Inflammatory and pulmonary function characteristics of bronchial asthma induced by COVID-19 infection

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Abstract. Bronchial asthma, a widely prevalent respiratory disease influencing individuals of all age groups worldwide, has been increasingly recognized as a global concern. While there exists a potentially heightened risk of severe coronavirus disease 2019 (COVID-19) in asthmatic patients, particularly those with non-allergic asthma, it is uncertain whether COVID-19 infection-induced bronchial asthma has its own unique clinical characteristics. The present study aimed to compare and analyze the pulmonary function and eosinophilic inflammation indices of patients with COVID-19 infection-induced bronchial asthma and those with typical bronchial asthma, and further deepen the understanding of COVID-19 infection-induced bronchial asthma. A retrospective analysis was conducted on the pulmonary function and inflammatory characteristics of 116 patients diagnosed with COVID-19 infection-induced bronchial asthma and treated in outpatient clinics after March 2023, as well as 113 patients with typical bronchial asthma diagnosed and treated from January 2022 to November 2022. The main clinical characteristics were cough, sputum, chest tightness, dyspnea and wheezing. There was no significant difference in clinical characteristics between the two groups. The results indicated that there was no significant difference in the total IgE, the absolute value and percentage of eosinophil, transoral FeNO, and trans-nasal FeNO in the peripheral blood samples of patients in the COVID-19 infection-induced bronchial asthma group compared with the typical bronchial asthma group. Although there was no significant difference between the two groups in the rates of impairment in ventilation function, reserve function, and small airway function, significant differences were identified in various indicators, including forced expiratory volume in 1 sec as a percentage of the predicted value (FEV1%), residual volume/total lung capacity (RV/TLC), peak expiratory flow (PEF), maximal expiratory flow rate at 75% (MEF75), maximal voluntary ventilation (MVV), FEV * 30, and residual volume (RV) between the two groups. The findings indicated that patients with COVID-19 infection-induced bronchial asthma exhibited a comparatively inferior pulmonary function versus those with typical bronchial asthma. However, it is important to note that the clinical impact of this disparity was not statistically significant.

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Introduction

Bronchial asthma, a long-standing ailment, is a prevalent respiratory disease that affects individuals of all ages globally (1). Patients with chronic persistent bronchial asthma frequently experience recurrent episodes, significantly disrupting their daily life and work (2). It is estimated that bronchial asthma affects ~300 million people worldwide, a number projected to rise to 400 million by 2025 (3). Bronchial asthma is responsible for ~500,000 hospitalizations annually, with ~250,000 deaths attributed to the disease each year (4). Bronchial asthma is distinguished by varying degrees of persistent inflammation and structural modifications in the airway (5). The most notable abnormalities include epithelial denudation, goblet cell metaplasia, subepithelial thickening, augmented airway smooth muscle mass, bronchial gland enlargement, angiogenesis, and modifications in extracellular matrix components, involving both large and small airways (6).

Alpha coronaviruses, including human coronavirus 229E (HCoV-229E) and HCoV-NL63, induce inflammation in the upper respiratory tract and have the potential to exacerbate asthma (7). Conversely, bate-coronaviruses, such as severe acute respiratory syndrome-CoV-2 (SARS-CoV-2), SARS-CoV, and Middle East respiratory syndrome-CoV (MERS-CoV), specifically target epithelial cells in both the upper and lower airways. SARS-CoV-2 serves as the causative agent for the disease known as coronavirus disease 2019 (COVID-19) (8). The COVID-19 pandemic originated in Wuhan, China, and rapidly spread worldwide. The COVID-19 pandemic, caused by the SARS-CoV-2, has emerged as a remarkable global health concern (9). COVID-19 manifests

in distinct stages following a median incubation period of 5 days, involving mild cases (80-90% of patients) characterized by upper and lower airway responses, while severe cases (10-20%) progress to bilateral pneumonia (10). A subset of patients with severe COVID-19 may develop acute respiratory distress syndrome, necessitating mechanical ventilation in an intensive care setting (11). With an estimated 300 million people globally affected by asthma (12), understanding the clinical characteristics of COVID-19-induced bronchial asthma is vital. Notably, patients with pre-existing comorbidities, such as diabetes and chronic obstructive pulmonary disease, are more prone to experience severe manifestations of COVID-19 (13). While there exists a potential heightened risk of severe COVID-19 in asthmatic patients, it is uncertain whether COVID-19 infection-induced bronchial asthma has its own unique clinical characteristics. The present study examined the medical history and clinical features of patients with COVID-19 infection-induced bronchial asthma, as well as those with typical bronchial asthma who were diagnosed and treated in our hospital. A comparative analysis was conducted to identify differences between these two groups of asthmatic patients.

Materials and methods

Patients. The clinical data of 116 patients with COVID-19 infection-induced bronchial asthma, who were diagnosed and treated in the outpatient department of Beijing Tsinghua Changgung Hospital (Beijing, China) after the COVID-19 pandemic (post-March 2023), were retrospectively analyzed. Additionally, data from 113 patients with typical bronchial asthma (no history of COVID-19 infection), who were diagnosed and treated before the pandemic (January 2022 to November 2022), were included for comparable analysis.

Inclusion and exclusion criteria. Inclusion criteria were as follows: patients who meet the GINA 2023 diagnostic criteria (https://ginasthma.org/2023-gina-main-report/) for bronchial asthma and were initially diagnosed with bronchial asthma; patients who aged between 18 and 75 years old; patients with no obvious abnormalities on chest X-ray or computed tomography (CT) examination.

Exclusion criteria were as follows: Patients with other respiratory diseases, such as lung infection and bronchiectasis; patients with severe diseases of other systems, including autoimmune diseases, myocardial infarction, heart failure, and malignant tumors; pregnant or lactating women.

Methods. The present study collected the medical history and clinical characteristics of patients with bronchial asthma induced by COVID-19 infection and patients with typical bronchial asthma who were diagnosed and treated in our hospital. The time from the onset of clinical manifestations of asthma to its diagnosis was recorded. Additionally, routine blood test results, serum total IgE levels, specific IgE levels, lung function, airway provocation test results, fractional exhaled nitric oxide (FeNO) levels, and other relevant information were collected. The differences between the two groups of asthmatic patients were then compared and analyzed. While the study concentrated on comparing clinical characteristics and pulmonary function, the treatment methods for both outpatient care and acute asthma control were standardized according to the *Global Initiative for Asthma (GINA) 2023* guidelines. All patients received treatment based on these guidelines, ensuring consistency in managing asthma symptoms. However, individualized adjustments in therapy might occur, especially in outpatient settings, which could introduce variability in the results. Future studies should provide more detailed descriptions of the specific treatment regimens used to assess their potential influence on clinical outcomes and pulmonary function, thereby improving the consistency and reproducibility of findings.

Pulmonary function tests. Once the acute phase of asthma attack was controlled, pulmonary function tests were completed in both groups, including pulmonary ventilation, pulmonary reserve function, small airway function, diffusion function, carbon monoxide dispersion to alveolar volume ratio, and residual volume/total lung capacity (RV/TLC). Pulmonary ventilation tests included forced expiratory volume in 1 sec as a percentage of the predicted value (FEV1%), forced expiratory volume in 1 sec to forced vital capacity ratio (FEV1/FVC), and peak expiratory flow (PEF). Maximal voluntary ventilation (MVV) was assessed to evaluate pulmonary reserve function. Small airway function tests included the maximal expiratory flow rates at 75, 50 and 25% of lung capacity (MEF75, MEF50 and MEF25) and the maximal mid-expiratory flow rate (MMEF75/25). Diffusion function tests included the single-breath diffusion capacity of carbon monoxide (DLCO SB) and DLCO normalized for alveolar volume (DLCO/VA).

Statistical analysis. SPSS 20.0 (IBM Corp.) and GraphPad 9.0 (GraphPad Software Inc.; Dotmatics) software were used to statistically analyze the data. The measurement data of normal distribution were expressed as the mean ± standard deviation ($X \pm s$), and analyzed using independent sample t-test. The measurement data of skewed distribution were expressed as the median (quartile), and analyzed via Mann-Whitney U test. The rates were compared using the χ^2 test. The Pearson correlation analysis was conducted to identify the factors associated with poor pulmonary function in the COVID-19 group. $P<0.05$ was considered to indicate a statistically significant difference.

Results

Patients' clinical characteristics. The range of age in the COVID-19 infection-induced bronchial asthma group was 21.9-74.7 years old, with an average age of 46.9±14.0 years. Among them, there were 69 women and 44 men, with a female-to-male ratio of 1.57. In the general bronchial asthma group, 7 (6.0%) cases had a history of allergy, including inhalation, food, drugs, 51 (44.0%) cases had allergic rhinitis, 12 (10.3%) cases had eczema, and 7 (6.0%) cases had chronic Hives. The range of age in the general bronchial asthma group was 21-77 years old, with an average age of 44.5±14.7 years old. Among them, there were 64 women and 30 men, with a female-to-male ratio of 2.13. In the COVID-19 infection-induced bronchial asthma group, there were 7 (7.4%) cases with allergic history, 37 (39.4%) cases with allergic

Table I. General data of the two groups.

	COVID-19 asthma (116)	Asthma (113)	P-value
Age, years	46.9±1.8	42.3±1.6	>0.05
Sex, female (%)	75 (64.7)	69 (61.1)	>0.05
History of allergy (%)	7 (6)	4 (3.5)	>0.05
Allergic Rhinitis (%)	51 (44)	52 (46)	>0.05
Nasal polyps (%)	0 (0)	0 (0.0)	>0.05
Eczema (%)	12 (10.3)	17 (15)	>0.05
Urticaria (%)	7 (6)	4 (3.5)	>0.05

Table II. Clinical symptoms of the two groups.

	COVID-19 asthma (116)	Asthma (113)	P-value
Duration, months	3.371±0.11	3.327±0.17	>0.05
Cough (%)	85 (73.3)	80 (70.8)	>0.05
Sputum (%)	13 (11.2)	12 (10.6)	>0.05
Chest tightness (%)	35 (27.6)	43 (38.1)	>0.05
Dyspnea (%)	35 (30.2)	28 (24.8)	>0.05
Wheezing (%)	3 (2.6)	5 (4.4)	>0.05

rhinitis, 9 (9.6%) cases with Nasal polyp, 10 (10.6%) cases with eczema, and 3 (3.2%) cases with chronic Hives (Table I).

The duration of disease was 3.371±0.11 months in the COVID-19 infection-induced bronchial asthma group and 3.327±0.17 months in the general bronchial asthma group, and there was no significant difference between the two groups (P>0.05). Patients' main clinical characteristics were cough, sputum, chest tightness, dyspnea and wheezing. There was no significant difference in patients' main clinical characteristics between the two groups (P>0.05) (Table II).

Comparison of inflammatory reaction between the two groups. Patients with COVID-19 infection-induced bronchial asthma and patients with common bronchial asthma all received routine peripheral blood tests. The results indicated no significant differences in total IgE levels, the absolute value and percentage of eosinophils (EOS), transoral FeNO, or transnasal FeNO in the peripheral blood between the COVID-19 infection-induced bronchial asthma group and the normal bronchial asthma group (P>0.05) (Table III, Fig. 1).

Comparison of pulmonary function between the two groups. Pulmonary ventilation, pulmonary reserve function, small airway function and diffusion function were evaluated between the COVID-19 infection-induced bronchial asthma and typical bronchial asthma groups. The pulmonary ventilation test indices, such as FEV1%, FEV1*30, and PEF were significantly lower in the COVID-19 infection-induced bronchial asthma group compared with the typical bronchial asthma group (P<0.05). The MVV index was also lower in the COVID-19 infection-induced bronchial asthma group

Table III. Comparison of pulmonary function indexes between COVID-19 asthma and asthma groups.

Indicator	Asthma (113)	COVID-19 asthma (116)	P-value
Vcmax	96.7±1.40	100.1±1.17	0.068
FVC	98.1±1.40	101.9±1.23	0.071
FEV1%	93.7±1.37	97.6±1.10	0.026
FEV1/FVC	81.4±0.67	80.36±0.77	0.293
PEF	99.0±1.58	92.1±1.67	0.003
MEF75	97.4±1.98	89.7±2.14	0.009
MEF50	81.5±2.13	76.4±2.48	0.121
MEF25	71.0±5.54	62.8±2.80	0.180
MMEF75/25	75.2±2.07	71.1±2.45	0.196
MVV	83.1±1.48	77.8±1.65	0.018
FEV1*30	78.9±0.93	74.8±1.33	0.012
DLCO SB	85.4±1.39	87.3±1.59	0.372
DLCO/VA	91.5±1.65	92.9±1.85	0.571
VA	97.4±1.53	96.5±1.71	0.691
R tot	130.9±5.23	132.6±4.06	0.794
RV	105.4±2.57	117.2±4.42	0.026
TLC	96.4±1.12	96.9±2.02	0.842
RV/TLC	38.5±0.83	42.5±1.45	0.008

FEV1%, forced expiratory volume in 1 sec as a percentage of the predicted value; MEF, maximal expiratory flow rate; RV, residual volume; MVV, maximal voluntary ventilation; TLC, total lung capacity; PEF, peak expiratory flow; DLCO SB, single-breath diffusion capacity of carbon monoxide.

(P<0.05). Among the small airway function test indices, MEF75 was reduced in the COVID-19 infection-induced bronchial asthma group (P<0.05). Additionally, RV and RV/TLC values were lower in the COVID-19 infection-induced bronchial asthma group (P<0.05, Table III, Fig. 2). However, no significant differences were identified in the overall rates of impairment in ventilation function, reserve function, small airway function, diffusion function, airway resistance, or the ratio of residual volume to total lung capacity (Table IV). These findings suggested that patients with COVID-19 infection-induced bronchial asthma exhibited comparatively reduced pulmonary function compared with those with bronchial asthma. However, this difference was not considered clinically significant.

The factors related to poor pulmonary function in the COVID-19 group. The Pearson correlation analysis was performed to detect the factors related to poor pulmonary function in the COVID-19 group. Patients were divided into two groups according to the median age of 45 years old. Pearson correlation analysis indicated that age was negatively correlated with MVV and FEV1*30 in the COVID-19 group (P<0.05), suggesting that the decline in pulmonary function was associated with advancing age. Moreover, sex was correlated with pulmonary function indices, including FEV1%, MEF75, MVV, FEV1*30, and RV/TLC in the COVID-19

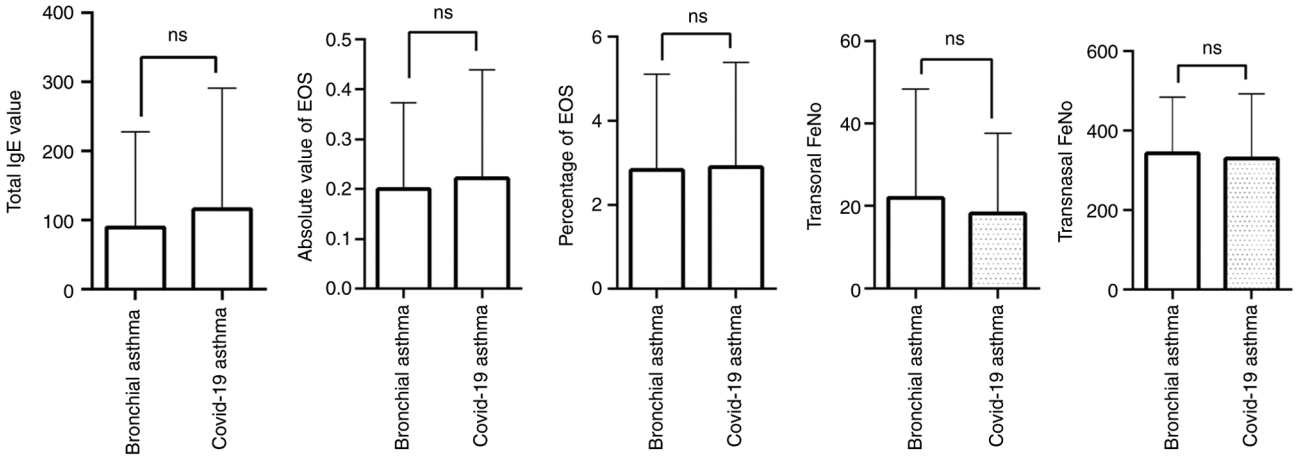

Figure 1. Comparison of inflammatory reactions between patients with COVID-19 infection-induced bronchial asthma and patients with typical bronchial asthma. There was no significant difference in the total IgE (IU/ml), the absolute value ($x10^9$) and EOS (%), transoral FeNO (ppd), and transnasal FeNO (ppd) in the peripheral blood samples of patients with COVID-19 infection-induced bronchial asthma compared with patients with typical bronchial asthma. EOS, percentage of eosinophil; ns, not significant.

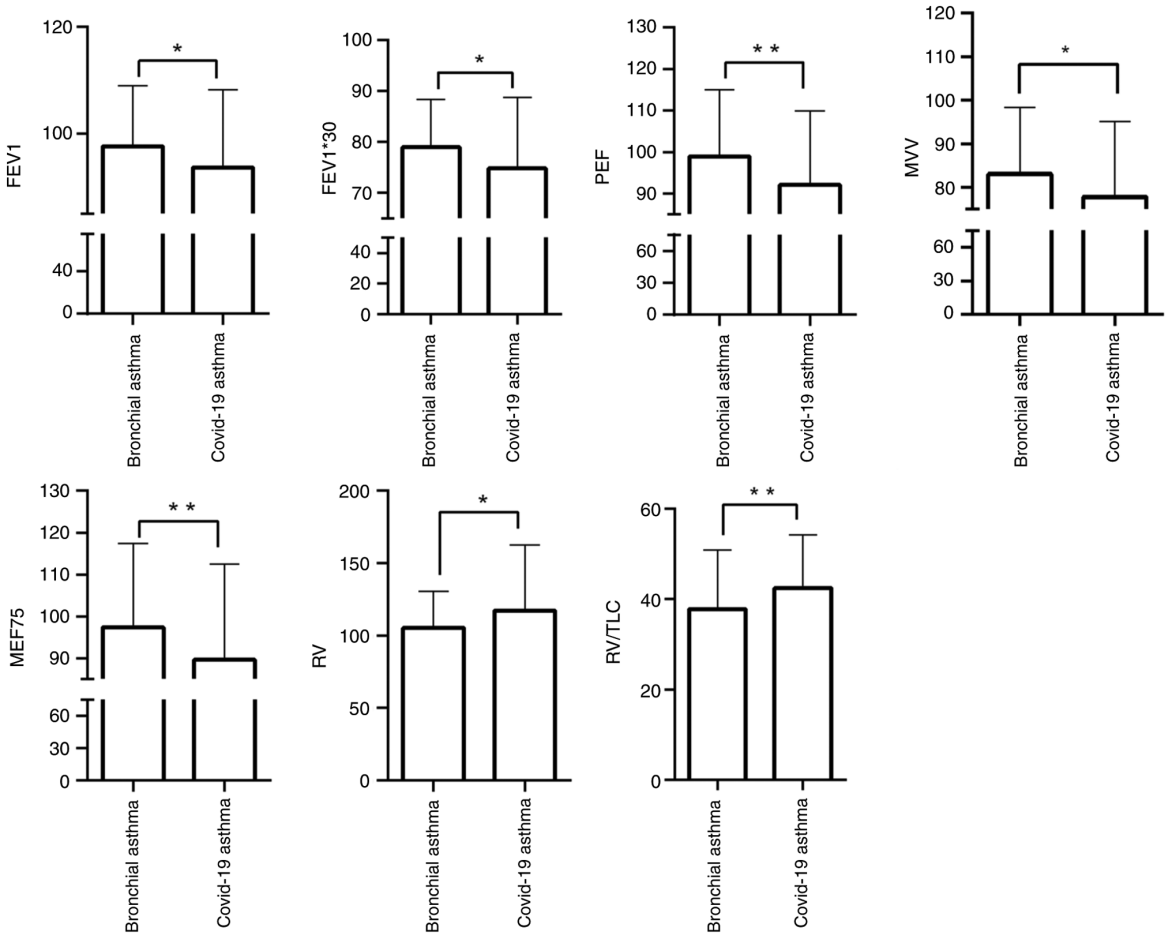

Figure 2. Comparison of pulmonary function between patients with COVID-19 infection-induced bronchial asthma and patients with typical bronchial asthma. FEV1 (FEV1/pred), FEV1*30 (l/min), PEF (PEF/pred), MEF75 (MEF75/pred) and MVV (MVV/pred) were higher in the COVID-19 infection-induced bronchial asthma group than the typical bronchial asthma group, while RV (RV-pred) and RV/TLC (%) were lower in the COVID-19 infection-induced bronchial asthma group than the typical bronchial asthma group. *P<0.05 and **P<0.01. FEV1%, forced expiratory volume in 1 sec as a percentage of the predicted value; MEF, maximal expiratory flow rate; RV, residual volume; MVV, maximal voluntary ventilation; TLC, total lung capacity; PEF, peak expiratory flow.

group (P<0.05), indicating that male patients' pulmonary function is comparatively inferior to that of female patients. However, the other indices, such as allergic rhinitis, cough, expectoration, chest, dyspnea and stridor, exhibited no correlation with pulmonary function in the COVID-19 group (P>0.05) (Table V).

Table IV. Comparison of pulmonary function between COVID-19 asthma and asthma groups.

Indicator	Asthma (113) (%)	COVID-19 asthma (116) (%)	P-value
Impairment of ventilatory function	27 (23.9)	34 (29.3)	0.41
Impairment of reserve function			0.37
Mildly impaired	36 (31.9)	36 (31.0)	
Moderately impaired	8 (7.1)	17 (14.7)	
Impairment of small airway function			0.50
Mildly impaired	37 (32.7)	27(23.3)	
Moderately/severely impaired	8 (7.1)	25(21.6)	
Impairment of diffusion function	42 (37.2)	37 (31.9)	0.49
Increased airway resistance	42 (37.2)	48(41.4)	0.57
Increased ratio of residual volume to total lung capacity	51 (45.1)	54 (46.6)	0.83

Table V. Factors related to poor lug functions in COVID-19 group.

Index	FEV1%	PEF	MEF75	MVV	FEV1*30	RV	RV/TLC	
Age (median age, 45 years)								
				-0.255	-0.301			Correlation coefficient
	0.827	0.670	0.710	0.007[a]	0.001[a]	0.944	<0.001[a]	P-value
Sex (Male 41, Female 75)								
	-0.241		-0.263	-0.197			-0.265	Correlation coefficient
	0.010[a]	0.300	0.005[a]	0.040[a]	0.697	0.422	0.006[a]	P-value
Allergic Rhinitis	0.892	0.272	0.356	0.889	0.656	0.402	0.917	P-value
Cough	0.904	0.172	0.345	0.972	0.810	0.278	0.501	P-value
Sputum	0.934	0.266	0.305	0.509	0.470	0.339	0.822	P-value
Chest tightness	0.986	0.986	0.771	0.462	0.756	0.188	0.507	P-value
Dyspnea	0.200	0.397	0.238	0.411	0.981	0.903	0.178	P-value
Wheezing	0.660	0.280	0.302	0.676	0.857	0.624	0.459	P-value

[a]P<0.05. FEV1%, forced expiratory volume in 1 sec as a percentage of the predicted value; PEF, peak expiratory flow; MEF, maximal expiratory flow rate; RV, residual volume; MVV, maximal voluntary ventilation; TLC, total lung capacity.

Discussion

The present study retrospectively analyzed 116 cases of COVID-19 infection-induced bronchial asthma and 113 cases of typical bronchial asthma. There was no significant difference between the two groups in terms of age, sex ratio, history of allergy, history of allergic rhinitis, history of nasal polyp, history of eczema, and history of chronic Hives. There was no significant difference in IgE, EOS, EOS percentage, oral FeNO, and nasal FeNO between the two groups. The pulmonary function of patients with COVID-19 infection-induced bronchial asthma appeared worse than that of patients with typical bronchial asthma.

The pathophysiological changes in bronchial asthma are mainly driven by airway smooth muscle spasms resulting from inflammation of the airway walls. This leads to recurrent symptoms, such as wheezing, shortness of breath, chest tightness and coughing (14,15). In late 2019, COVID-19 outbreak, caused by SARS-CoV-2, rapidly evolved into a global pandemic, resulting in millions of fatalities (16). Patients diagnosed with bronchial asthma are subjected to additional stressors due to the potential development of COVID-19 and the impact of the COVID-19 pandemic on societal and health-related services. Although clinical trials for safe and efficacious antiviral agents are ongoing and vaccine development programs have been accelerated, the long-term effects of SARS-CoV-2 infection are becoming increasingly recognized (17). The COVID-19 pandemic has imposed remarkable challenges to the routine management and diagnosis of bronchial asthma, including reduced face-to-face consultations, limitations in conducting spirometry, and

restrictions on traditional pulmonary rehabilitation and home care programs (18).

Eosinophil inflammation is a significant factor in the pathogenesis of asthma. The parameters used to assess eosinophilic inflammation include the absolute count and percentage of EOS, total IgE level in the bloodstream, transoral FeNO, and trans-nasal FeNO. These parameters are essential diagnostic indicators for asthma and are also valuable for evaluating the degree of inflammation control in asthmatic patients (19-21). The present study found no significant differences in the absolute count and percentage of EOS, IgE level, transoral FeNO, and trans-nasal FeNO between the two groups, suggesting comparable levels of eosinophilic inflammation. In allergic asthma, eosinophilic inflammation is generally positively correlated with the degree of pulmonary dysfunction (22,23), demonstrating that higher levels of eosinophilic inflammation are mainly associated with more severe pulmonary function deficits.

Pulmonary function is a fundamental measure for evaluating asthma (24,25). The small airway, typically defined as having a diameter of ≤2 mm, is a critical site for the early development of several pulmonary diseases (26). In asthma, inflammation of the small airway wall can lead to significant airway smooth muscle contraction, resulting in recurrent symptoms, such as wheezing, dyspnea, chest tightness and cough (27-29). Despite the substantial cumulative cross-sectional area of small airways, their resistance accounts for <20% of total airway resistance (30). Traditional pulmonary function tests mainly fail to detect abnormalities in small airways, whereas specialized small airway function tests are more sensitive in identifying early pathological changes (31). The emergence of COVID-19 has presented unique challenges for patients with bronchial asthma, and studies suggested potential interactions between the virus and asthma pathophysiology, including small airway function and systemic inflammation (16-18). However, direct comparisons between COVID-19-induced bronchial asthma and typical asthma remain scarce, particularly concerning pulmonary dysfunction. Previous studies have highlighted the importance of assessing small airway function as an early marker for pulmonary diseases (26,31). Previous research (32) has found no significant impact of COVID-19 on asthma in children, while studies on adult populations suggest variable outcomes, including exacerbations and prolonged symptoms (33,34). These findings underscore the need for further exploration of pulmonary function in adult patients with COVID-19-induced bronchial asthma. Eosinophilic inflammation, a hallmark of asthma pathogenesis, has been extensively studied, and parameters, such as total IgE, FeNO and eosinophil count served as diagnostic and prognostic markers (19-21). Although the findings indicated no significant differences in these markers between the two groups, scholars (22,23) suggested that variations in eosinophilic inflammation may influence asthma severity, warranting further investigation in the context of COVID-19. The COVID-19 pandemic has disrupted traditional asthma management practices, including diagnostic procedures and treatment adherence, potentially altering disease trajectories (17,18).

In a study by Tosca *et al* (32), COVID-19 was found to have no significant impact on pulmonary function or asthma control in children. By contrast, isolated reports in adults have described asthma exacerbations associated with COVID-19, although such events are not prevalent in the general population (33,34). The current analysis suggested a potential link between COVID-19 and asthma, particularly concerning pulmonary function (35). The present study identified significant differences in key pulmonary function parameters, comprising FEV1, RV/TLC, PEF, MEF75, MVV, FEV1*30 and RV, between the two groups. These results indicated that patients with COVID-19 infection-induced bronchial asthma exhibited reduced pulmonary function compared with those with typical bronchial asthma. While no statistically significant differences were identified in the overall rates of ventilation function impairment, reserve function impairment, or small airway function impairment, the findings suggest that patients with COVID-19-induced bronchial asthma may have subclinical lesions contributing to distinct clinical features compared with those with non-COVID-19-induced bronchial asthma. It is noteworthy that the present study included only non-critically ill patients receiving outpatient treatment. As a result, variations in the completion of diagnostic tests among patients led to inconsistencies in the statistical analysis of certain indicators. The Pearson correlation analysis revealed that both age and sex were significantly associated with poorer pulmonary function in the COVID-19 infection-induced bronchial asthma group. However, these findings require further investigation to determine how these factors interact with COVID-19 infection and whether they have a more notable impact on pulmonary function over time. Although the present study identified differences in specific pulmonary function tests, such as FEV1%, MEF75, and MVV, between the two groups, the lack of statistical significance across all parameters suggests that the clinical implications of COVID-19's impact on asthma may be more remarkable. These differences may not indicate a substantial deviation in overall pulmonary function, particularly in non-critically ill patients. Further exploration of the pathophysiological mechanisms contributing to these differences in pulmonary function is essential. Specifically, the role of COVID-19 in exacerbating asthma symptoms may involve complex inflammatory processes that affect airway smooth muscle function, while the exact mechanisms remain to be fully elucidated.

Although the present study found poorer pulmonary function in patients with COVID-19 infection-induced bronchial asthma, the potential mechanisms underlying these differences were not thoroughly explored. Future studies should investigate how COVID-19-induced alterations in airway function, such as changes in airway smooth muscle tone or inflammation beyond eosinophil-mediated pathways, may contribute to these findings. Although the observed differences in pulmonary function were not statistically significant in terms of impairment rates across functions, the poorer pulmonary function in patients with COVID-19 infection-induced bronchial asthma highlights the potential need for adjusted management strategies. These may include more frequent monitoring, early intervention, and personalized treatment plans to better address the unique needs of this patient population.

The findings suggested that while the pulmonary function in patients with COVID-19 infection-induced bronchial asthma was slightly poorer than that of patients with typical asthma, these differences might not necessitate entirely

distinct treatment protocols, while indicated the need for enhanced monitoring and individualized management strategies, particularly for older and male patients who exhibited poorer pulmonary function. Given the observed reduction in key pulmonary function indices, such as FEV1% and MEF75, in COVID-19-induced asthma, additional interventions concentrating on maintaining airway patency and preventing further pulmonary decline might be especially beneficial for this subgroup of patients. Management strategies for COVID-19-induced asthma can benefit from incorporating early detection and treatment of airway inflammation, as well as an elevated concentration on small airway function given the potential subclinical changes observed in these patients. Future studies should investigate whether the pathophysiological mechanisms unique to COVID-19-induced asthma, such as possible non-eosinophilic inflammatory pathways, require specific therapeutic modifications beyond standard asthma treatments, particularly in terms of long-term control medications and pulmonary rehabilitation. While current treatment guidelines, such as GINA, provide a robust framework for asthma management, the subtle pulmonary function differences in COVID-19-induced asthma suggest that treatment intensification or modifications may be warranted to address specific functional impairments.

The present study has several limitations. Firstly, the differing inclusion periods for the two groups, one group diagnosed and treated prior to the COVID-19 pandemic and the other afterward, might introduce variability stemming from temporal variations in environmental factors, clinical practices, or healthcare accessibility. Secondly, the retrospective nature and single-center setting of the study could limit the generalizability of the findings and result in incomplete or inconsistent data for certain variables. Thirdly, although the present study analyzed routine inflammatory markers, such as total IgE, eosinophil count, eosinophil percentage, and FeNO level, the retrospective design and concentration on clinical outpatient data limited the inclusion of more advanced biomarkers. Consequently, these parameters might not fully capture the complexity of the inflammatory and immunological responses in patients with COVID-19 infection-induced bronchial asthma. Future investigations will benefit from the inclusion of advanced inflammatory and immunological markers, such as cytokines [for examle, interleukin-6 (IL-6), IL-13, and tumor necrosis factor-α (TNF-α)] and chemokines, to provide a more comprehensive evaluation of the inflammatory profile in this patient population. Lastly, while the sample size was adequate for preliminary analysis, it might not provide sufficient statistical power to detect more significant differences between the two groups. Future prospective, multicenter studies are essential with standardized methodologies to confirm and expand the findings. To enhance the understanding of these findings' clinical significance, future research should concentrate on conducting prospective studies with larger, multi-center cohorts. Such studies should investigate the long-term effects of COVID-19 on asthmatic patients, emphasizing both the underlying pathophysiological mechanisms and their implications for clinical practice. Particular attention should be given to develop personalized therapeutic strategies for patients with COVID-19-induced bronchial asthma.

In conclusion, although no significant difference was found between the two groups in the rates of impairment in ventilation function, reserve function, and small airway function, significant differences were identified in various indicators, including FEV1, RV/TLC, PEF, MEF75, MVV, FEV*30 and RV between the two groups. The findings indicated that pulmonary function of patients with COVID-19 infection-induced bronchial asthma was worse than that of patients with typical asthma.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

MQZ, JQZ and XDM contributed substantially to the conception and design of the study and acquisition of data, as well as analysis and interpretation of the data. MQZ, LNZ and PZ were involved in interpretation of the data, and in drafting the manuscript. MQZ, JQZ and XDM have participated sufficiently in the study to take public responsibility for appropriate portions of the content and agree to be accountable for all aspects of the study in ensuring that questions related to the accuracy or integrity of any part of the study are appropriately investigated and resolved. MQZ and JQZ confirm the authenticity of all the raw data. All authors have read and approved the final version of the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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