Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 2, Issue 6, June 2026
Website: usajournals.org

PREVENTION OF POST-VIRAL PULMONARY FIBROSIS IN CHILDREN FOLLOWING VIRAL PNEUMONIA: PATHOGENETIC BASIS, RISK FACTORS, AND CURRENT STRATEGIES — A LITERATURE REVIEW

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Abstract

This article presents a systematic literature review examining the prevention of post-viral pulmonary fibrosis in children following viral pneumonia. Publications from 2010 to 2024 indexed in PubMed, Scopus, Web of Science, and Cochrane Library were analysed. The review addresses the pathogenetic mechanisms of post-infectious pulmonary fibrosis in the paediatric population, age-specific risk factors, approaches to early detection of fibrotic changes, and the evidence base for pharmacological and non-pharmacological preventive strategies. Particular attention is given to fibrotic outcomes following SARS-CoV-2, influenza, adenovirus, and RSV pneumonias in children. The analysis demonstrates that post-viral pulmonary fibrosis in children constitutes a clinically significant complication of severe viral pneumonia whose prevention requires a proactive multidisciplinary approach integrating vaccination, lung-protective ventilation, early respiratory rehabilitation, and structured follow-up. Critical gaps in the paediatric evidence base and perspectives for Central Asian research are identified.

Keywords: Pulmonary fibrosis, viral pneumonia, children, post-infectious complications, prevention, SARS-CoV-2, TGF-β, respiratory rehabilitation, literature review.

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1. INTRODUCTION

Viral pneumonia remains one of the leading causes of morbidity and mortality in children worldwide, accounting for the majority of pneumonia-related hospital admissions in the paediatric age group (Rudan et al., 2013; McAllister et al., 2019). The principal viral aetiological agents — RSV, influenza A and B, human metapneumovirus, adenovirus, and SARS-CoV-2 — produce a spectrum of pulmonary injury ranging from mild bronchiolitis to severe diffuse alveolar damage and ARDS (Jain et al., 2015; Shi et al., 2020). While the majority of viral pneumonias in children resolve without permanent sequelae, a clinically important subset results in persistent structural alterations of the lung parenchyma, including post-infectious pulmonary fibrosis (PIPF) (Stein et al., 2017; George et al., 2020).

PIPF is characterised by excessive deposition of collagens I and III in the alveolar walls and interstitium, driven by dysregulated repair mechanisms following viral injury. Clinical consequences include restrictive ventilatory impairment, impaired gas exchange, and, in severe cases, progressive respiratory failure (Sgalla et al., 2018; Wynn, 2011). The COVID-19 pandemic has substantially intensified scientific interest in this topic, with emerging data documenting radiological fibrotic changes in children following severe COVID-19-associated pneumonia and MIS-C (Granot et al., 2021; Ojo et al., 2020). In Uzbekistan and the Fergana region, systematic data on PIPF incidence and prevention in children are lacking. The aim of this review is to synthesise current evidence on the pathogenesis, risk factors, and prevention of post-viral pulmonary fibrosis in children.

2. PATHOGENETIC MECHANISMS

The pathogenesis of PIPF involves a complex cascade of cellular and molecular events initiated by viral injury to the alveolar epithelium (Wynn, 2011; Sgalla et al., 2018). Type I and type II alveolar epithelial cells undergo necrosis and apoptosis following direct viral cytopathic effects, activating innate immune responses with a surge of pro-inflammatory cytokines including IL-1β, IL-6, and TNF-α. Under physiologically regulated repair conditions, type II epithelial cells

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proliferate to restore the alveolar surface. However, when the inflammatory stimulus is severe — as in ARDS or infection by highly cytopathic viruses such as adenovirus and SARS-CoV-2 — the repair process is dysregulated, with myofibroblasts producing excessive extracellular matrix under TGF-β1 stimulation (Bhatt et al., 2020).

Age-specific features of the paediatric immune response, particularly the Th2-skewed cytokine microenvironment with elevated IL-4 and IL-13, are inherently pro-fibrotic, potentially amplifying the fibrogenic response in young children (Wynn, 2011). In COVID-19, the cytokine storm with massive IL-6 and IL-1β release leads to diffuse alveolar damage and subsequent fibrosis, while spike protein-mediated ACE2 downregulation provides an additional pro-fibrotic mechanism independent of inflammation severity (Granot et al., 2021).

3. RISK FACTORS FOR PIPF IN CHILDREN

Clinical risk factors include: ARDS with PaO2/FiO2 ratio below 200 mmHg, invasive mechanical ventilation — particularly with high tidal volumes and elevated FiO2 — adenoviral and COVID-19-associated aetiology, and bacterial superinfection (Stein et al., 2017; Bhatt et al., 2020). Adenoviral pneumonia historically carries the highest risk of permanent pulmonary sequelae in children, with reported rates of 15–60% in hospitalised cohorts.

Host factors include: age under 2 years, prematurity and bronchopulmonary dysplasia, primary immunodeficiency disorders, and genetic polymorphisms in TGF-β1 and IL-6 genes. Nutritional deficiencies — particularly vitamin D and zinc deficiency, prevalent in the Fergana region — may impair epithelial repair capacity, warranting prospective investigation in the regional context.

4. EARLY DETECTION OF FIBROTIC CHANGES

High-resolution computed tomography (HRCT) of the chest remains the gold standard for detecting fibrotic lung changes, identifying ground-glass opacities, reticulation, and traction bronchiectasis at early stages (Bhatt et al., 2020; Sonnweber et al., 2021). Low-dose protocols should be employed in children. Pulmonary function testing — spirometry, bodyplethysmography, and DLCO —

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provides objective documentation of restrictive ventilatory patterns and impaired gas exchange. In children under 5 years, impulse oscillometry represents a validated alternative. Serum biomarkers including KL-6, SP-D, and MMP-7 have shown diagnostic utility in adult PIPF but require validation in paediatric cohorts (Sgalla et al., 2018).

5. PHARMACOLOGICAL PREVENTION STRATEGIES

The evidence base for pharmacological prevention of PIPF in children remains limited. Systemic corticosteroids used in severe viral pneumonias with ARDS demonstrate anti-inflammatory effects, but their impact on long-term fibrotic outcomes in children is not established by randomised controlled trials (Bhatt et al., 2020). Anti-fibrotic agents — pirfenidone and nintedanib — licensed for idiopathic pulmonary fibrosis in adults, have not been studied in paediatric PIPF prevention and cannot be recommended outside clinical trials (Sgalla et al., 2018). Macrolide antibiotics, particularly azithromycin, demonstrate immunomodulatory and anti-fibrotic properties including TGF-β expression reduction, representing a promising research direction for paediatric populations (George et al., 2020). N-acetylcysteine as an antioxidant and mucolytic agent warrants prospective investigation for reducing oxidative stress-mediated fibrosis in post-viral paediatric settings.

6. NON-PHARMACOLOGICAL PREVENTION STRATEGIES

Vaccination against principal viral respiratory pathogens represents the most important primary prevention measure. Annual influenza vaccination, pneumococcal vaccination, COVID-19 vaccination in eligible age groups, and RSV prophylaxis with palivizumab in high-risk infants collectively reduce the incidence and severity of viral pneumonias and thereby the population-level burden of PIPF (McAllister et al., 2019). Expanding vaccination coverage in the Fergana region represents a priority public health action.

Lung-protective mechanical ventilation — tidal volume limitation to 6 mL/kg ideal body weight, plateau pressure below 28–30 cmH2O, and FiO2 minimisation — is the most evidence-based intervention for reducing ventilator-induced lung

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injury and fibrotic risk in children requiring intensive respiratory support (Bhatt et al., 2020). Respiratory rehabilitation in the early post-acute period — breathing exercises, airway clearance techniques, and graduated aerobic training — is recommended for children with post-viral restrictive lung function deficits, supported by evidence from adult post-COVID-19 and post-ARDS cohorts (Antoniou et al., 2022; Sonnweber et al., 2021). Nutritional support and correction of vitamin D and zinc deficiencies are recommended as adjunctive measures for supporting epithelial repair capacity.

7. PERSPECTIVES FOR FERGANA REGION AND UZBEKISTAN

Development of evidence-based PIPF prevention strategies in the Fergana region and Uzbekistan is constrained by limited HRCT availability in regional paediatric facilities, absence of structured post-viral pneumonia follow-up programmes, and lack of published epidemiological data on PIPF incidence in Uzbek children. Priority actions include establishing structured follow-up protocols for at-risk children, investing in paediatric respiratory diagnostics capacity at tertiary centres, and initiating prospective observational studies. The high prevalence of vitamin D deficiency and relatively low influenza vaccination coverage in the Fergana region represent key modifiable risk factors for targeted intervention.

8. CONCLUSION

Post-viral pulmonary fibrosis represents a clinically significant complication of severe viral pneumonia in children, whose pathogenesis involves TGF-β1-driven dysregulated alveolar repair. Risk factors include ARDS severity, invasive mechanical ventilation, adenoviral and SARS-CoV-2 aetiology, early age, and immunodeficiency. Prevention rests on a multi-level strategy encompassing vaccination, lung-protective ventilation, early respiratory rehabilitation, and systematic post-discharge HRCT follow-up in at-risk children. Development of regionally adapted prevention protocols for paediatric patients in the Fergana region and Uzbekistan is an urgent research and clinical priority.

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