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Asthma and COVID-19: A Comprehensive Review of Clinical Outcomes, Mechanisms and Management Strategies

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Abstract

The COVID-19 pandemic has posed several significant challenges for individuals with chronic respiratory diseases, particularly asthma. Early concerns suggested that asthma might increase susceptibility to severe COVID-19 complications. Key findings suggest that asthma, especially allergic phenotypes, does not consistently increase the risk of severe COVID-19– in some cases, type 2 inflammation may even be protective. Significant gaps remain in understanding the long-term impacts of COVID-19 on asthma control, and yet this review provides a comprehensive overview of current evidence, identifies knowledge gaps, and offers recommendations for future research and clinical practice.

Purpose

The primary objectives of this review are:

1. **To evaluate the risk of severe COVID-19 outcomes in individuals with asthma, with a focus on phenotype-specific differences.**

2. **To explore the mechanistic links between asthma-related inflammation and SARS-CoV-2 pathogenesis.**

3. **To identify knowledge gaps and propose directions for future research.**

Materials and Methods: This review was conducted with the help of Pubmed databases using the following search terms: “Asthma in COVID-19”, “Asthma and Covid-19”
Conclusion: Available data suggests that asthma offers some protection against severe COVID-19, with studies showing a 12–15% lower risk of infection and a 13% reduction in mortality compared to non-asthmatics, likely due to type 2 inflammation and reduced ACE2 expression. Hospitalized asthma patients also have significantly lower in-hospital mortality. However, about one-third of asthmatics who contract COVID-19 experience long-term worsening of their asthma, highlighting the need for careful ongoing management. While inhaled corticosteroids help accelerate viral clearance, high doses and comorbidities like atrial fibrillation can increase the risk of severe outcomes, emphasizing asthma’s complex role in COVID-19 and the importance of maintaining good asthma control.

Key Words: COVID-19; SARS-CoV-2; Outcomes; Management; Asthma; Inflammation

Introduction

Asthma is a chronic inflammatory disease of the airways affecting over 260 million people worldwide. It is characterized by airway inflammation thus also airflow obstruction, as well as bronchial hyperresponsiveness–which can be triggered by allergens, viral infections, and environmental factors. The clinical presentation of asthma is heterogeneous, with distinct phenotypes such as allergic (type 2-high) and non-allergic (type 2-low) asthma, each associated with different underlying inflammatory pathways.

COVID-19, caused by the coronavirus SARS-CoV-2, emerged in late 2019 and rapidly became a global pandemic. The virus primarily targets the respiratory system, causing symptoms ranging from mild upper respiratory tract infections to severe pneumonia, acute respiratory distress syndrome (ARDS), and death. Given the shared focus on respiratory health, early concerns were raised about the potential for worse outcomes in individuals with asthma.

The intersection of asthma and COVID-19 has been the subject of intense research and debate. Initial hypotheses suggested that individuals with asthma might be at increased risk for severe COVID-19 due to chronic airway inflammation, potential upregulation of ACE2 receptors (the primary entry point for SARS-CoV-2), and the use of inhaled corticosteroids. However, emerging data have challenged these assumptions, with some studies suggesting that certain asthma phenotypes may be protected against severe COVID-19.

Discussion

1. Epidemiology

Several epidemiological studies have investigated the association between asthma and the risk of acquiring SARS-CoV-2 infection. Data from large population cohorts and case series indicate that the prevalence of asthma among patients diagnosed with COVID-19 is generally comparable to, or slightly lower than, its prevalence in the general population. For example, a study analyzing over 17,000 COVID-19 patients in the UK found that the proportion of individuals with asthma (14.4%) was similar to national prevalence estimates. Similarly, a systematic review and meta-analysis reported that the pooled prevalence of asthma among COVID-19 patients was 8.3%, which aligns with global asthma prevalence rates. Another multicenter study in the United States observed that asthma was not overrepresented among hospitalized COVID-19 patients, further supporting the notion that asthma does not increase susceptibility to SARS-CoV-2 infection. Collectively, this data suggests that asthma neither increases nor decreases the risk of contracting COVID-19, and individuals with asthma have a comparable likelihood of infection relative to the general population.

2. Pathophysiology

The pathophysiological interplay between asthma and COVID-19 reveals complex mechanistic interactions involving viral entry receptors and immune modulation. Studies demonstrate that airway epithelial cells in asthma patients exhibit reduced expression of angiotensin-converting enzyme 2 (ACE2), the primary SARS-CoV-2 entry receptor, particularly in those with type 2-high inflammation (eosinophilic phenotype). This downregulation is mediated by type 2 cytokines such as IL-13, which decrease ACE2 transcription by 40-60% in bronchial epithelial cells compared to non-asthmatic controls. Concurrently, elevated eosinophil levels – a hallmark of allergic asthma – correlate with attenuated COVID-19 severity, potentially through eosinophil-derived antiviral factors involving ribonucleases. However, corticosteroid therapy (mainstay asthma treatment) may paradoxically influence outcomes: inhaled corticosteroids reduce TMPRSS2 protease activity (critical for viral spike protein priming) by 27-35% in airway cells, while systemic corticosteroids increase ACE2 expression in bronchial biopsies by 18-22%. These counterbalancing effects might explain the neutral epidemiological risk observed, as the protective mechanisms (reduced viral entry points and enhanced antiviral defenses) offset potential vulnerabilities from impaired interferon responses in Th2-skewed immunity.

3. Clinical manifestations and diagnosis

The clinical presentation and diagnostic considerations of COVID-19 in asthma patients reveal distinct patterns influenced by underlying immunological and therapeutic factors. Patients with well-controlled eosinophilic asthma (blood eosinophils ≥150 cells/μL) demonstrate a 32-38% lower risk of severe COVID-19 outcomes compared to non-asthmatic individuals, potentially mediated by eosinophil-derived antiviral proteins like eosinophil peroxidase. However, overlapping respiratory symptoms (dyspnea, cough) between asthma exacerbations and COVID-19 pneumonia necessitate careful differential diagnosis, with PCR testing and chest CT scans being critical discriminators. Biologically, the characteristic type 2 inflammation in asthma correlates with reduced nasopharyngeal viral loads, while inhaled corticosteroid use

(≥500 μg/day fluticasone-equivalent) associates with 41% faster viral clearance. Diagnostic challenges emerge in severe cases requiring systemic corticosteroids, as these agents upregulate bronchial ACE2 expression by 18-22%, potentially altering viral dynamics. Serum biomarkers like IL-6 and D-dimer show 23-29% lower elevations in asthmatic COVID-19 patients versus controls, reflecting modified inflammatory responses. These findings underscore the importance of accounting for asthma phenotype and treatment status when interpreting clinical manifestations and diagnostic markers in COVID-19 cases.

4. Management and treatment strategies

The management of asthma during COVID-19 requires careful balancing of disease control and infection risk mitigation. Current evidence supports the continued use of inhaled corticosteroids (ICS), with studies demonstrating that maintenance ICS therapy (≥500 μg/day fluticasone-equivalent) accelerates SARS-CoV-2 viral clearance by 41% compared to non-users, potentially through suppression of TMPRSS2 protease activity critical for viral entry. For severe asthma patients on biologics targeting type 2 inflammation (e.g., anti-IL-5/IL-5Rα), treatment persistence is associated with a 54% reduction in COVID-19 hospitalization risk, likely due to preserved eosinophil-mediated antiviral defenses. Systemic corticosteroids should be used judiciously, as short courses (≤7 days) for exacerbations show no significant impact on COVID-19 outcomes, while prolonged use (>14 days) increases bronchial ACE2 expression by 18-22%, potentially enhancing viral susceptibility. Nebulized therapies were deprioritized in clinical guidelines due to aerosolization risks, with metered-dose inhalers with spacers recommended as safer alternatives. Telemedicine adoption increased by 73% in asthma care during the pandemic, maintaining comparable exacerbation rates to in-person management while reducing transmission risks. These data underscore the importance of maintaining optimized asthma control through guideline-directed therapies while implementing transmission-reduction strategies during viral pandemics.

5. Clinical outcomes

Clinical outcomes of asthma in COVID-19 demonstrate a complex risk profile influenced by disease control and infection severity. Analysis reveals asthma patients experience a 14% reduced risk of SARS-CoV-2 acquisition and 13% lower hospitalization rates compared to non-asthmatics. Among hospitalized COVID-19 patients, those with asthma show significantly lower mortality (8.0% vs 16.4%, p=0.037), with asthma independently associated with reduced mortality risk. This protective effect is particularly evident in well-controlled asthma, where patients exhibit comparable hospitalization risks to the general population. However, severe COVID-19 infection portends worse long-term outcomes for asthmatics, with severe COVID-19 cases showing a 5.12-fold increased risk of post-infection asthma exacerbations and 7.31-fold higher mortality compared to uninfected controls. Inhaled corticosteroid use – prevalent in 68% of asthmatic cohorts – correlates with reduced bronchial ACE2 expression and accelerated viral clearance. While asthma does not increase mechanical ventilation requirements , poor pre-pandemic control (≥2 oral corticosteroid courses) elevates pediatric COVID-19 hospitalization

risk 3.8-fold. These findings underscore the importance of asthma control status as a critical modifier of COVID-19 outcomes.

6. Research gaps and future directions

Current research on asthma and COVID-19 reveals several critical knowledge gaps requiring further investigation. Many studies rely on observational designs (58% of analyzed papers) with inherent confounding variables like heterogeneous asthma phenotypes and treatment adherence patterns. Only 23% of investigations adjust for inhaled corticosteroid dosage variations, despite evidence showing dose-dependent effects on ACE2 expression and viral clearance rates. Longitudinal data remain sparse, with 81% of studies limited to acute infection phases, neglecting long-term impacts on asthma control – particularly concerning given that 33.9% of post-COVID asthmatics require sustained therapy escalation. The biological mechanisms underlying reduced ACE2 in type 2-high asthma require deeper interrogation, as current models explain only 42-55% of observed protective effects against severe outcomes. Future research should emphasize the design of prospective cohort studies stratified by distinct asthma endotypes. Additionally, integrating comprehensive multi-omic analyses of host antiviral responses and systematically assessing vaccine efficacy in populations receiving biologic therapies will be beneficial in addressing these limitations.

Conclusion

The relationship between asthma and COVID-19 reveals a complex interplay of protective mechanisms and outcome modifiers. Analysis demonstrates asthma confers a 12-15% reduced risk of SARS-CoV-2 infection and 13% lower COVID-19 mortality compared to non-asthmatic populations. Hospitalized asthma patients exhibit significantly lower in-hospital mortality (8.0% vs 16.4%, p=0.037), with asthma independently associated with reduced mortality risk. This protection appears mediated by type 2 inflammation mechanisms – including reduced ACE2 expression (40-60% downregulation in eosinophilic phenotypes) and enhanced antiviral activity from elevated eosinophil peroxidase levels. However, post-COVID sequelae pose distinct challenges, with 33.9% of infected asthmatics experiencing chronic worsening of control requiring therapy escalation, compared to 11.4% in non-COVID counterparts (p<0.001). While maintenance inhaled corticosteroids (≥500 μg/day fluticasone-equivalent) accelerate viral clearance by 41%, pre-existing high-dose ICS use and atrial fibrillation independently increase severe outcome risks in hospitalized patients. These findings underscore asthma's dual role – its immunological profile mitigates acute COVID-19 severity, but suboptimal control and post-infection complications necessitate vigilant long-term management.

Disclosure

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Conceptualisation and Methodology: MJ,
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Check: MJ, MP, WK, KW
Formal analysis: MP, MJ, KK, AM
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In preparing this work, the author(s) utilized Perplexity AI for the purpose of enhancing the clarity and readability of the text. After using this tool, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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