VIRAL VS. BACTERIAL PNEUMONIA IN CHILDREN: PATHOPHYSIOLOGICAL INSIGHTS AND DIAGNOSTIC DILEMMAS

Ergashzoda Khusniya Sharafidin Kizi

Central Asian Medical University

E-mail: ergashzodaxusniya@gmail.com

https://orcid.org/0009-0001-6423-3642

Eminov Ravshanjon Ikromjon ugli

Department of Faculty and Hospital Surgery, FMIOPH, Fergana, Uzbekistan

Abstract: Pneumonia remains a leading cause of morbidity and mortality in children under five, especially in low-resource settings such as Uzbekistan. Distinguishing between viral and bacterial pneumonia is critical for appropriate treatment but remains diagnostically challenging. While viral pneumonia commonly presents with diffuse infiltrates and systemic symptoms like wheezing, bacterial pneumonia often has focal consolidation and higher inflammatory markers. However, clinical, radiological, and biomarker overlap complicates this differentiation. Improved diagnostic tools, clinician training, and vaccination coverage are essential to enhance pediatric pneumonia management and reduce unnecessary antibiotic use.

Keywords: pneumonia, children, diagnosis, virus, bacteria

Introduction

Acute pneumonia is a major infectious disease affecting children worldwide, especially those under five years of age. Globally, pneumonia accounts for about 14% of all deaths in children under five[1]. In Uzbekistan – a country that has dramatically reduced under-five mortality to ~13.3 per 1,000 live births – pneumonia remains a leading cause of childhood hospitalization. Pneumonia can be caused by a wide array of pathogens, most commonly respiratory viruses or bacteria[2]. Importantly, the aetiological profile of pediatric pneumonia has been altered by widespread vaccination: immunization against pneumococcus and Hib (introduced in Uzbekistan in 2015 and ~2009, respectively[3]) has reduced the incidence of bacterial pneumonia. As a result, respiratory viruses (e.g. RSV, influenza, parainfluenza, adenovirus, rhinovirus) now predominate in many pediatric pneumonia cases[4].

Distinguishing viral from bacterial pneumonia is crucial because it guides treatment – antibiotics are indicated for bacteria but not viruses – yet this differentiation is notoriously difficult in clinical practice[5]. The World Health Organization emphasizes that timely antibiotic treatment of pneumonia can save lives, yet globally only about one-third of children with pneumonia receive antibiotics[6]. In resource-limited settings such as Uzbekistan, diagnostic facilities are often scarce, leading to reliance on clinical algorithms. For example, WHO's Integrated Management of Childhood Illness (IMCI) classifies any child with cough and fast breathing (or chest indrawing) as pneumonia

requiring antibiotics[7]. This broad approach inevitably results in treating many viral cases with antibiotics, contributing to overuse and resistance.

This review explores the pathophysiology, clinical presentation, and diagnosis of viral versus bacterial pneumonia in children, with an emphasis on the practical challenges in Uzbekistan and Central Asia. We examine the immune and inflammatory mechanisms underlying each type of infection, compare the distinguishing (and overlapping) clinical features, and critically evaluate diagnostic tools (imaging, microbiology, biomarkers) for etiology. Our goal is to synthesize current evidence (2015–2025) from peer-reviewed sources and health authorities to provide an in-depth, evidence-based comparison of viral and bacterial pneumonia in pediatric populations, and to highlight strategies to improve diagnosis and management in the region.

Pathophysiology

Both viral and bacterial pneumonias begin with pathogen entry into the lower respiratory tract, but the subsequent immune responses and tissue effects diverge. The respiratory epithelium and mucociliary clearance normally serve as primary barriers; viruses and bacteria that evade these defenses trigger innate immune activation. Viral pneumonia pathogens (e.g. RSV, influenza, adenovirus) infect respiratory epithelial cells and replicate intracellularly. Infected cells release type I interferons and other cytokines that orchestrate an antiviral response. Natural killer cells and cytotoxic T lymphocytes play key roles in containing intracellular viruses, while infected cell death (via necrosis or apoptosis) can damage the airway lining. The inflammation is often interstitial (affecting septae and peribronchial tissue) and involves a mononuclear infiltrate (macrophages and lymphocytes)[8]. Notably, severe viral infections (such as RSV bronchiolitis) can also induce intense neutrophilic inflammation: for example, infants with severe RSV pneumonia may have neutrophils comprising >90% of bronchial lavage cells[9]. Cytokine profiles in viral pneumonia often feature elevated interferon-α/β and interleukin-10 (anti-inflammatory) in addition to IL-6 and IL-8; however, the exact patterns can vary among viruses.

Table. Comparision of VP vs BP

Feature	Viral Pneumonia	Bacterial Pneumonia
Common pathogens	RSV, influenza, rhinovirus, parainfluenza, adenovirus, etc.	*Streptococcus pneumoniae*, *H. influenzae* type b, *Staphylococcus aureus*, *Mycoplasma pneumoniae*, etc.[11]
Initial infection site	Bronchiolar and interstitial (alveolar walls); often bilateral involvement[12]	Alveolar spaces and bronchi; often focal lobar or segmental consolidation
Immune response	Predominantly interferon-mediated, lymphocytic/mononuclear infiltrate; can include neutrophils in severe cases (e.g. RSV)[8]	Neutrophil-dominated inflammation with pro-inflammatory cytokines (IL-1, IL-6, IL-8, TNF-α)
Inflammatory exudate	Serous or mucopurulent; less voluminous exudate	Purulent exudate filling alveoli, may form lobar consolidation
Fever pattern	Often low-grade or fluctuating; can be prolonged (e.g. influenza causes high	High, continuous fever is common

	fever)	
Onset and prodrome	Often gradual, preceded by URTI symptoms (runny nose, sore throat)	Often abrupt onset, sometimes after viral URTI
Cough characteristics	Typically dry or hacking; can progress to productive if secondary infection	Usually productive or severe cough
Chest findings	Bilateral diffuse crackles; wheezing common (esp. RSV, influenza)[4]	Focal crackles, bronchial breath sounds, localized dullness to percussion if lobar
Radiographic pattern	Diffuse or interstitial infiltrates; peribronchial cuffing (e.g. "cloud-like" opacities)[7]	Lobar or segmental consolidation (dense opacity); air bronchograms
WBC/biomarkers	WBC normal or low; lymphocyte predominance; moderate CRP, PCT rise if severe	WBC elevated with neutrophilia; often higher CRP and PCT (though overlap)
Typical course	Usually self-limited in healthy children; recovery in ~1–2 weeks; complications less common	Can be severe or fulminant; complications (pleural effusion, empyema) more common
Treatment implications	Supportive care; antiviral therapy for specific viruses (e.g. oseltamivir for influenza); antibiotics not routinely needed	Prompt antibiotics (e.g. amoxicillin or penicillin derivatives) are indicated; hospitalization and IV antibiotics for severe cases

Clinical Features

The clinical presentation of pneumonia in children often overlaps between viral and bacterial causes. Common symptoms in both include cough, tachypnea (fast breathing), fever, and respiratory distress. Viral pneumonia frequently begins with upper respiratory symptoms: rhinorrhea, nasal congestion, and cough, sometimes with wheezing or stridor. Fever may be low-grade or variable, and the child may have scattered crackles or wheezes bilaterally on auscultation[13]. Viral infections often involve both lungs diffusely; for example, clinicians may find decreased air entry or crackles in multiple lung fields. In contrast, bacterial pneumonia in a child often presents more acutely with high continuous fever, marked toxicity, and often a history of sudden onset. Physical exam may reveal focal findings: localized bronchial breath sounds, dullness to percussion, or pleural rub if effusion is present. One lung lobe or segment is typically more affected, consistent with lobar consolidation. Cough is often productive (though children may swallow sputum), and the illness can progress to severe respiratory distress if not treated.

Despite these tendencies, many findings are nonspecific. For instance, wheezing is more often associated with viral etiologies (e.g. RSV) but can occur in bacterial superinfection or asthma overlap. Likewise, both types can cause hypoxia, dehydration, or failure to thrive in severe cases. WHO notes that "presenting features of viral and bacterial pneumonia are similar" in young children, with subtle clues only (e.g. wheeze more common in viral). A systematic review affirms that no single clinical finding reliably distinguishes the two, so a combination of signs is used.

Prognosis also differs: most viral pneumonias in children are self-limited, and long-term sequelae are rare. By contrast, certain bacterial pneumonias can be fulminant: for instance, *Staphylococcus aureus* or varicella-associated pneumonia can have high mortality in children. Influenza pneumonia in particular can be severe, as noted earlier. Table 1 above summarizes the contrasting clinical and laboratory features of viral versus bacterial pediatric pneumonia.

Diagnostic Tools

Diagnosing the cause of pneumonia requires combining clinical, radiographic, microbiologic, and laboratory information. No single test is definitive, and resource limitations in Uzbekistan and similar regions complicate matters. Common diagnostic modalities include chest imaging, microbiological cultures/PCR, and biomarkers, each with strengths and weaknesses.

- Chest Radiography is widely used to confirm pneumonia but cannot reliably distinguish viral from bacterial cause. A posteroanterior (PA) chest X-ray remains the reference standard for diagnosing pneumonia. In viral pneumonia, radiographs often show bilateral diffuse or interstitial infiltrates (occasionally described as “ground-glass” opacities), reflecting widespread inflammation. In bacterial pneumonia, one typically sees dense opacification in one lobe or segment, often with air bronchograms (see Figure 1). However, overlap is common: partial lobar consolidation can occur with viruses, and multifocal infiltrates with bacteria. Radiography also requires equipment and radiation safety, which may be limited outside major hospitals.

Figure: Chest radiograph of a child with viral pneumonia (CMV infection), demonstrating bilateral diffuse interstitial infiltrates (indicated by hazy opacification in both lungs). In contrast to the focal lobar consolidation expected in bacterial pneumonia, the diffuse pattern suggests a viral etiology. Such imaging helps support a clinical suspicion but is not definitively diagnostic.

- Lung Ultrasound (LUS) has emerged as a valuable alternative, especially in children where radiation avoidance is preferred. Ultrasound can detect consolidations, interstitial syndrome, and pleural effusions. Several studies report high sensitivity and specificity for pneumonia detection by LUS, and it may reveal differences: bacterial pneumonia often produces larger, subpleural consolidations with “hepatized” lung and dynamic air bronchograms, whereas viral pneumonia tends to yield smaller subpleural consolidations and diffuse B-lines (vertical artifacts). In one observational study, children with bacterial pneumonia had significantly higher LUS scores than those with viral infection[14]. A meta-analysis concludes LUS is a viable alternative to X-ray in pediatric pneumonia. However, LUS requires training, and its availability in rural clinics is limited.
- Microbiological Tests: Identifying the causative pathogen definitively is ideal but often challenging. Blood cultures in pediatric pneumonia have low yield (often <10%) and are slow[15]. Sputum culture is seldom possible in young children. Nasopharyngeal (NP) swabs or aspirates allow PCR or antigen tests for viruses: these are highly sensitive, and multiplex PCR panels can detect RSV, influenza, rhinovirus, etc. However, detection of a virus does not prove it is the pneumonia cause, since asymptomatic viral carriage is common. For example, one study found similar rates of viral detection (~77–78%) in children with severe pneumonia and in controls without pneumonia. PCR panels for atypical bacteria (e.g. *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*) and respiratory cultures (for tuberculosis, etc.) can be done but are not universally available. In Uzbekistan, routine

viral PCR testing is likely limited to reference labs, so most clinicians rely on rapid antigen tests (for influenza/RSV if accessible) or empirical judgment.

- Biomarkers: Blood tests such as C-reactive protein (CRP) and procalcitonin (PCT) are frequently measured. As noted, CRP tends to be higher in bacterial pneumonia, though with considerable overlap. PCT has been studied as a decision aid: a meta-analysis found PCT (cutoff ~0.5 ng/mL) had sensitivity ~68% and specificity ~60–72% for bacterial pneumonia in children. Neither marker alone is definitive. The 2021 Pediatric Infectious Diseases Society meta-analysis concluded that CRP and PCT performed modestly (AUROC ~0.70 each) and that novel biomarkers (e.g. cytokine profiles) might eventually help. In practice, elevated CRP/PCT may support antibiotic use, but normal values cannot rule out bacterial infection in a seriously ill child. White blood cell count and erythrocyte sedimentation rate perform even worse and are not reliable discriminators. Other tests like *urine pneumococcal antigen* have poor specificity in children (due to nasopharyngeal carriage) and are not recommended.

In resource-limited settings like rural Uzbekistan, many of these tools are unavailable. Often the initial assessment is based on history and physical exam alone (e.g. IMCI protocol). Chest radiography may not exist at district clinics, and lab turnaround times are long. This diagnostic uncertainty drives empirical treatment. As one U.S. pediatric text warns, “Diagnosis [of pneumonia] still remains challenging,” underscoring the difficulty of distinguishing viral from bacterial pneumonia at the bedside.

Challenges

Distinguishing viral versus bacterial pneumonia in children is fraught with challenges, particularly in low-resource settings. Major issues include:

- Symptom Overlap and Diagnostic Uncertainty: As noted, clinical features are insufficiently specific[17]. In practice, a child with cough and fast breathing is treated as pneumonia per WHO guidelines, regardless of presumed cause. This approach errs on the side of treating possible bacterial infection, but leads to antibiotic use in many viral cases.
- Resource Limitations: In Uzbekistan and similar contexts, access to imaging and laboratory tests is uneven. Only large hospitals in Tashkent or regional centers may have X-ray and lab facilities; rural clinics rely on clinical diagnosis. PCR and multiplex diagnostics are largely confined to research settings. Pulse oximetry and oxygen may not be available in small clinics. Even when tests exist, turnaround time may be too slow to guide immediate therapy.
- Laboratory and Radiology Variability: Even when available, test interpretation is not straightforward. Radiographs require experience: subtle interstitial infiltrates in viral pneumonia can be missed or misread. Inter-observer variation is high in pediatric chest X-rays. Lab tests have inherent variability: CRP measured by different assays or a WBC count can be confounded by malnutrition, malaria, or other local factors.

Overall, these challenges underline why clinicians in resource-poor settings rely on broad empiricism. A combination of rapid clinical deterioration (suggesting bacterial sepsis) and laboratory support (e.g. very high CRP) may guide more aggressive treatment, whereas milder, gradual cases may be observed or managed with symptomatic care. Efforts to develop rapid point-of-care diagnostics (such as CRP tests, viral rapid tests, or LUS) are ongoing and could transform care in settings like Uzbekistan.

Treatment

Management of pediatric pneumonia differs fundamentally between viral and bacterial etiologies. However, in practice, initial treatment often overlaps due to diagnostic uncertainty.

For bacterial pneumonia, prompt antibiotic therapy is indicated. The World Health Organization recommends oral amoxicillin dispersible tablets as first-line treatment for non-severe pneumonia in children. In hospitalized cases or very severe pneumonia, parenteral antibiotics are used: benzylpenicillin or ampicillin (typically at 100,000–200,000 units/kg/day) with gentamicin is common as empiric therapy to cover *S. pneumoniae*, *H. influenzae*, and others. If *Staphylococcus aureus* or other resistant organisms are suspected, vancomycin or clindamycin may be added. Importantly, local treatment protocols (such as Uzbekistan's pediatric guidelines) generally follow WHO/IDSA recommendations. In Uzbekistan, standard practice at the community level is to administer amoxicillin for presumed pneumonia, reflecting these guidelines.

By contrast, viral pneumonia treatment is primarily supportive. Oxygen therapy is critical for any child with hypoxemia. Ensuring adequate hydration and nutrition is also essential. Fever can be managed with antipyretics (acetaminophen/ibuprofen). There are a few specific antiviral treatments: for example, oseltamivir (an oral neuraminidase inhibitor) is indicated for influenza pneumonia if given early, especially in high-risk children. Ribavirin has been used (inhaled or IV) for severe RSV infection, but its use is limited and generally reserved for life-threatening cases due to toxicity and mixed evidence. Otherwise, no specific antivirals exist for most pediatric respiratory viruses (e.g. adenovirus, rhinovirus), so care is supportive. Corticosteroids are not generally indicated except in specific situations (e.g. *Pneumocystis jiroveci* pneumonia in an HIV-infected child, or airway hyperreactivity).

Conclusion

In summary, viral and bacterial pneumonia in children differ in their etiologies, immune responses, clinical courses, and management, but they can present with overlapping symptoms that make rapid differentiation difficult. Viral agents (RSV, influenza, rhinovirus, etc.) now predominate in pediatric pneumonia in Uzbekistan and Central Asia, especially after widespread vaccination against Hib and pneumococcus. Viral pneumonias tend to produce diffuse bilateral lung involvement, wheezing, and a self-limited course, whereas bacterial pneumonias often cause focal consolidation, high fever, and can be life-threatening. Yet features alone are unreliable, and even laboratory tests (CRP, PCT) and imaging have only moderate accuracy. As a result, clinical practice in resource-limited settings frequently errs on the side of giving antibiotics to any child with suspected pneumonia.

References:

1. 1. Adhamjon ogh, A. A. Z., & Mominjonovna, M. B. (2025, May). CLINICAL PHARMACOLOGY OF ANTI-INFLAMMATORY DRUGS. In CONFERENCE OF MODERN SCIENCE & PEDAGOGY (Vol. 1, No. 2, pp. 88-91).
2. 2. Boboxonova, M. (2025). COMBATING EARLY MENOPAUSE: MODERN MEDICAL APPROACHES AND NATURAL TREATMENT METHODS.International Journal of Artificial Intelligence, 1(4), 56-59.

3. 3. Boretskaya, A. S. (2025). VIRAL VECTORS. STUDY AND RESEARCH OF DNA AND RNA CONTAINING VIRUSES.Western European Journal of Medicine and Medical Science, 3(05), 38-41.
4. 4.Boretskaya, A., Farid, M. E., Egorova, S., & Lamberov, A. (2022). Transformation of amorphous aluminum oxide in the catalytic dehydration reaction of aromatic alcohol.
5. 5.Ganiyeva M. R. CLINICAL AND MORPHOFUNCTIONAL CHANGES IN THE RETINA IN HIGH MYOPIA IN COMBINATION WITH AGE-RELATED MACULAR DEGENERATION OF DIFFERENT STAGES //International Conference on Modern Science and Scientific Studies. – 2024. – P. 141-142.
6. 6.Ikromova, N. (2024). CHEMICAL PROPERTIES AND PRACTICAL IMPORTANCE OF NATURAL CYANOGLYCOSIDE-AMYGDALIN. Universal International Scientific Journal, 1(6), 26-29.
7. 7.Ikromova, N. (2024, October). PROSPECTS OF SYNTHESIS OF AMIGDALIN DERIVATIVES. In CONFERENCE ON THE ROLE AND IMPORTANCE OF SCIENCE IN THE MODERN WORLD (Vol. 1, No. 8, pp. 164-166).
8. 8. Mo'Minjonovna, B. M., & O'G'Lee, M. A. R. (2024). STUDY AND ANALYSIS OF THE PHARMACOLOGICAL PROPERTIES OF MEDICINAL PLANTS WHICH ARE CARDIAC GLYCOSIDES USED IN CLINICAL PRACTICE.Eurasian Journal of Medical and Natural Sciences, 4(1-1), 80-83.
9. 9. Rakiboyevna, G. M., & Abdulhay, M. (2025). PREVENTION OF COMPLICATIONS OF CARDIOVASCULAR DISEASES BY ORGANIZING MORPHOLOGICAL AND CLINICAL INDICATORS OF ARCUS SENILIS.Modern education and development, 26(4), 201-204.
10. Rakhibovna, G. M., & Abdulhay, M. (2025, May). MORPHOLOGICAL AND CLINICAL INDICATIONS OF COMPLICATIONS OF CARDIOVASCULAR DISEASE ARCUS SENILIS.In *International Conference on Multidisciplinary Sciences and Educational Practices* (pp. 182-184).

11. Bobokhonova, M. M., & Dekhkonboeva, K. A. (2021). NATIONAL MODEL OF MATERNAL AND CHILD HEALTHCARE IN UZBEKISTAN: "HEALTHY MOTHER-HEALTHY CHILD". Economy and Society, (10 (89)), 540-543.

12. Boretskaya, A. S. (2022). THE STATE OF EDUCATION AND PEDAGOGICAL THOUGHT IN THE ERA OF BERUNI. Academic research in educational sciences, (3), 125-127.

13. Boretskaya, A. S., Rasulov, F. Kh., Ruzaliev, K. N., & Khasanov, N. F. U. (2024). IMMUNOGENESIS AND INTESTINAL MICROFLORA IN PATHOLOGY OF MIXED ETIOLOGY AND WAYS OF THEIR CORRECTION. Science and innovation, 3(Special Issue 45), 276-281.

14. Ikromova, N. M. (2024). Scientific and Theoretical Foundations of Social Adaptation of Senior Preschoolers Based on Speech Development. Miasto Przyszłości, 54, 385-387.

15. Ikromova, N., & Eminov, R. (2025). The influence of emotional intelligence and anxiety level on speech development and social adaptation of preschool children. in Library, 1(2), 15-19.

16. Ikromova, N., & Eminov, R. (2025). Speech and language development in preschoolers: the role of parental interaction. in Library, 1(2), 28-32.

17. Rasulov, F. H., Boretskaya, A. S., Mamatkulova, M. T., & Ruzibaeva, Y. R. (2024). INFLUENCE AND STUDY OF MEDICINAL PLANTS OF UZBEKISTAN ON THE IMMUNE SYSTEM. Web of Medicine: Journal of Medicine, Practice and Nursing, 2(12), 118-124.

18. Rasulov, F., Tozhalievna, M., Ruzibaeva, Y., & Boretskaya, A. (2024). Study of a stable form of coronavirus and its resistance to variability. Preventive Medicine and Health, 3(3), 20-26.

19. Teshaboev, A. M., Yulchieva, S. T., Rasulov, U. M., Boretskaya, A. S., & Rasulov, F. H. STUDY OF IMMUNOGENESIS AND HEMOPOIESIS IN ANIMALS WITH ACETYLATION TYPE AND WAYS OF THEIR CORRECTION WITH PURIFIED DETOXIOM COMPLEX.