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Original Article

Efficacy and immune-inflammatory modulation of combined glucocorticoid and anti-tuberculosis therapy in patients with bronchial asthma and pulmonary tuberculosis

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Abstract: Objective: To evaluate the efficacy and immunomodulatory effects of glucocorticoid combination therapy with anti-tuberculosis treatment in patients with asthma complicated by pulmonary tuberculosis. Methods: Clinical data of 128 patients with bronchial asthma complicated with pulmonary tuberculosis were retrospectively analyzed. According to the treatment plan, the patients were divided into a control group and an observation group. The improvements in clinical symptoms, lung function indexes, immune and inflammation markers, chest X-ray results, sputum negative conversion rate, and incidence of adverse reactions were compared between the two groups. Results: Patients in the observation group demonstrated superior improvement in clinical symptoms compared to the control group. After six months of treatment, both groups demonstrated significant improvements in pulmonary function scores relative to baseline, with the observation group showing greater improvement than the control group. Inflammatory and immune markers were significantly reduced in both groups compared to pre-treatment levels, with the reduction being greater in the observation group than in the control group. After 6 months of treatment, lesion absorption and cavitary closure were more favorable in the observation group. At the 6-month follow-up, the bacteriological recurrence rate in the observation group was significantly lower than that of the control group. Conclusion: The combined use of glucocorticoids and anti-tuberculosis drugs provides dual advantages: it effectively controls asthma while enhancing the efficacy of anti-tuberculosis treatment.

Keywords: Glucocorticoids, anti-tuberculosis therapy, bronchial asthma, pulmonary tuberculosis, immune inflammatory levels

Introduction

Bronchial asthma and pulmonary tuberculosis are both common respiratory diseases. Bronchial asthma is a heterogeneous disease characterized by chronic airway inflammation and hyperresponsiveness, mainly manifested as recurrent wheezing, shortness of breath, chest tightness, and cough [1]. In contrast, tuberculosis is a chronic infectious disease caused by *Mycobacterium tuberculosis*, commonly presenting with symptoms such as cough, expectoration, low-grade fever, and night sweats [2]. With the complexity of changes in the global tuberculosis epidemic, the aging population, and the increasing number of immunocompromised individuals, the incidence of bronchial asthma combined with pulmonary tuberculosis is increasing year by year. Developing a reasonable, effective, and safe treatment plan has become a focus of clinical attention [3].

Anti-tuberculosis treatment is the basis of pulmonary tuberculosis management, but conventional regimens have limited efficacy in controlling asthma symptoms [4]. Glucocorticoids, as the core drug for asthma control, exert strong anti-inflammatory and immunomodulatory effects. However, their application in patients with active tuberculosis has long been controversial, mainly due to concerns about their potential to inhibit cellular immunity, which may lead to tuberculosis spread or recurrence

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[5]. Studies have shown that, when combined with anti-tuberculosis treatment, inhaled corticosteroids can better control asthma symptoms without significantly increasing the risk of tuberculosis recurrence, and may have a positive effect on tuberculosis outcome by regulating the immune-inflammatory responses [6]. However, clinical studies on this combined approach remain limited, especially randomized controlled trials on patients with bronchial asthma complicated by pulmonary tuberculosis or studies with in-depth analyses of immune-inflammatory markers.

Therefore, this study systematically evaluated the effects of glucocorticoid combined with anti-tuberculosis treatment on clinical symptoms, lung function, immune-inflammatory levels, imaging findings, and safety in patients with bronchial asthma complicated with pulmonary tuberculosis, aiming to provide evidence for clinical management.

Materials and methods

Study design

This retrospective study was approved by the Ethics Committee of Fuzhou Pulmonary Hospital of Fujian. Clinical records of 128 patients with concomitant bronchial asthma and pulmonary tuberculosis were reviewed. All patients were admitted to Fuzhou Pulmonary Hospital of Fujian from January 2022 to December 2024. Patients were assigned to either a control group (n=61) or an observation group (n=67) according to their treatment regimens.

Patients expressed their treatment preferences during clinical management. The control group comprised patients who, due to strong personal preference, firmly declined corticosteroid treatment regimens and proactively opted for non-hormonal alternatives. The observation group comprised patients who received the standard treatment regimen, involving inhaled corticosteroids as recommended by their physicians.

Inclusion criteria: (1) Patients meeting the diagnostic criteria for asthma outlined in the *Guidelines for the Prevention and Treatment of Bronchial Asthma (2024 Edition)* [7]: ① Recurrent wheezing, dyspnea, chest tightness, or cough, often associated with exposure to allergens, cold air, physical or chemical irritants, upper respiratory tract infections, or physical exertion; ② Wheezing (primarily during expiration) heard in both lungs during an attack, with a prolonged expiratory phase; ③ The above-mentioned symptoms and signs can be alleviated with treatment or resolve spontaneously; ④ Positive bronchodilator test (FEV_1 improvement rate ≥12%, and absolute increase in FEV_1 ≥200 mL) or positive bronchial provocation test; (2) Patients meeting the diagnostic criteria for pulmonary tuberculosis outlined in the World Health Organization's *Comprehensive guidelines for tuberculosis* [8]: ① Positive sputum smear or culture for *Mycobacterium tuberculosis*; ② Chest imaging demonstrating tuberculous lesions accompanied by systemic symptoms such as low-grade fever, night sweats, fatigue, and weight loss.

Exclusion criteria: (1) Concurrent pulmonary diseases other than tuberculosis; (2) Severe hepatic or renal insufficiency, immunodeficiency disorders; (3) Hypersensitivity to study drugs, including isoniazid, rifampicin, or budesonide; (4) Recent corticosteroid or anti-tuberculosis therapy within the preceding three months; (5) Pregnant or lactating women; (6) Patients with psychiatric disorders.

Based on the inter-group difference in sputum culture conversion rates after six months of treatment, efficacy analysis was conducted with α=0.05 (two-tailed). The total sample size N=128 yielded a calculated test power of 81.15% for this study.

Treatment

The control group received anti-tuberculosis therapy combined with formoterol dry powder inhaler plus montelukast sodium tablets. The anti-tuberculosis regimen comprised isoniazid tablets (Chongqing Huabang Pharmaceutical Co., Ltd., National Drug Approval No. H19993004) 0.3 g per dose, once daily, orally; Rifampicin capsules (Harbin Pharmaceutical Group Sanjing Northern Pharmaceutical Factory, National Drug Approval No. H23021322) 0.45 g per dose, once daily, administered orally on an empty stomach; Pyrazinamide tablets (Zhejiang Anbite Pharmaceutical Co., Ltd., National Drug Approval No. H20227053) 1.5 g per dose, once daily, administered orally; Ethambutol tablets (Dajiaweikang Biopharma-

ceutical Co., Ltd., National Drug Approval No. H20227142) 0.75 g per dose, once daily, orally. Treatment was initiated with a four-drug regimen for two months (intensive phase), subsequently maintained on a combination of isoniazid and rifampicin for an additional four months (consolidation phase) [9, 10]. Additionally, the control group received: Formoterol dry powder inhaler (Chengda Tianqing Pharmaceutical Group Co., Ltd., National Drug Approval No. H20103179), 4.5 μg per dose, once daily. Montelukast sodium tablets (Ogalon Pharmaceutical Technology Co., Ltd., National Drug Approval No. HJ20181187), 10 mg, once daily, 30 minutes before bedtime [11]. The treatment course lasted 6 months. Total dose of formoterol was calculated as: 4.5 μg per inhalation × 1 inhalation per day × 180 days = 810 μg (0.81 mg).

The observation group received anti-tuberculosis therapy combined with glucocorticoid treatment. The anti-tuberculosis regimen was identical to that in the control group, supplemented with budesonide/formoterol powder for inhalation (Chongqing Haimoni Pharmaceutical Co., Ltd., National Drug Approval No. HC20181007), at a dose of 160 μg/4.5 μg per puff, administered twice daily. Patients were instructed to rinse their mouths after inhalation to prevent oral candidiasis [12]. The treatment course lasted six months. Cumulative total dose of budesonide: 160 μg/inhalation × 2 inhalations/day × 180 days = 57,600 μg (57.6 mg). Cumulative total dose of formoterol: 4.5 μg/inhalation × 2 inhalations/day × 180 days = 1,620 μg (1.62 mg).

Observation indicators

General information: Baseline data were collected for all patients, including sex, age, duration of illness, smoking history, medical history, asthma severity, and types of tuberculosis.

Time to clinical symptom improvement: The daily changes in patient symptoms recorded by healthcare professionals in medical records were reviewed. Complete resolution of symptoms was used as the criterion. The following indicators were collected: (1) Time of wheezing resolution: The time at which auscultation revealed no wheezing in both lungs; (2) Time of dyspnea resolution: The time at which patients exhibited no significant dyspnea during routine activities (e.g., walking 500 meters); (3) Time to resolution of cough: The time at which cough frequency was ≤2 episodes per day, including both daytime and night-time episodes [13].

Pulmonary function indicators: Pulmonary function test results documented in the medical records prior to treatment and at the conclusion of the sixth month of treatment were reviewed. A desktop spirometer (Shenzhen Ruilian Medical Co., Ltd., model: RLC-2000) was used to measure Forced Vital Capacity (FVC), Forced Expiratory Volume in One Second (FEV_1), FEV_1/FVC, and Maximum Mid-Expiratory Flow (MMEF). Each parameter was assessed three times, with the optimal value recorded [14]. Simultaneously, asthma control and quality of life assessment results were reviewed. The Asthma Control Test (ACT) developed by Nathan et al. [15] was employed to evaluate asthma control. This questionnaire comprises five questions, with a total score ranging from 0 to 25. The Cronbach's alpha of this scale is 0.770 [16, 17].

The Asthma Quality of Life Questionnaire (AQLQ) developed by Juniper was employed [18], comprising four dimensions: activity limitations, symptoms, emotional functioning, and environmental triggers. This 32-item questionnaire assigns a score of 1 to 7 to each item, with higher scores corresponding to better quality of life. The Chinese version of this scale had a Cronbach's alpha coefficient of 0.930 [19, 20].

Inflammatory markers: Serum test results documented in the medical records prior to treatment and at the conclusion of the sixth month of treatment were reviewed. Fasting venous blood was collected from patients, centrifuged at 3000 r/min for 10 minutes to separate serum, and C-reactive protein (CRP) levels were measured using the fully automated biochemical analyzer (Siemens Healthineers, model: ADVIA Chemistry XPT). Interleukin-6 (IL-6) levels were measured using an automated immunoassay analyzer (Siemens Healthineers, model: Atellica IM) [21].

Immunological indicators: Serum immunological data test results documented in the medical records prior to treatment and at the conclusion of the sixth month of treatment were reviewed. Using the aforementioned serum

samples, levels of immunoglobulin E (IgE), IgG, and IgA were measured using a fully automated chemiluminescent immunoassay analyzer (Siemens Healthineers, model: Atellica-IM) [22].

Chest X-ray and sputum culture conversion status: Chest imaging findings documented in the medical records at the conclusion of the sixth month of treatment were reviewed. Chest radiographs in both anteroposterior and lateral views were acquired using a digital X-ray camera (Philips Healthcare, model: DigitalDiagnost C50). Two radiologists conducted double-blind readings to assess lesion absorption, which was classified as significant absorption (lesion absorption ≥50%), partial absorption (lesion absorption 25%-49%); no change (lesion absorption <25% or no absorption); and progression (lesion enlargement or new lesions). Cavity closure status was also assessed, and classified into complete closure (cavity disappearance), reduction (cavity diameter reduction ≥50%), no change (cavity diameter reduction <50% or no change), or enlargement (cavity diameter increases) [23].

Sputum bacteriological test results were reviewed, and sputum *Mycobacterium tuberculosis* was detected using acid-fast staining of smears. Three consecutive negative smears were defined as sputum bacteriological conversion. The sputum bacteriological conversion rate was calculated as: Conversion rate = Number of patients achieving sputum bacteriological conversion/Number of sputum-positive patients at enrolment × 100%. A six-month follow-up period commenced after treatment completion, during which sputum smears and cultures were reviewed every two months. A recurrence of positive results was defined as bacteriological relapse [24].

Incidence of adverse reactions: Adverse reactions recorded weekly in the medical records during the treatment period were reviewed, including diarrhea, pharyngeal discomfort, and abnormal liver function (alanine aminotransferase or aspartate aminotransferase increased more than 1.5 times the upper limit of normal). The incidence rate of adverse reactions was computed using the formula: (Number of cases with adverse reactions/Total number of cases) × 100%.

Statistical analysis

All data analyses were performed using Statistical Product and Service Solutions 27.0 (IBM, Armonk, NY, USA). The normality of measurement data was assessed using Kolmogorov-Smirnov test. For variables conforming to a normal distribution, data were presented as mean ± standard deviation (SD). Differences between the two groups were compared using the independent samples *t*-test, whereas intragroup comparisons (before and after treatment) were conducted with the paired *t*-test. Counted data were expressed as frequencies and percentages [*n* (%)] and analyzed using the chi-square test. Ordinal data were assessed using the Wilcoxon rank-sum test. Power analysis was performed using G*Power 3.1 software, where a power level exceeding 80% was considered indicative of clinically relevant differences. Graphs and tables were created using GraphPad Prism software. Statistical significance was defined as $P<0.05$.

Results

Comparison of general patient characteristics

No significant differences were observed between the two groups in terms of general characteristics, including sex, age, disease duration, smoking history, underlying medical conditions, asthma severity, or type of pulmonary tuberculosis, indicating comparability between the two groups (all $P>0.05$; **Table 1**).

Comparison of the time to clinical symptom improvement

Patients in the observation group experienced significantly shorter durations for the resolution of wheezing (6.82±2.85 days vs. 8.44±3.79 days), dyspnea (5.98±1.89 days vs. 6.82±2.24 days), and cough (7.04±2.59 days vs. 9.49±3.08 days) compared to the control group (all $P<0.05$) (**Table 2**).

Comparison of pulmonary function indicators before and after treatment

Before treatment, there were no significant differences between the two groups in FEV_1, FVC, FEV_1/FVC, MMEF, ACT score, or AQLQ score (all $P>0.05$). After 6 months of treatment,

Table 1. Comparison of patient demographics between the two groups

		Observation group (*n*=67)	Control group (*n*=61)	t/χ^2	*P*
Sex (*n*, %)	Male	39 (58.21)	33 (54.10)	0.219	0.640
	Female	28 (41.79)	28 (45.90)		
Age (yrs; mean ± SD)		53.12±11.02	50.61±11.25	1.274	0.205
Disease duration (months; mean ± SD)		7.46±3.22	8.69±3.83	1.973	0.051
Smoking history (*n*, %)		23 (34.33)	19 (31.15)	0.147	0.702
Basic medical history (*n*, %)		19 (28.36)	16 (26.23)	0.073	0.787
Severity of asthma (*n*, %)	Mild	27 (40.30)	28 (45.91)	0.517	0.605*
	Moderate	30 (44.78)	24 (39.34)		
	Severe	10 (14.92)	9 (14.75)		
Types of Tuberculosis (*n*, %)	Primary	8 (11.94)	7 (11.48)	0.504	0.777
	Secondary	48 (71.64)	41 (67.21)		
	Haematogenous dissemination	11 (16.42)	13 (21.31)		

*: Wilcoxon rank-sum test; SD: Standard Deviation.

Table 2. Comparison of time to resolution of clinical symptoms between the two groups (days)

	Observation group (*n*=67)	Control group (*n*=61)	*t*	*P*
Time to resolution of wheezing	6.82±2.85	8.44±3.79	2.748	0.007
Time to resolution of dyspnea	5.98±1.89	6.82±2.24	2.300	0.023
Time to resolution of cough	7.04±2.59	9.49±3.08	4.885	<0.001

both groups demonstrated significant improvement in the aforementioned pulmonary function tests and quality of life scores compared with pre-treatment levels. Notably, the observation group demonstrated more pronounced improvements in FEV_1, FVC, FEV_1/FVC ratio, MMEF, ACT score, and AQLQ score compared to the control group (all *P*<0.05) (**Figure 1**).

Comparison of inflammatory markers before and after treatment

Prior to treatment, no significant differences were observed between the two groups in CRP or IL-6 levels (*P*>0.05). After 6 months of treatment, inflammatory markers in both groups showed significant reductions compared to pre-treatment levels. Also, the reductions in CRP and IL-6 levels were greater in the observation group compared to the control group (*P*<0.05) (**Figure 2**).

Comparison of immunological markers before and after treatment

Before treatment, no significant differences were observed between the two groups in the levels of IgE, IgG, and IgA (*P*>0.05). Following 6 months of treatment, these markers showed significant reductions in both groups compared to baseline levels. Notably, the observation group demonstrated superior improvement in IgE levels, IgG levels, and IgA levels compared to the control group (*P*<0.05) (**Figure 3**).

Comparison of chest X-ray findings and sputum culture conversion rates

After six months of treatment, regarding lesion absorption, 61.19% of patients in the observation group showed significant absorption, 28.36% demonstrated partial absorption, 10.45% showed no change, and no cases showed progression. In the control group, 35.93% of patients exhibited significant absorption, 43.75% showed partial absorption, 17.19% demonstrated no change, and 2 cases showed progression. The observation group showed superior lesion absorption outcomes compared with the control group (*P*<0.05). Regarding cavity closure, among the 34 patients with cavities in the observation group, 55.89% achieved complete closure, 32.35% experienced reduction, 8.82% showed no change, and 1 case showed enlargement. In the control group, among 37 patients with cavi-

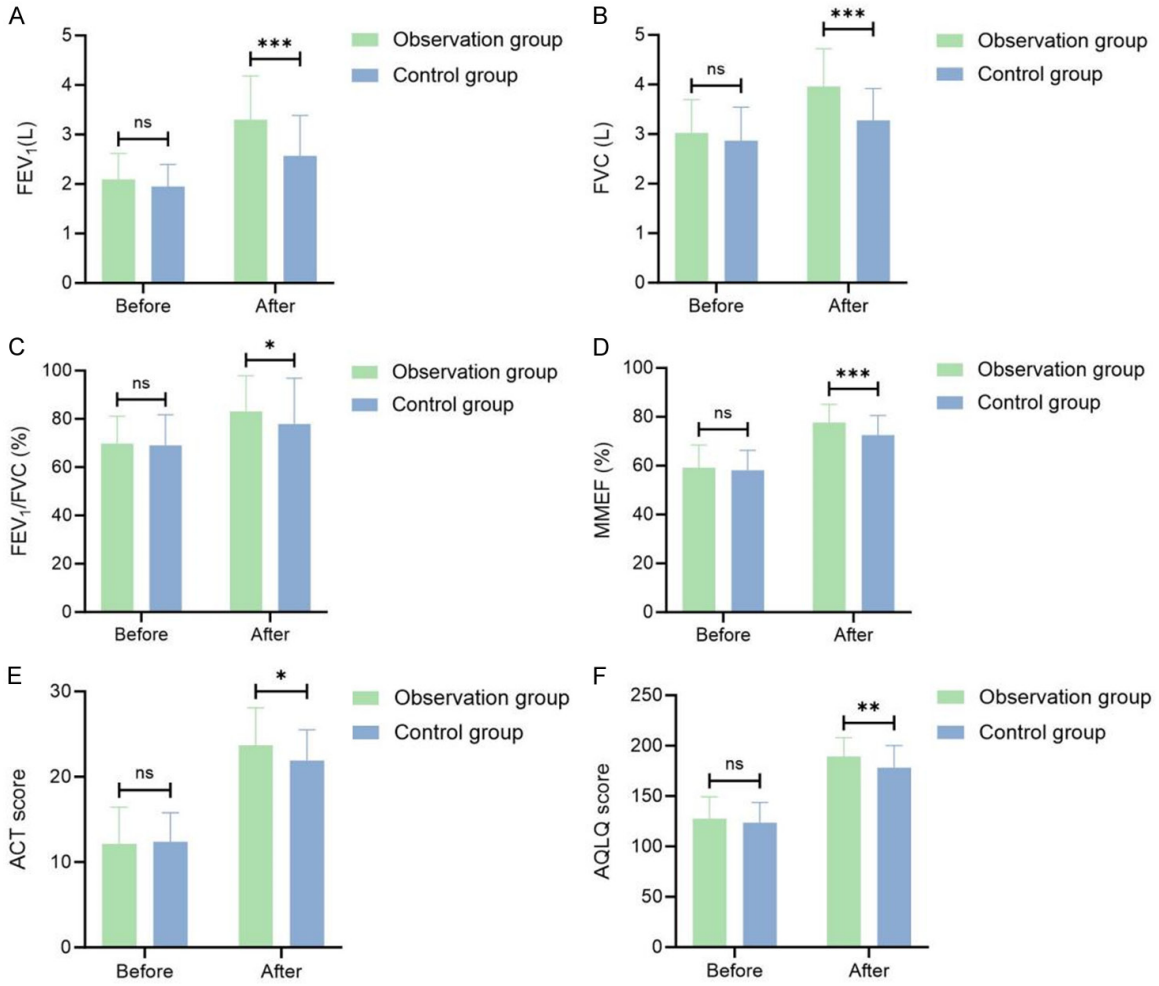

Figure 1. Comparison of pulmonary function indicators between the two groups before and after treatment. A. FEV_1; B. FVC; C. FEV_1/FVC; D. MMEF; E. ACT score; F. AQLQ score (ns: not significant; *P<0.05; **P<0.01; ***P<0.001). Notes: FVC, Forced Vital Capacity; FEV_1, Forced Expiratory Volume in One Second; MMEF, Maximum Mid-Expiratory Flow; ACT, Asthma Control Test; AQLQ, Asthma Quality of Life Questionnaire.

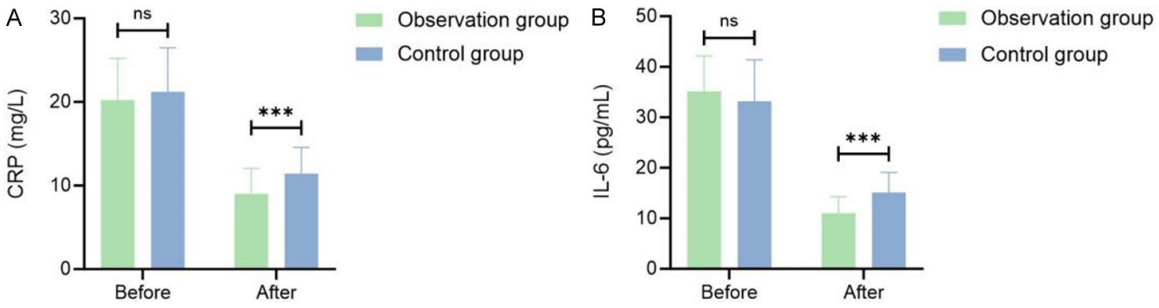

Figure 2. Comparison of inflammatory markers between the two groups before and after treatment. A. CRP; B. IL-6 (ns: not significant; ***P<0.001). Notes: CRP, C-reactive protein; IL-6, Interleukin-6.

ties, 24.32% achieved complete closure, 45.95% experienced cavity reduction, 21.62% showed no change, and 3 cases showed enlargement. Cavity closure outcomes were superior in the observation group compared to the control group (P<0.05) (**Table 3**; **Figures 4**, **5**).

At baseline, 59 patients in the observation group and 56 patients in the control group

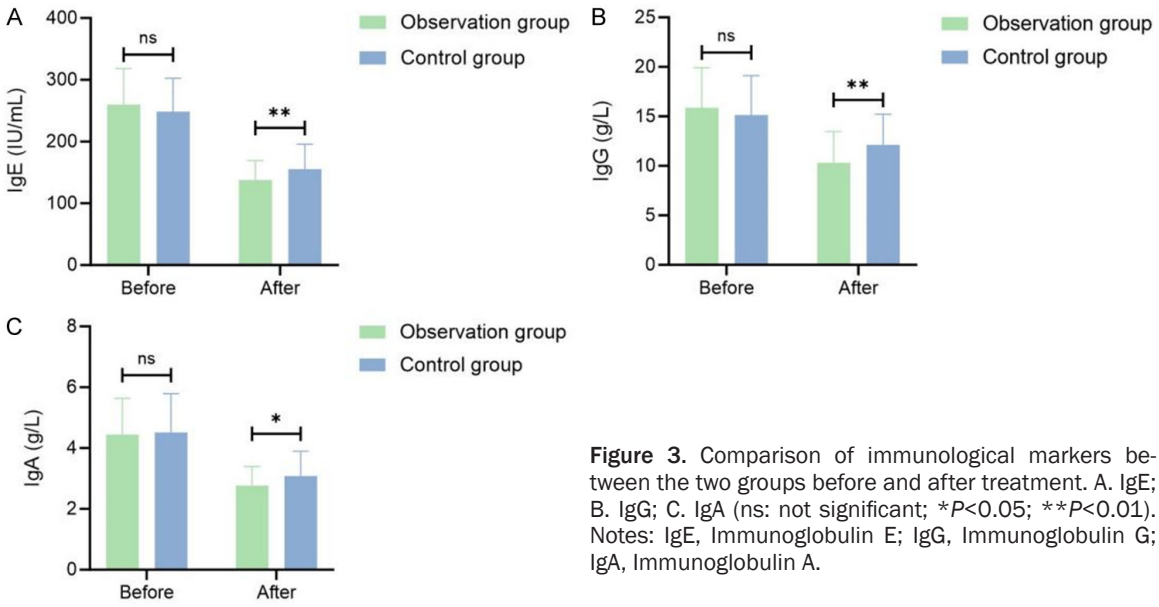

Figure 3. Comparison of immunological markers between the two groups before and after treatment. A. IgE; B. IgG; C. IgA (ns: not significant; *P<0.05; **P<0.01). Notes: IgE, Immunoglobulin E; IgG, Immunoglobulin G; IgA, Immunoglobulin A.

Table 3. Comparison of chest X-ray findings between the two groups

		Observation group (n=67)	Control group (n=61)	χ^2	P
Lesion absorption	Significant absorption	41 (61.19)	21 (34.43)	10.446	0.015
	Partial absorption	19 (28.36)	28 (45.90)		
	No change	7 (10.45)	10 (16.39)		
	Progression	0 (0.00)	2 (3.28)		
Cavity closure	Complete closure	19 (55.89)	9 (24.32)	8.107	0.046
	Reduction	11 (32.35)	17 (45.95)		
	No change	3 (8.82)	8 (21.62)		
	Enlargement	1 (2.94)	3 (8.11)		

Note: 34 patients in the observation group had cavities, and 37 patients in the control group had cavities.

showed positive sputum bacterial tests. Following treatment, the sputum culture conversion rate was significantly higher in the observation group at the 2-month (65.67%), 4-month (80.60%), and 6-month (88.06%) follow-ups compared to the controls (P<0.05). At the 6-month follow-up after treatment completion, the bacteriological recurrence rate in the observation group (4.48%) was significantly lower than that of the control group (16.39%) (P<0.05) (**Table 4**).

Comparison of adverse reaction occurrence

Over the treatment period, comparisons between the two groups revealed no significant differences in the incidence of specific adverse reactions (diarrhea, pharyngeal discomfort, abnormal liver function) or the total incidence rate (P>0.05) (**Table 5**).

Discussion

The difficulty in treating bronchial asthma complicated by pulmonary tuberculosis lies in the conflicting pathophysiological mechanisms of the two diseases and their treatment needs [25]. The pathological feature of asthma is chronic airway inflammation, which requires inhibition of the inflammatory response to control symptoms. Pulmonary tuberculosis, caused by *Mycobacterium tuberculosis* infection, relies on the body's normal cellular immune function to remove the pathogens [26, 27]. Through retrospective analysis, this study found that in patients with bronchial asthma complicated by pulmonary tuberculosis, the combined use of glucocorticoids and anti-tuberculosis therapy demonstrated significant advantages over anti-tuberculosis therapy alone across multiple clinical indicators.

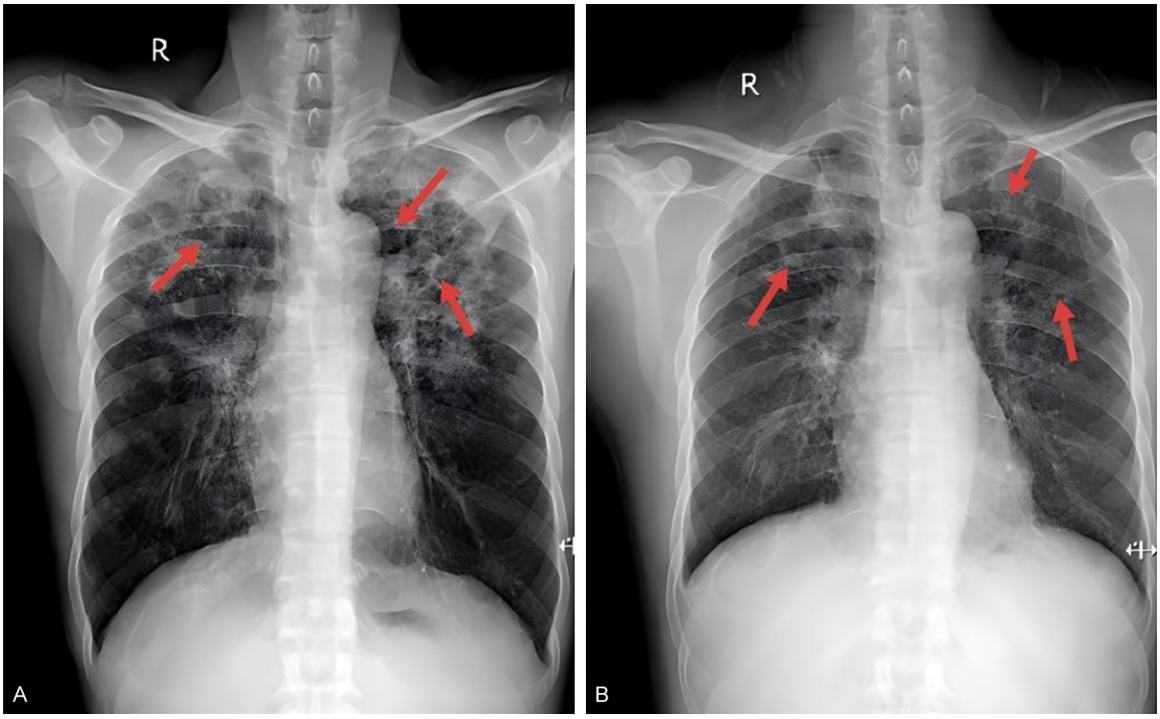

Figure 4. Representative chest X-ray findings in the observation group before and after treatment. A. Before treatment; B. After treatment.

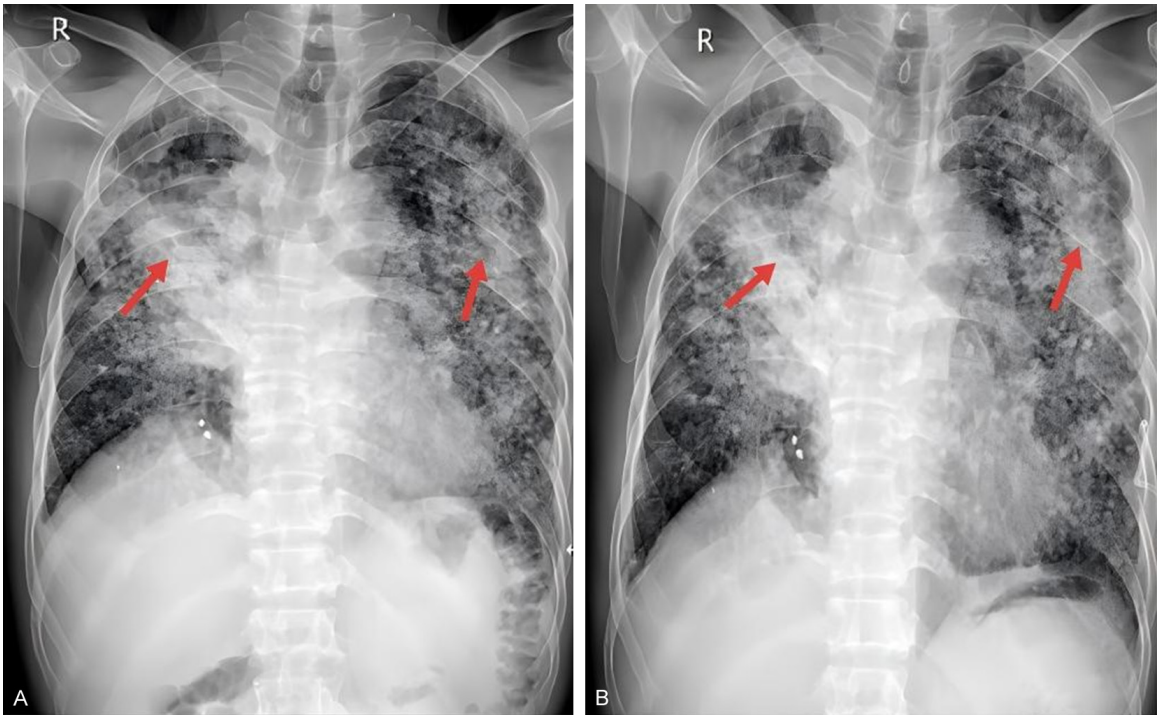

Figure 5. Representative chest X-ray findings in the control group before and after treatment. A. Before treatment; B. After treatment.

From the perspective of clinical symptom improvement, the time to resolution of wheezing, dyspnea, cough, and other symptoms was significantly shorter in the observation group than

Table 4. Comparison of sputum culture conversion rates between the two groups

	Observation group (*n*=67)	Control group (*n*=61)	χ^2	*P*
Before treatment	8 (11.94)	5 (8.20)	0.490	0.484
Two months after treatment	44 (65.67)	29 (47.54)	4.283	0.039
Four months after treatment	54 (80.60)	39 (63.93)	4.462	0.035
Six months after treatment	59 (88.06)	41 (67.21)	8.119	0.004
Recurrence rate	3 (4.48)	10 (16.39)	4.969	0.026

Table 5. Comparison of adverse reaction rates between the two groups

	Observation group (*n*=67)	Control group (*n*=61)	χ^2	*P*
Diarrhea	4 (5.97)	6 (9.84)	0.235	0.628
Pharyngeal discomfort	5 (7.46)	7 (11.48)	0.605	0.197
Abnormal liver function	1 (1.49)	3 (4.92)	0.364	0.436
Total incidence	10 (14.92)	16 (26.23)	2.521	0.112

in the control group. This is consistent with the findings of Suzuki et al. [6], which reported that inhaled glucocorticoids can effectively control asthma symptoms without increasing the risk of tuberculosis recurrence. This outcome is likely due to the strong anti-inflammatory effect of glucocorticoids. Budesonide targets cells in airway inflammation by inhibiting the activation and chemotaxis of inflammatory cells in the airway mucosa, reducing the release of inflammatory mediators such as IL-6 and CRP, thereby alleviating airway mucosal edema and relieving asthma symptoms [28]. Compared to anti-tuberculosis treatment alone, which only addresses *Mycobacterium tuberculosis* infection, the combined treatment not only controls asthma-related airway inflammation but also treats the tuberculosis infection, achieving a more rapid relief of symptoms [29].

After six months of treatment, improvements in pulmonary function tests such as FEV_1, FVC, FEV_1/FVC, and MMEF, as well as in ACT and AQLQ scores, were more pronounced in the observation group than in the control group. From a pathological perspective, long-term airway inflammation in asthmatic patients can lead to airway remodeling, manifested as airway smooth muscle thickening and mucosal fibrosis, which in turn leads to irreversible decline in lung function [30]. The lung tissue destruction and cavitary formation caused by tuberculosis further aggravate ventilation dysfunction [31]. While anti-tuberculosis therapy alone can gradually clear pathogens, it fails to effectively suppress asthma-related airway inflammation, making it difficult to halt airway remodeling. In contrast, the combination therapy allows glucocorticoids to suppress inflammatory responses, potentially delaying airway remodeling, while anti-tuberculosis treatment progressively repairs lung tissue damage. The synergistic effect of both therapies achieves significant improvement in lung function [32]. In addition, the significant improvement of AQLQ score in this study was consistent with the conclusion of Choy et al. [19], who found that the improvement of quality of life in asthma patients was positively correlated with the degree of airway inflammation control. The combination therapy further reduces the impact of symptoms on patients' daily activities and emotional states by simultaneously controlling asthma and tuberculosis.

In the regulation of immune inflammation, the decrease in inflammatory indexes such as CRP and IL-6 and immune markers such as IgE, IgG and IgA was greater in the observation group than in the control group. Gai et al. [33] noted in their study of tuberculosis complicated by airway disease that *Mycobacterium tuberculosis* infection activates the innate immune system, prompting macrophages to release substantial quantities of inflammatory mediators such as IL-6 and CRP. These factors further exacerbate airway mucosal damage and hyperresponsiveness. Conversely, the chronic inflammation associated with asthma suppresses T-lymphocyte proliferation and activation, thereby inhibiting the body's specific immune response to *Mycobacterium tuberculosis* and

rendering infection difficult to control [34]. The observed reduction in serum IL-6 and CRP levels suggests that corticosteroids may mitigate airway mucosal damage by inhibiting inflammatory signaling pathways (e.g., NF-κB), thereby reducing the release of pro-inflammatory factors from inflammatory cells including macrophages [35]. Concurrently, corticosteroid regulation of IgE, IgG, and IgA may be related to its modulation of Th1/Th2 balance and suppression of excessive B-cell activation. Although corticosteroids may suppress cellular immunity, their systemic exposure is low when administered as inhaled corticosteroids, and this is unlikely to cause tuberculosis dissemination, particularly with the administration of effective anti-tuberculosis drugs. Moreover, by alleviating asthma-related airway inflammation, edema, and spasm, corticosteroids may improve local microcirculation and drug penetration, thereby enhancing the distribution and bactericidal efficacy of anti-tuberculosis drugs at the lesion site. This may accelerate cavity closure and lesion absorption [36]. Additionally, the observed decrease in IgE, IgG, and IgA levels in the observation group indicated that glucocorticoids exert a regulatory effect on immunoglobulin levels, consistent with the findings of Jabara et al. [37].

From the perspective of tuberculosis control and prognosis, the observation group outperformed the control group in terms of chest X-ray lesion absorption, cavity closure, sputum negative conversion rate, and bacteriological recurrence rate. On the one hand, the standardized quadruple anti-tuberculosis regimen used in combination therapy effectively inhibits the proliferation of *Mycobacterium tuberculosis*, which provides a basis for the safe use of glucocorticoids. On the other hand, although glucocorticoids inhibit cellular immunity to a certain extent, their combined use with effective anti-tuberculosis treatment may improve local drug permeability and the immune microenvironment. By controlling asthma-related inflammation and reducing airway tissue damage and fibrosis, this combination improves the efficacy of anti-tuberculosis drugs and promotes lesion repair [38, 39]. Regarding sputum culture conversion, the observation group demonstrated a higher conversion rate than the control group. This observation is consistent with the findings by Isabella et al. [10], which documented a positive correlation between the effectiveness of anti-tuberculosis treatment and patients' airway patency. The combined treatment regimen employed in this study effectively improved airway patency by alleviating asthma-related airway inflammation, reducing mucosal edema, and relieving bronchospasm. This not only facilitated more thorough distribution of anti-tuberculosis drugs to infected sites but also promoted sputum clearance, indirectly reducing bacterial retention. In terms of safety, the overall incidence of adverse reactions was lower in the observation group than in the control group, with no serious adverse reactions reported, confirming the safety of the combination therapy. Sun et al. [40] showed that budesonide powder inhalation directly targets the airway through local administration, with less systemic absorption. Additionally, gargling after inhalation can further reduce local adverse reactions such as oral candida infection, which may explain the lower incidence of adverse reactions in the observation group.

This study employed non-randomized grouping based on patient treatment preferences, which carries a risk of selection bias due to possible differences in disease severity and treatment adherence between groups. However, a series of targeted control measures were implemented to maximize comparability. First, baseline data were collected, and rigorous statistical testing revealed no significant differences between groups in key characteristics, including sex, age, disease duration, asthma severity, tuberculosis type, smoking history, and underlying medical conditions; Second, treatment protocols were rigorously standardized: both groups received identical anti-tuberculosis regimens and guideline-recommended standard asthma adjunctive therapy, with only the core intervention (corticosteroid use) differing between the groups. Operational procedures and treatment duration were unified to further minimize bias. Finally, objective and standardized observation metrics and assessment protocols were employed. Pulmonary function, inflammatory markers, and immunological markers were measured using identical instruments. Chest X-ray readings were performed in a double-blind manner, and sputum culture conversion was determined based on clear, objective criteria to reduce the influence of subjective errors and compliance differences.

In summary, glucocorticoid combined with anti-tuberculosis drugs in the treatment of bronchial asthma complicated by pulmonary tuberculosis can quickly relieve symptoms, improve lung function, and promote lesion absorption, through the synergistic effect of anti-inflammatory-regulation of immunity-clearance of pathogens, with good safety. The primary innovations of this study are as follows: First, this study addressed the clinical challenge of treating bronchial asthma complicated by active pulmonary tuberculosis by investigating the synergistic effects of combining inhaled corticosteroids with standard anti-tuberculosis therapy. This offers a novel therapeutic approach for tuberculosis patients, where corticosteroid use has traditionally been contraindicated. Second, it systematically evaluated the clinical benefits and risks of combined therapy through multidimensional assessment (e.g., symptoms, pulmonary function, immunological and inflammatory markers, imaging, bacteriological outcomes, and safety).

However, this study had certain limitations. First, the small sample size and single-center design may have introduced selection bias, necessitating future multi-center, large-scale studies to validate these findings. Second, the six-month follow-up period is relatively short, and extended observation is required to assess long-term efficacy and safety of the combined treatment. Finally, differences in formoterol dosage between treatment groups were not corrected through stratified analysis or propensity score matching, which may have influenced assessment of pulmonary function improvement and symptom relief, thereby interfering with the evaluation of the independent effect of corticosteroids. Consequently, the results cannot be fully attributed to the efficacy of corticosteroids. To address this limitation, future research will explore existing clinical data. Specifically, stratified analysis will be employed to subdivide patients based on formoterol dosage and compare the impact of corticosteroid use versus non-use on efficacy measures within the same formoterol dosage group. Additionally, propensity score matching can be used to balance formoterol dosage and other potential confounding factors between groups, thereby revalidating the independent efficacy of corticosteroids in the treatment of bronchial asthma complicated by pulmonary tuberculosis.

Furthermore, this retrospective clinical study was constrained by limitations in clinical sample preservation conditions and detection methods, preventing the conduct of relevant molecular biology experiments. In future research, we will collect additional clinical samples and use flow cytometry to detect the proportions of Th1 and Th2 cells and the IFN-γ/IL-4 ratio in patients' peripheral blood. Western blot will be employed to detect NF-κB p65 phosphorylation levels, thereby clarifying the core pathways through which glucocorticoids regulate inflammatory responses. Concurrently, *in vitro* cell culture combined with *in vivo* animal experiments will be conducted to investigate the effects of glucocorticoids on the immune response.

Conclusion

The combination of inhaled corticosteroids with standard anti-tuberculosis treatment effectively shortens the remission time of clinical symptoms, improves lung function test and quality of life scores, and reduces serum inflammatory markers and immunoglobulin levels. This combination therapy also demonstrates improved radiographic lesion resolution, elevated sputum conversion rates, and reduced bacteriological relapse, without introducing additional adverse effects. Through its synergistic effects on anti-inflammation, immunomodulation, and pathogen clearance, the combination of glucocorticoids with anti-tuberculosis drugs represents an effective and safe clinical treatment option.

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Disclosure of conflict of interest

None.

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