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ANTIBIOTIC RESISTANCE IN PEDIATRIC COMMUNITY-ACQUIRED PNEUMONIA: CURRENT CHALLENGES IN DIAGNOSIS AND TREATMENT

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ABSTRACT Background: Community-acquired pneumonia (CAP) remains a major cause of morbidity, hospitalization, and antibiotic use among children worldwide. Although respiratory viruses account for a substantial proportion of pediatric pneumonia, bacterial pathogens continue to cause severe disease and complications. Increasing antimicrobial resistance, particularly among Streptococcus pneumoniae and Mycoplasma pneumoniae, presents important challenges for effective treatment.

Objective: To evaluate the current patterns and mechanisms of antibiotic resistance in pediatric community-acquired pneumonia, identify major diagnostic difficulties, and examine evidence-based approaches to antimicrobial treatment and stewardship.

Materials and Methods: A narrative review of international scientific literature was conducted, focusing on publications and clinical recommendations available through November 2026. Relevant clinical studies, systematic reviews, randomized controlled trials, and international guidelines were analyzed. Particular attention was given to bacterial etiology, antimicrobial resistance, molecular diagnostic methods, and antibiotic treatment duration.

Results: Published evidence demonstrates considerable geographical variation in the etiology and antimicrobial resistance of pediatric CAP. A 2025 systematic review involving 20,141 children reported a pooled macrolide resistance proportion of 61% among investigated M. pneumoniae infections, with substantial regional differences. A 2026 South Korean multicenter surveillance study involving 740 hospitalized children identified atypical pathogens in 43.6% of cases and pyogenic bacterial pathogens in 2.7%. A separate 2025 study involving 772 children found that only 57.1% received guideline-concordant antibiotic management. Randomized trials have demonstrated that shorter antibiotic courses may achieve favorable outcomes in selected children with uncomplicated CAP while reducing unnecessary antimicrobial exposure.

Conclusion: Effective management of pediatric CAP requires appropriate differentiation of viral, typical bacterial, and atypical infections; consideration of local antimicrobial susceptibility patterns; and adherence to evidence-based treatment recommendations. Antimicrobial stewardship, improved diagnostic capacity, and continuous resistance surveillance are essential for optimizing clinical outcomes.

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Keywords: pediatric pneumonia, community-acquired pneumonia, antibiotic resistance, antimicrobial stewardship, Streptococcus pneumoniae, Mycoplasma pneumoniae, macrolide resistance, molecular diagnostics, antibiotic therapy.

INTRODUCTION

Community-acquired pneumonia is one of the most important infectious diseases affecting children worldwide. It represents a substantial cause of pediatric outpatient consultations, emergency department visits, hospital admissions, and mortality, particularly in low- and middle-income countries. Despite advances in vaccination, antimicrobial treatment, and supportive care, pneumonia continues to present significant diagnostic and therapeutic challenges.

The etiology of pediatric CAP is heterogeneous and varies according to patient age, vaccination status, geographical location, seasonal circulation of respiratory pathogens, and underlying medical conditions. Respiratory viruses, including respiratory syncytial virus, influenza virus, human metapneumovirus, and other respiratory pathogens, account for a considerable proportion of infections in preschool-aged children. However, bacterial pathogens remain important causes of severe pneumonia, pleural complications, and systemic infection.

Among the most clinically significant bacterial agents are Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus, and Mycoplasma pneumoniae. Their relative contributions vary across healthcare settings. Moreover, bacterial and viral pathogens may coexist, further complicating clinical diagnosis and antimicrobial decision-making.

The emergence and dissemination of antibiotic-resistant microorganisms have created additional difficulties in the management of childhood pneumonia. Antimicrobial resistance may develop through genetic mutations, horizontal gene transfer, modification of antimicrobial targets, production of antibiotic-inactivating enzymes, and altered membrane permeability.

In S. pneumoniae, resistance to beta-lactam antibiotics primarily involves alterations in penicillin-binding proteins. Macrolide resistance may be associated with target-site modification or active drug efflux. In M. pneumoniae, mutations affecting the 23S ribosomal RNA are important mechanisms of resistance to macrolide antibiotics.

The clinical consequences of antibiotic resistance may include prolonged fever, delayed recovery, increased hospitalization, and the need to modify antimicrobial treatment. However, these outcomes depend on the pathogen, infection severity, the antimicrobial agent used, and the availability of appropriate alternative therapy.

A further difficulty is the frequent inability to establish a definitive microbiological diagnosis at the initial clinical assessment. Symptoms such as fever, cough, tachypnea, and respiratory distress occur in both viral and bacterial infections. Radiological findings and inflammatory biomarkers may provide useful information, but they cannot reliably distinguish all etiological categories.

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Consequently, clinicians frequently initiate empirical antibiotic treatment before identifying the causative microorganism. Although timely antibiotic therapy is essential for children with suspected serious bacterial pneumonia, unnecessary or excessively broad-spectrum antibiotic use contributes to adverse effects and antimicrobial selection pressure.

Recent international recommendations have emphasized appropriate empirical antibiotic selection, shorter treatment courses in uncomplicated disease, and regular reassessment of antibiotic necessity.

The aim of this study is to examine contemporary antimicrobial resistance patterns in pediatric community-acquired pneumonia, analyze their diagnostic and therapeutic implications, and identify strategies for improving rational antibiotic use in children.

MATERIALS AND METHODS

This study was designed as a narrative review of international scientific publications addressing the etiology, diagnosis, antimicrobial resistance, and management of pediatric community-acquired pneumonia.

Relevant scientific literature published between 2015 and November 2026 was examined, with particular emphasis on research from 2024–2026. The review included multicenter observational studies, systematic reviews, meta-analyses, randomized clinical trials, and clinical guidelines from international healthcare organizations.

Scientific evidence was obtained from publications indexed in major medical databases, including PubMed, and from official sources such as the World Health Organization, the National Institute for Health and Care Excellence, and the Centers for Disease Control and Prevention.

The reviewed evidence was organized into four analytical domains: etiological characteristics of pediatric pneumonia, molecular mechanisms and epidemiology of antimicrobial resistance, diagnostic strategies, and evidence-based antibiotic management.

Descriptive and comparative methods were applied to summarize published findings. Antimicrobial resistance data were interpreted with consideration of geographical location, study population, microbiological methods, and pathogen distribution.

The study did not include original patient recruitment, clinical interventions, microbiological experiments, or independent statistical analysis. All numerical outcomes presented in the article were obtained from previously published investigations.

Because the review was narrative rather than systematic, pooled estimates were not independently calculated, and the findings should not be considered a comprehensive quantitative assessment of global resistance prevalence.

RESULTS

Etiological Characteristics of Pediatric Community-Acquired Pneumonia

The reviewed literature indicates that childhood pneumonia is caused by a wide range of respiratory pathogens, with substantial differences in distribution across age groups and geographical regions.

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Jain and colleagues conducted a large population-based investigation of community-acquired pneumonia requiring hospitalization among children in the United States. Among 2,222 children with radiographically confirmed pneumonia and adequate specimens for microbiological testing, at least one respiratory virus was detected in 66%, bacteria alone in approximately 8%, and both bacterial and viral pathogens in approximately 7% [1].

These findings demonstrate the considerable contribution of respiratory viruses to pediatric CAP and highlight the importance of avoiding unnecessary antibiotic treatment when bacterial infection is unlikely.

However, the relative contribution of bacterial and atypical pathogens may increase during particular epidemic periods.

Kim and colleagues published a multicenter prospective surveillance investigation in 2026 involving 740 children hospitalized with CAP across 17 hospitals in South Korea. A potential etiological pathogen was identified in 541 patients (73.1%). Atypical pathogens accounted for 43.6% of the cases, viral pathogens for 26.8%, and pyogenic bacterial pathogens for 2.7% [2].

During the peak M. pneumoniae epidemic period, atypical CAP accounted for 57.0% of cases. These findings demonstrate the importance of considering contemporary pathogen circulation when making diagnostic and empirical treatment decisions.

Nevertheless, the low proportion of identified pyogenic bacterial infections should not be interpreted as evidence that conventional bacterial pneumonia is clinically insignificant. Severe pyogenic infections may be associated with respiratory support requirements, prolonged hospitalization, and intensive care admission.

Mechanisms of Antibiotic Resistance in Major Respiratory Pathogens

Antimicrobial resistance in pediatric CAP involves several distinct molecular and microbiological mechanisms.

In Streptococcus pneumoniae, reduced susceptibility to penicillin and related beta-lactam antibiotics is primarily caused by structural alterations in penicillin-binding proteins. These changes reduce antibiotic affinity for bacterial targets and may affect therapeutic activity.

Pneumococcal resistance to macrolides is frequently associated with methylation of the ribosomal target mediated by erm(B) or antibiotic efflux associated with mef genes.

In Haemophilus influenzae, beta-lactam resistance may involve beta-lactamase production or alterations in penicillin-binding proteins. Consequently, susceptibility to individual antimicrobial agents cannot be assumed without considering the local microbiological context.

Staphylococcus aureus may exhibit methicillin resistance through the acquisition of altered penicillin-binding proteins encoded by resistance-associated genetic elements. Methicillin-resistant S. aureus is particularly important in selected cases of severe, necrotizing, or complicated pneumonia.

Mycoplasma pneumoniae presents a different microbiological challenge because it lacks a bacterial cell wall. Consequently, beta-lactam antibiotics are intrinsically ineffective against this organism.

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Macrolides inhibit bacterial protein synthesis through interaction with the 50S ribosomal subunit. However, mutations in the 23S ribosomal RNA gene, particularly substitutions corresponding to positions A2063 and A2064, may reduce macrolide binding and produce antimicrobial resistance.
These differences demonstrate that antibiotic resistance is not a uniform biological phenomenon and requires pathogen-specific diagnostic and therapeutic interpretation.

Table 1. Major Pathogens and Antimicrobial Resistance Mechanisms in Pediatric CAP

Pathogen	Principal resistance mechanism	Diagnostic and therapeutic significance
Streptococcus pneumoniae	Altered penicillin-binding proteins; macrolide target modification or efflux	Susceptibility depends on antibiotic, breakpoint, and infection site
Mycoplasma pneumoniae	23S rRNA mutations	Reduced effectiveness of macrolide treatment
Haemophilus influenzae	Beta-lactamase production; altered penicillin-binding proteins	May reduce susceptibility to selected beta-lactams
Staphylococcus aureus	Methicillin resistance mediated by altered penicillin-binding proteins	Important in selected severe and complicated infections

Source: Narrative synthesis of established microbiological mechanisms and contemporary clinical guidance.

Macrolide Resistance in Mycoplasma pneumoniae

Macrolide-resistant M. pneumoniae has emerged as an important international concern, particularly in East Asian countries.
Yang and colleagues published a systematic review and meta-analysis in 2025 involving 65 studies and 20,141 pediatric patients. The overall pooled proportion of macrolide-resistant M. pneumoniae was 61% (95% confidence interval [CI], 54–68%) [3].
Substantial geographical differences were identified. The reported pooled resistance proportions were approximately 68% in China, 61% in Japan, and 63% in South Korea.
These estimates reflect the studies included in the meta-analysis and should not be interpreted as the prevalence of resistance among all children worldwide.

The same analysis demonstrated that macrolide-resistant infection was associated with longer fever duration and hospitalization. Compared with macrolide-susceptible infections, resistant infections were associated with approximately 1.97 additional days of fever and 1.96 additional days of hospitalization. The odds of severe pneumonia were also increased in the resistant group (odds ratio [OR], 2.45; 95% CI, 1.75–3.44).
These findings suggest that macrolide resistance may contribute to delayed clinical improvement and increased treatment complexity.

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However, resistance patterns differ considerably between countries. According to CDC guidance updated in May 2026, macrolide resistance among M. pneumoniae infections has remained below 10% in the United States [4].

The considerable geographical variation reinforces the importance of local antimicrobial surveillance and cautions against applying resistance estimates from one region to another.

Diagnostic Challenges in Pediatric Pneumonia

Early and accurate diagnosis of bacterial pneumonia remains challenging because clinical manifestations overlap substantially with those of viral respiratory infections.

Common symptoms include fever, cough, tachypnea, chest discomfort, reduced feeding, and respiratory distress. These manifestations provide important clinical information but are not specific to a single microbial etiology.

Chest radiography may identify pulmonary consolidation, interstitial abnormalities, and pleural complications. However, radiological patterns alone cannot reliably distinguish bacterial from viral pneumonia or determine antibiotic susceptibility.

Inflammatory biomarkers such as C-reactive protein and procalcitonin are frequently used to support clinical assessment. Elevated levels may increase suspicion of bacterial infection, but neither biomarker can independently establish the causative microorganism or confirm antimicrobial resistance.

Microbiological investigations are therefore important when results are likely to influence treatment.

Blood cultures may help identify invasive bacterial pathogens, particularly in children with severe infection or suspected sepsis. Nevertheless, their diagnostic yield is often limited.

Molecular tests, including polymerase chain reaction assays, offer opportunities for rapid identification of selected respiratory pathogens. However, detection of microbial nucleic acid in upper respiratory specimens does not always establish that the detected organism caused lower respiratory tract disease.

For example, pneumococcal carriage in the nasopharynx may occur without invasive bacterial infection. Similarly, detection of a respiratory virus does not completely exclude bacterial coinfection.

Modern diagnostic strategies should therefore combine clinical findings, disease severity assessment, microbiological investigations when indicated, and knowledge of local pathogen circulation.

The NICE pneumonia guideline, updated in 2025, recommends against routine microbiological testing in children with non-severe CAP. For severe cases, blood cultures and age-appropriate respiratory sampling may be considered according to clinical indications [5].

Guideline-Concordant Antibiotic Treatment

Appropriate antibiotic selection is essential for balancing effective treatment with antimicrobial stewardship.

Antoon and colleagues conducted a multicenter study published in 2025 involving 772 children presenting with CAP to two pediatric emergency departments in the United States.

Among these patients, 573 children (74.2%) received antibiotics, while 441 (57.1%) received management consistent with the treatment guidelines used in the study [6].

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Therefore, more than 40% of the study population received treatment that did not fully correspond to guideline recommendations.

These observations indicate that inappropriate antibiotic use remains an important challenge even in specialized pediatric healthcare institutions.

The World Health Organization recommends oral amoxicillin for selected children aged 2–59 months with uncomplicated pneumonia, including cases characterized by fast breathing without general danger signs [7].

Similarly, the 2025 NICE guideline identifies amoxicillin as a first-line oral antibiotic for many children with non-severe CAP. However, treatment selection must be adapted to patient age, clinical severity, allergy status, complications, and local microbiological information.

For suspected atypical pneumonia caused by M. pneumoniae, macrolides may be considered when clinically indicated. Nevertheless, their use should take into account regional resistance patterns and the possibility of treatment failure.

Alternative antimicrobial agents for confirmed or strongly suspected macrolide-resistant infection require specialist consideration, particularly in younger children, because age-related safety issues and other clinical factors influence drug selection.

Shorter Antibiotic Courses and Antimicrobial Stewardship

Recent clinical trials have examined whether shorter antibiotic treatment courses can provide outcomes comparable to longer treatment regimens in selected children with uncomplicated pneumonia. The SCOUT-CAP randomized clinical trial, published by Williams and colleagues in 2022, included 380 children with uncomplicated outpatient CAP who demonstrated early clinical improvement [8].

A five-day antibiotic strategy produced clinical outcomes comparable to a ten-day strategy while reducing antibiotic exposure. The shorter treatment approach was also associated with a lower abundance of antimicrobial resistance genes in respiratory microbiota.

The CAP-IT randomized clinical trial, involving 824 randomized children, investigated different doses and durations of oral amoxicillin therapy. Three-day treatment was noninferior to seven-day treatment for the primary outcome of antibiotic re-treatment in the studied population [9].

These findings contributed to growing support for shorter treatment durations in carefully selected patients.

The NICE guideline updated in 2025 recommends a three-day antibiotic course for children aged three months to eleven years with non-severe, uncomplicated CAP and without underlying disease. Treatment should be reassessed and extended when clinically necessary [5].

However, these recommendations should not be generalized to severe, complicated, or unstable patients.

Table 2. Selected Clinical Studies Relevant to Pediatric CAP and Antibiotic Stewardship

Study	Population	Principal finding

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Jain et al., 2015	2,222 hospitalized children	Respiratory viruses detected in 66%
Yang et al., 2025	20,141 children across 65 studies	Pooled macrolide resistance proportion of 61%
Antoon et al., 2025	772 children	57.1% received guideline-concordant management
Kim et al., 2026	740 hospitalized children	Atypical pathogens identified in 43.6%
Williams et al., 2022	380 children	Five-day therapy had comparable clinical response to ten-day therapy
Bielicki et al., 2021	824 randomized children	Three-day amoxicillin treatment was noninferior to seven days for the primary outcome

Note: The studies differ in populations, methods, and outcomes. The findings are presented for descriptive comparison rather than pooled statistical analysis.

DISCUSSION

The present review demonstrates that antimicrobial resistance in pediatric community-acquired pneumonia is a complex clinical problem involving microbial evolution, diagnostic uncertainty, regional epidemiology, and prescribing practices.

One of the central challenges is the difficulty of distinguishing bacterial pneumonia from respiratory viral infections at the initial clinical assessment. Because many children with pneumonia present with similar respiratory symptoms regardless of etiology, antibiotic treatment is frequently initiated empirically.

Although this approach is appropriate in selected patients with suspected serious bacterial infection, unnecessary antibiotics may increase adverse drug reactions, affect the respiratory and intestinal microbiota, and contribute to antimicrobial selection pressure.

The findings of the EPIC investigation and the more recent South Korean surveillance study highlight the importance of considering non-pyogenic causes of pediatric CAP. Respiratory viruses and atypical pathogens may account for substantial proportions of cases, particularly during specific epidemic periods.

Nevertheless, the relative frequency of pathogens depends on age, geographical region, vaccination history, microbiological testing methods, and study definitions. Consequently, data from one healthcare system cannot be assumed to represent the epidemiology of another country.

The increasing recognition of macrolide-resistant M. pneumoniae is particularly important. The 2025 meta-analysis demonstrated considerable regional variation in macrolide resistance, with high pooled proportions in East Asian populations.

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The clinical significance of these findings is supported by associations between resistance and prolonged fever, extended hospitalization, and increased odds of severe disease. However, these associations do not establish that resistance alone causes all observed complications. Host immune responses, disease severity, delayed diagnosis, and variations in treatment may also contribute.

A major implication is that empirical treatment strategies should consider the prevalence of specific pathogens and local susceptibility patterns rather than relying exclusively on international averages.

For typical bacterial pneumonia, narrow-spectrum antibiotics remain appropriate in many uncomplicated cases. The presence of antimicrobial resistance at a population level does not automatically justify routine broad-spectrum treatment for every child.Antibiotic escalation should be guided by clinical deterioration, microbiological findings, suspected complications, or other well-defined indications.Another important finding concerns the role of diagnostic stewardship. Increased availability of molecular testing may facilitate faster pathogen identification, but indiscriminate testing can create interpretation problems.Positive molecular results must be evaluated within the clinical context, particularly when an organism may colonize the upper respiratory tract without causing pneumonia.Appropriate use of laboratory investigations may therefore be more valuable than simply increasing the number of tests performed.Antimicrobial stewardship is also essential for reducing unnecessary treatment duration. The SCOUT-CAP and CAP-IT trials provide evidence that shorter antibiotic courses can be effective in selected children with uncomplicated pneumonia.Such strategies may reduce cumulative antibiotic exposure without compromising treatment outcomes when used in appropriate populations.

However, duration recommendations must be individualized. Children with pleural effusion, empyema, necrotizing pneumonia, persistent clinical instability, or other serious complications may require different treatment approaches.The implementation of stewardship programs should involve pediatricians, clinical microbiologists, pharmacists, infectious disease specialists, and infection prevention professionals.Particular attention should be given to locally developed antimicrobial susceptibility reports, regular evaluation of prescribing patterns, and timely reassessment of empirical treatment.

For healthcare institutions in Uzbekistan, future research should focus on the distribution of bacterial pathogens responsible for childhood CAP, antibiotic susceptibility profiles, and the prevalence of macrolide-resistant M. pneumoniae.

Prospective microbiological investigations and multicenter surveillance would provide important information for adapting international treatment recommendations to local clinical conditions.

The major limitation of the present article is its narrative review design. The included studies represent different geographical populations, diagnostic approaches, and clinical settings. No independent pooled analysis was performed, and the findings cannot establish precise resistance prevalence in Uzbekistan.

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Further research is necessary to identify effective diagnostic algorithms, establish reliable local antimicrobial resistance databases, and determine the impact of stewardship interventions on pediatric treatment outcomes.

CONCLUSION

Antibiotic resistance represents an important and evolving challenge in pediatric community-acquired pneumonia.

The reviewed scientific literature demonstrates that childhood pneumonia has a heterogeneous etiology, with respiratory viruses, typical bacterial pathogens, and atypical microorganisms contributing differently across populations and epidemic periods.

Macrolide-resistant Mycoplasma pneumoniae is of particular concern in several regions and may be associated with prolonged fever, increased hospitalization, and greater treatment complexity.

Accurate diagnosis requires the integration of clinical assessment, appropriate laboratory investigations, and current epidemiological information. Neither imaging nor inflammatory biomarkers alone can reliably establish the causative pathogen or determine antimicrobial susceptibility.

Evidence-based antibiotic management should prioritize appropriate empirical therapy, consideration of local resistance patterns, timely reassessment, and avoidance of unnecessary broad-spectrum treatment.

Shorter antibiotic courses are supported by clinical trial evidence for selected children with uncomplicated pneumonia, although treatment duration must be individualized according to disease severity and clinical response.

Strengthening microbiological surveillance, antimicrobial stewardship, vaccination programs, and clinical guideline adherence may contribute to improved pediatric outcomes and more responsible antibiotic use.

Future studies should prioritize local resistance monitoring and the development of standardized diagnostic and therapeutic strategies appropriate for different healthcare environments.

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