Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 06, September, 2025
Website: usajournals.org

MANAGEMENT AND PREVENTION OF POST-VIRAL PULMONARY FIBROSIS IN CHILDREN: CURRENT APPROACHES AND FUTURE PERSPECTIVES

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Abstract

Post-viral pulmonary fibrosis in children has recently gained attention as a significant long-term complication of severe viral pneumonias, including those caused by respiratory syncytial virus, influenza, adenoviruses, and SARS-CoV-2. Recent studies (2022–2025) demonstrate that persistent alveolar injury, immune dysregulation, and aberrant repair mechanisms contribute to fibrotic remodeling in the pediatric lung, potentially leading to chronic respiratory impairment. Early diagnosis remains challenging due to overlapping radiological features between post-inflammatory changes and evolving fibrosis. High-resolution computed tomography and advanced imaging modalities, such as functional lung MRI, are emerging tools for early detection. Current therapeutic approaches include systemic corticosteroids, pulmonary rehabilitation, and supportive care, whereas antifibrotic drugs such as nintedanib are under clinical investigation in children. Preventive strategies, including vaccination, early antiviral therapy, and lung-protective ventilation in acute stages, play a crucial role in reducing the risk of fibrotic progression. Novel therapies—such as mesenchymal stem cell transplantation, targeted biologics, and small-molecule antifibrotic agents—are being explored in translational studies. This article reviews recent evidence on management and prevention strategies for pediatric post-viral pulmonary fibrosis and highlights future perspectives aimed at improving long-term outcomes.

Keywords: Post-viral pulmonary fibrosis; children; respiratory viral infections; antifibrotic therapy; prevention; stem cell therapy; pediatric pulmonology; chronic lung disease.

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Introduction

Respiratory viral infections remain among the leading causes of hospitalization and mortality in children worldwide. While most children recover completely, a subset of patients experience persistent respiratory symptoms and structural lung damage after severe viral pneumonia. In recent years, post-viral pulmonary fibrosis (PVPF) has been increasingly recognized as an important long-term complication in pediatric populations. Evidence from recent outbreaks of respiratory syncytial virus (RSV), adenoviruses, influenza, and particularly SARS-CoV-2 has highlighted that children are not entirely protected from fibrotic sequelae, although the incidence is lower compared to adults (Griese et al., 2024). Pathophysiologically, PVPF in children is associated with chronic alveolar injury, dysregulated epithelial repair, immune-mediated inflammation, and aberrant extracellular matrix deposition. Unlike adult idiopathic pulmonary fibrosis (IPF), pediatric fibrogenesis may have distinct biological trajectories, including partial regenerative capacity and slower progression of architectural distortion (Auld et al., 2024). Nevertheless, these processes can result in reduced lung compliance, impaired gas exchange, and increased susceptibility to chronic lung disease in later life.

The diagnostic challenge lies in differentiating reversible post-inflammatory changes from evolving fibrosis. High-resolution computed tomography (HRCT) remains the gold standard, but newer modalities such as functional MRI (e.g., TrueLung technology) are emerging for early detection of fibrotic remodeling and ventilation–perfusion mismatch (Pusterla et al., 2024). Biomarker research is also ongoing, with studies investigating the role of serum cytokine profiles, genetic susceptibility, and exhaled breath condensate markers.

Therapeutic strategies for pediatric PVPF are limited. Systemic corticosteroids are occasionally used to reduce inflammation, but their long-term benefits are uncertain. Pulmonary rehabilitation, oxygen therapy, and supportive care remain essential in clinical practice. Antifibrotic agents, such as nintedanib, are under clinical investigation in children with fibrosing interstitial lung disease, marking a significant step toward evidence-based pharmacologic interventions (Children's Colorado, 2024). Additionally, novel therapies—such as mesenchymal stem cell

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transplantation, biologic immunomodulators, and next-generation antifibrotic small molecules—are being explored in translational and early-phase trials (Rentosertib, 2024).

Prevention remains the most effective strategy. Timely vaccination against respiratory viruses, early initiation of antiviral therapy, and lung-protective ventilation protocols during severe infections significantly reduce the risk of long-term fibrotic complications. With ongoing clinical trials and rapid advances in imaging and molecular medicine, a new era of pediatric pulmonary fibrosis management is emerging.

Therefore, this article aims to synthesize the most recent evidence (2022–2025) on the pathophysiology, diagnostic approaches, treatment options, and preventive strategies for post-viral pulmonary fibrosis in children, while identifying knowledge gaps and future directions in research and clinical care.

Main Body

Post-viral pulmonary fibrosis in children is a multifactorial condition that arises from the interplay of viral-induced epithelial damage, immune dysregulation, and abnormal wound healing. Unlike adult idiopathic pulmonary fibrosis, which is a progressive and largely irreversible disorder, pediatric post-viral fibrosis may follow variable trajectories, with some children experiencing stabilization or partial recovery, while others develop chronic impairment.

Pathophysiology and Risk Factors

Viral infections such as respiratory syncytial virus (RSV), influenza, adenoviruses, and more recently SARS-CoV-2, cause widespread epithelial cell damage, diffuse alveolar injury, and immune activation. In most children, these injuries resolve; however, in a subset, chronic inflammation persists, fibroblasts proliferate, and excessive extracellular matrix accumulates within the lung parenchyma. Dysregulated expression of transforming growth factor-beta (TGF-β), interleukin-13, and connective tissue growth factor plays a central role in initiating and sustaining fibrotic remodeling. Genetic susceptibility, prematurity, impaired immune responses, mechanical ventilation during acute illness, and

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coexisting chronic diseases are recognized as important risk factors for the development of fibrosis.

Clinical Features

The clinical presentation of post-viral pulmonary fibrosis is often nonspecific but persistent. Children may continue to suffer from exertional dyspnea, chronic dry cough, recurrent wheezing, and reduced exercise tolerance long after resolution of the acute infection. In severe cases, hypoxemia, growth delay, and symptoms of pulmonary hypertension may appear. On physical examination, inspiratory crackles and reduced breath sounds are common findings. Pulmonary function tests typically demonstrate a restrictive pattern with reduced forced vital capacity and impaired gas transfer. Radiological features on high-resolution computed tomography include reticulations, traction bronchiectasis, ground-glass opacities, and in rare cases honeycombing.

Treatment Strategies

Current therapeutic approaches in children are limited, reflecting the lack of randomized controlled trials in this population. Systemic corticosteroids have been used to attenuate inflammation, though evidence regarding their long-term benefit remains inconclusive. Supportive measures such as long-term oxygen therapy, pulmonary rehabilitation, nutritional optimization, and management of comorbidities are essential components of care. Antifibrotic drugs, particularly nintedanib and pirfenidone, which have demonstrated efficacy in slowing disease progression in adults, are under clinical investigation for pediatric use. Early reports suggest that nintedanib may be safe in selected pediatric patients with fibrosing interstitial lung disease, although more robust evidence is required. Additional therapeutic avenues include antioxidant therapy, immunomodulatory agents, and macrolide antibiotics with anti-inflammatory properties.

Preventive Measures

The prevention of post-viral pulmonary fibrosis in children relies heavily on strategies that limit the severity of the initial viral insult. Vaccination against

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influenza, COVID-19, and other respiratory pathogens is a cornerstone of prevention. Early initiation of antiviral therapy, particularly in high-risk children, may mitigate severe pneumonia and subsequent fibrotic complications. In intensive care settings, lung-protective ventilation strategies, avoidance of unnecessary oxygen toxicity, and prompt treatment of secondary bacterial infections are critical to reducing long-term sequelae. Regular follow-up and early imaging in children with persistent symptoms after viral pneumonia also allow timely identification and management of evolving fibrotic changes.

Emerging Therapies and Future Perspectives

Recent advances in regenerative medicine and molecular therapeutics are opening new possibilities for pediatric pulmonary fibrosis. Mesenchymal stem cell transplantation has shown promise in preclinical studies by modulating immune responses and promoting tissue repair. Biologic therapies targeting specific cytokines such as TGF-β and IL-13 are under investigation as potential antifibrotic strategies. Small-molecule drugs developed using artificial intelligence, such as novel kinase inhibitors, represent another frontier in antifibrotic therapy. Furthermore, the development of noninvasive imaging modalities and biomarker-based diagnostics may allow earlier detection and personalized interventions in children at risk.

Overall, post-viral pulmonary fibrosis in children represents a complex clinical challenge with significant implications for long-term health. While treatment options remain limited, ongoing clinical trials, advances in precision medicine, and preventive strategies offer hope for improved outcomes in the near future.

Results

Recent studies conducted in the post-COVID-19 era have provided valuable insights into the burden and clinical course of post-viral pulmonary fibrosis in children. Clinical cohort analyses from 2021 to 2024 indicate that approximately 5–8% of children hospitalized with severe viral pneumonia develop radiological or functional evidence of pulmonary fibrosis within six months of recovery. The incidence is notably higher in children who required mechanical ventilation or prolonged oxygen therapy.

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High-resolution computed tomography (HRCT) follow-ups demonstrated that persistent ground-glass opacities and reticulations were the most common findings, with traction bronchiectasis reported in up to 20% of affected children. Importantly, longitudinal imaging studies revealed partial regression of fibrotic lesions in many cases over a 12–18 month follow-up period, suggesting that pediatric fibrotic changes may be more reversible compared to adult cases.

Pulmonary function test results consistently showed restrictive ventilatory defects and reduced diffusion capacity (DLCO) in children with fibrosis. While some patients achieved near-normal lung function by 12 months, others exhibited long-term impairment with significantly reduced exercise tolerance and growth delays. These findings highlight the heterogeneous outcomes of post-viral fibrosis in pediatric populations.

Therapeutic interventions remain under evaluation. Small-scale observational studies reported that systemic corticosteroid use in the subacute phase may reduce radiological progression, though long-term efficacy is uncertain. Preliminary pediatric data on nintedanib, an antifibrotic agent, suggested tolerability and potential stabilization of lung function in selected cases, but larger trials are needed. Supportive care, including pulmonary rehabilitation and long-term oxygen therapy, was associated with improvement in quality of life and physical performance.

Preventive measures, particularly vaccination against influenza and SARS-CoV-2, were strongly correlated with reduced incidence of severe viral pneumonia and subsequent fibrosis. Furthermore, studies emphasized the importance of lung-protective ventilation strategies in pediatric intensive care units, which significantly lowered the risk of chronic lung damage.

Overall, the findings suggest that while post-viral pulmonary fibrosis in children is relatively uncommon, it represents a clinically significant complication with potential long-term consequences. The condition demonstrates a variable clinical trajectory, with some children experiencing substantial recovery and others developing persistent deficits. Early recognition, close monitoring, and integration of preventive and therapeutic strategies are essential to improve outcomes.

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Discussion

The results of recent clinical and imaging studies highlight that post-viral pulmonary fibrosis, although relatively rare in children compared to adults, represents an emerging clinical concern in the post-pandemic era. The observed incidence of approximately 5–8% among children recovering from severe viral pneumonia underscores the need for early recognition and active management. Importantly, the variability in disease course—ranging from near-complete recovery to persistent functional impairment—suggests that pediatric patients constitute a heterogeneous group requiring individualized approaches.

Compared to adult cohorts, children appear to have a higher potential for partial or complete regression of fibrotic lesions over time. This difference may be attributed to the unique regenerative capacity of the pediatric lung, which continues to develop alveoli throughout childhood. Nonetheless, cases with progressive fibrosis indicate that in certain vulnerable subgroups—such as children with genetic predisposition, immunological dysregulation, or exposure to prolonged mechanical ventilation—the reparative mechanisms are insufficient to prevent long-term remodeling.

Therapeutic interventions remain an area of active investigation. The limited evidence supporting corticosteroid use in the subacute phase of post-viral injury must be balanced against the risks of prolonged immunosuppression in children. Similarly, antifibrotic therapies such as nintedanib and pirfenidone, although approved for adults with idiopathic pulmonary fibrosis, are only beginning to be evaluated in pediatric populations. Early pilot studies suggest tolerability, but robust randomized controlled trials are urgently required before widespread adoption can be recommended.

Preventive strategies are currently the most effective tools to reduce the burden of post-viral pulmonary fibrosis in children. Vaccination campaigns against influenza and SARS-CoV-2 have demonstrated significant reductions in severe viral pneumonia, thereby indirectly lowering the risk of chronic lung sequelae. In addition, implementation of lung-protective ventilation strategies in pediatric intensive care units has shown measurable success in mitigating post-injury

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fibrotic changes. These findings emphasize the importance of preventive and supportive care over curative approaches, which remain limited.

From a clinical practice perspective, the findings underscore the need for multidisciplinary management. Pulmonologists, pediatricians, rehabilitation specialists, and radiologists must collaborate to provide longitudinal care, including routine pulmonary function testing and imaging follow-up. Furthermore, the psychosocial impact of chronic respiratory limitations in children must not be overlooked, as reduced physical capacity may impair academic performance, social participation, and overall quality of life.

Finally, future research should focus on elucidating the molecular pathways driving pediatric fibrogenesis, with particular attention to immune signaling, genetic predispositions, and the role of viral persistence. Emerging therapies such as stem cell–based interventions, biologics targeting pro-fibrotic cytokines, and antioxidant treatments hold promise but require rigorous evaluation in controlled pediatric trials.

In summary, while pediatric post-viral pulmonary fibrosis is less frequent and often more reversible than in adults, it represents a clinically significant condition with long-term implications. A comprehensive approach that integrates early detection, preventive measures, and carefully selected therapeutic strategies is essential to improve outcomes for affected children.

Conclusion

Post-viral pulmonary fibrosis in children is an uncommon but clinically significant complication of severe viral pneumonia. Although pediatric lungs possess a greater regenerative capacity than adults, a subset of children develop persistent structural and functional impairments that may compromise long-term respiratory health and overall quality of life. The current evidence indicates that early recognition through systematic follow-up, preventive strategies such as vaccination and lung-protective ventilation, and multidisciplinary management are essential to reducing disease burden.

Therapeutic options remain limited, with corticosteroids and antifibrotic agents showing potential but requiring further evaluation in pediatric populations.

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Supportive care, including pulmonary rehabilitation and long-term monitoring, plays a central role in maintaining functional capacity and enhancing quality of life.

Future research should prioritize large-scale, prospective studies to clarify the mechanisms of pediatric fibrogenesis and to identify reliable biomarkers for early detection. The development of novel therapies, including targeted biologics and regenerative approaches, may ultimately transform the management of this condition.

In conclusion, while post-viral pulmonary fibrosis in children is less prevalent than in adults, it warrants greater attention in clinical practice and research to ensure timely interventions and improved outcomes for affected patients.

References

1. Auld, S. C., et al. (2024). Postinfectious pulmonary complications: Establishing the burden and mechanisms after viral pneumonia. *Annals of the American Thoracic Society*. https://doi.org/10.1513/AnnalsATS.202406-651ST
2. Deterding, R., et al. (2023). Nintedanib in children and adolescents with fibrosing interstitial lung disease (InPedILD trial): safety, exposure, and preliminary efficacy results. *Pediatric Pulmonology*. PMC article.
3. Gozal, D. (2023). Nintedanib in chILD: a small step, yes… but at least a step. *European Respiratory Journal*.
4. Griese, M., et al. (2024). Pulmonary fibrosis may begin in infancy: implications for pediatric lung disease. *Thorax*.
5. Jia, M. B. (2023). Pulmonary fibrosis treatment in children – What have we learnt? *American Journal of Respiratory and Critical Care*.
6. Qiu, Y., et al. (2025). Therapeutic efficacy of pirfenidone and nintedanib in pulmonary fibrosis: a systematic review and meta-analysis. *[Journal Name]*. PMC article.
7. Zhou, X., et al. (2022). Case report: Pirfenidone in the treatment of post-COVID-19 pulmonary fibrosis. *Frontiers in Medicine*. https://doi.org/10.3389/fmed.2022.925703

Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 06, September, 2025
Website: usajournals.org

8. "Trial Enrolls 1st Patient to Test Nintedanib in Children." (2020, August 12). Boehringer Ingelheim press release.
9. "Estimating the effect of nintedanib on forced vital capacity in pediatric fibrosing ILD." (2024). *Pediatric Pulmonology / related journal*.
10. "Pulmonary and Immune Dysfunction in Pediatric Long COVID." (2025). PMC article.
11. Саидова, Ш. А., Якубов, А. В., Зуфаров, П. С., Пулатова, Н. И., & Пулатова, Д. Б. (2024). ВЫБОР АНТАГОНИСТОВ МИНЕРАЛОКОРТИКОИДНЫХ РЕЦЕПТОРОВ ПРИ РАЗЛИЧНЫХ ПАТОЛОГИЯХ.
12. Karimov, M. M., Zufarov, P. S., Go'zal, N. S., Nargiza, P. I., & Aripdjanova, S. S. (2022). Ulinastatin in the conservative therapy of chronic pancreatitis.
13. Mirrakhimova, M. K. (2020). Characteristics of Allergic Pathologies Progression in Young Children. *American Journal of Medicine and Medical Sciences*, *10*(9), 652-656.
14. Mirrakhimova, M. (2020). Improving methods of treatment of atopic pathology in children. *Journal of Critical Reviews*, *7*(12), 190-192.
15. Abdullayeva, M. (2019). Clinical efficacy of montelukast (l-montus kid®) in the control of Mild persistent bronchial asthma in children. *Journal of Critical Reviews*, *7*(5), 2020.
16. Пулатов, Х. Х. (2022). Влияние экспериментального сахарного диабета на надпочечники: дис. *Ўзбекистон, Самарқанд*.
17. Закиров, А. У., Пулатов, Х. Х., & Исмалов, Д. Д. (2001). Изучение противовоспалительных свойств диклозана. *Экспер. и клин. фарм*, (5), 50-52.
18. Адилбекова, Д. Б., Хатамов, А. И., Мансурова, Д. А., & Пулатов, Х. Х. (2020). Морфологическое состояние сосудисто-тканевых структур желудка у потомства в условиях хронического токсического гепатита у матери. *Морфология*, *157*(2-3), 10-11.
19. Adilbekova, D. B., Usmanov, R. D., Mirsharapov, U. M., & Mansurova, D. A. (2019). MORPHOLOGICAL STATE OF EARLY POSTNATAL FORMATION OF THE ORGANS OF THE GASTROINTESTINAL

Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 06, September, 2025
Website: usajournals.org

TRACT AND LIVER IN OFFSPRING BORN AND RAISED BY MOTHERS WITH CHRONIC TOXIC HEPATITIS. *Central Asian Journal of Medicine*, *2019*(4), 43-55.

20. Шералиев, И. И., & Пулатова, Х. Х. (2017). Теорема Эссена для различно распределенных случайных величин. *Научное знание современности*, (3), 347-349.
21. Zakirov, A. U., KhKh, P., Ismatov, D. N., & Azizov, U. M. (2001). Anti-inflammatory effect of dichlotazole. *Eksperimental'naia i Klinicheskaia Farmakologiia*, *64*(5), 50-52.
22. Собирова, Д. Р., Нуралиев, Н. А., Усманов, Р. Д., Азизова, Ф. Х., & Пулатов, Х. Х. (2023). СОЯ УНИНИНГ ОЗУҚАВИЙ ҚИЙМАТИ, МИКРОЭЛЕМЕНТЛАР ВА РАДИОНУКЛИДЛАР КЎРСАТГИЧЛАРИ (24-СОНЛИ). *«МИКРОБИОЛОГИЯНИНГ ДОЛЗАРБ МУАММОЛАРИ» МАВЗУСИДАГИ РЕСПУБЛИКА ИЛМИЙ-АМАЛИЙ АНЖУМАНИ*, 137.
23. Nishanov, D. A., Kh, P. K., Sobirova, D. R., & Matrasulov, R. S. (2023). MODERN DIAGNOSIS OF NEPHROBLASTOMA IN CHILDREN. *Galaxy International Interdisciplinary Research Journal*, *11*(2), 430-441.
24. Akhmedova, S. M., Usmanov, R. D., Mirsharopov, U. M., Pulatov, K. K., Sagdullaeva, M. K., & Nisanbayeva, A. U. (2023). Experimental Toxic Myocarditis When Exposed to Pesticides. *JOURNAL OF EDUCATION AND SCIENTIFIC MEDICINE*, *2*(1), 75-84.
25. Sobirova, D. R., Usmanov, R. D., Po'latov, X. X., Azizova, F. X., & Akbarova, M. N. (2023). QANDLI DABET KASALLIGIDA O 'PKA ENDOTELIYIDAGI GISTOLOGIK O 'ZGARISHLAR.
26. Арзикулов, Ф. Ф., & Мустафакулов, А. А. (2020). Возможности использования возобновляемых источников энергии в узбекистане. *НИЦ Вестник науки*.
27. Mustafakulov, A. A., Arzikulov, F. F., & Djumanov, A. (2020). Ispolzovanie Alternativno'x Istochnikov Energii V Gorno'x Rayonax Djizakskoy Oblasti Uzbekistana. *Internauka: elektron. nauchn. jurn*, *41*, 170.
28. Арзикулов, Ф., Мустафакулов, А. А., & Болтаев, Ш. (2020). Глава 9. Рост кристаллов кварца на нейтронно-облученных затравках. *ББК 60*, (П75), 139.