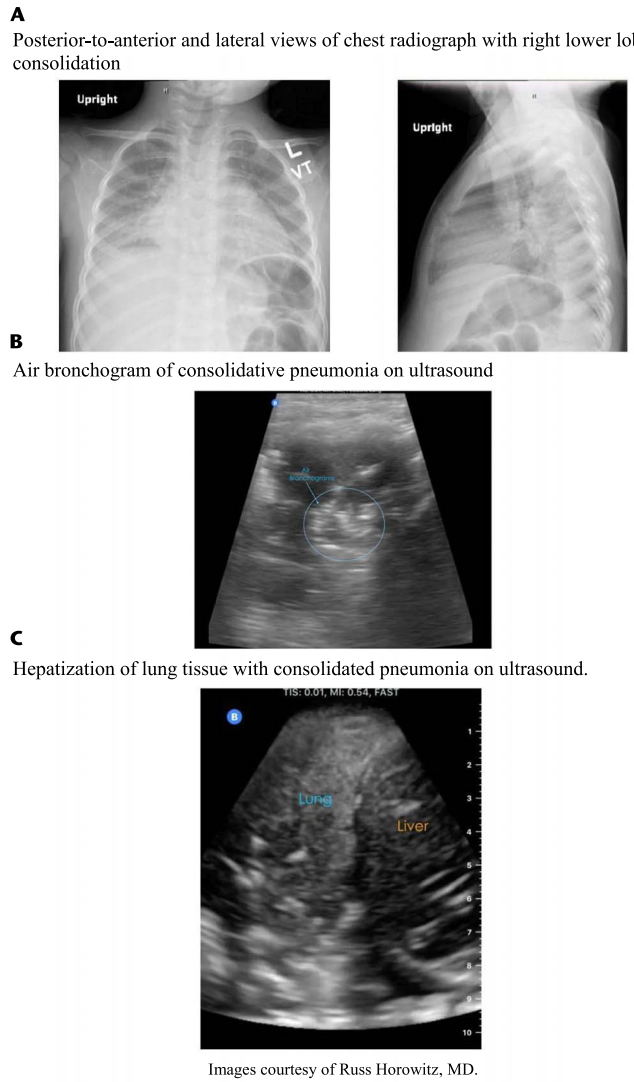

FIGURE 2. Chest radiography and lung ultrasound findings in a child with CAP (A). Posterior-to-anterior and lateral views of chest radiograph with right lower lobe consolidation (B). Air bronchogram of consolidative pneumonia on ultrasound (C). Hepatization of lung tissue with consolidated pneumonia on ultrasound. Images courtesy of Russ Horowitz, MD.

S. pneumoniae. Second- or third-generation cephalosporins, levofloxacin, or oral linezolid may be prescribed for those who have allergies to amoxicillin.[7] Traditionally, antibiotics have been administered for 7 to 10 days for children with CAP. However, one randomized trial found that a course of high-dose amoxicillin for 5 days is equally effective as a course of 10 days of high-dose amoxicillin.[70] In another recent randomized trial, children with CAP who were treated with 5 days of high-dose amoxicillin had better clinical response to treatment, more rapid resolution of symptoms, and less colonization with antibiotic-resistant bacteria after treatment than those treated with a 10-day course.[71] In another trial of more than 800 pediatric outpatients with clinically diagnosed CAP in the United Kingdom and Ireland, low-dose amoxicillin (ie, 30–50 mg/kg/day) and treatment duration of 3 days were noninferior to high-dose amoxicillin (ie, 70–90 mg/kg/day) and treatment duration of 7 days.[72] Taken together, these trials suggest that a course of antibiotics of 3 to 5 days is likely sufficient in children with mild CAP being treated as outpatients.

Macrolides (eg, azithromycin) may be considered for school-aged children and adolescents in whom there is suspicion for *Mycoplasma,* which is more common in these age groups.[5,25] However, macrolide monotherapy does not provide adequate coverage for *S. pneumoniae* and should not be prescribed routinely, particularly for hospitalized patients.[73]

The 2011 IDSA guidelines recommend that most children who require hospitalization for CAP should be treated with narrow-spectrum parenteral antibiotics. For fully immunized children aged older than 3 months who have uncomplicated CAP, intravenous ampicillin is recommended.[7] Children who are not immunized, or partially immunized, and those who require intensive care for CAP should be treated with third-generation cephalosporins (eg, ceftriaxone).[7] Macrolides may be added should a child with CAP have a suspected atypical pathogen or not improve with empirical ampicillin.[7] Intravenous vancomycin may be added for severely ill-appearing children and those who have empyemas or sepsis.

Future Directions

Accurate and efficient approaches to differentiate bacterial from viral etiologies are urgently needed for children with CAP. Although currently not widely available for clinical use, multiplex panels based on host response gene expression signatures may be able to discriminate viral from bacterial etiologies for acute respiratory tract infections among children.[74,75] In addition, serum protein assays are under development that assess host immune response proteins, and may also help distinguish viral from bacterial infections.[76,77] Such approaches, once available at the bedside, may advance differentiating viral from bacterial CAP. Although not widely available for clinical use, proadrenomedullin, a vasodilatory peptide with both antimicrobial and antiinflammatory properties, has demonstrated good discriminatory value in identifying children who develop severe CAP.[78] However, studies are needed to validate its performance and assess its use, along with other newer biomarkers, on clinical outcomes among children evaluated for CAP. Lastly, clinical prediction rules to identify children at risk of severe CAP are being further developed and externally validated and, if they demonstrate adequate discriminatory value, may be used in the future to better assign risk of severe CAP among children evaluated in the emergency department.

CONCLUSIONS

The CAP is a common cause of morbidity and mortality in children globally. Although microbiological pathogens vary by age, viral pathogens predominate as the etiology of CAP in children. However, evidence informing the need to treat a child with antibiotics or not is still emerging. When treatment is indicated for suspected bacterial CAP, narrow-spectrum antibiotics (ie, amoxicillin for children discharged home, ampicillin for those admitted with uncomplicated CAP) should be administered. Hospitalization is indicated in children with CAP requiring intravenous fluids, supplemental oxygen or respiratory support or those with, or at substantial risk of developing complicated CAP. Future advances in diagnostics and risk stratification may help better identify children at risk of severe outcomes.

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