International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-06-231*

Comparative Efficacy of Intranasal Corticosteroids versus Novel Antihistamines in Allergic Rhinitis.

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Received: 02-04-2026
Revised: 24-04-2026
Accepted: 13-05-2026
Published: 30-06-2026

Abstract

Background: Allergic rhinitis (AR) is a highly prevalent, IgE-mediated inflammatory disease of the nasal mucosa. While both Intranasal Corticosteroids (INCS) and novel second-generation oral antihistamines (NAHs) are frequently prescribed, continuous debate exists regarding their comparative efficacy in resolving comprehensive symptom profiles, particularly nasal congestion. ***Objective:*** To comparatively evaluate the clinical efficacy, objective airflow improvement, and safety profiles of a highly selective INCS (Fluticasone Furoate) versus a novel non-sedating antihistamine (Bilastine) in patients with moderate-to-severe persistent allergic rhinitis. ***Methods:*** This prospective, randomized, placebo controlled, parallel-group clinical study includes a total of 140 adult patients were randomized into two groups (Group A: Fluticasone Furoate 110 mcg/day; Group B: Bilastine 20 mg/day) for an 8-week duration. The primary efficacy endpoint was the reduction in the Total Nasal Symptom Score (TNSS). Secondary endpoints included Peak Nasal Inspiratory Flow (PNIF) and the Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ). ***Results:*** Both pharmacological interventions significantly reduced baseline TNSS ($p < 0.001$). However, Group A demonstrated a statistically superior reduction in the overall TNSS at week 8 compared to Group B. The disparity was primarily driven by the resolution of nasal congestion, where INCS yielded a 78% improvement versus 34% in the NAH group. Objective PNIF measurements corroborated these clinical findings. Both drugs exhibited excellent tolerability profiles with no serious adverse events. ***Conclusion:*** While novel antihistamines provide rapid and effective relief of histamine-driven symptoms (pruritus, sneezing), intranasal corticosteroids exhibit superior overall clinical efficacy in moderate-to-severe AR, primarily due to their profound suppression of late-phase mucosal inflammation and severe nasal congestion.

Keywords: Corticosteroids, Allergic Rhinitis, Efficacy, Antihistamine, Allergic Rhinitis.

INTRODUCTION

Allergic rhinitis (AR) is a pervasive, chronic inflammatory disorder characterized by rhinorrhea, paroxysmal sneezing, nasal pruritus, and mucosal congestion. Driven by an abnormal immunoglobulin E (IgE) response to ubiquitous environmental aeroallergens, AR affects an estimated 10% to 30% of the global adult population. Beyond the immediate rhinological manifestations, AR significantly impairs cognitive function, sleep architecture, and overall productivity, representing a substantial socioeconomic burden. [1-2]

The pathophysiology of AR is bifurcated into an early-phase and a late-phase reaction. The early phase occurs within minutes of allergen exposure, driven predominantly by mast cell degranulation and the massive release of histamine, leukotrienes, and prostaglandins. Histamine, acting via peripheral H1 receptors, stimulates the trigeminal nerve endings and local vasculature, resulting in acute sneezing, itching, and rhinorrhea. Conversely, the late-phase reaction, peaking 4 to 12 hours post-exposure, is an intricate cellular response characterized by the profound mucosal infiltration of eosinophils, basophils, T-lymphocytes (specifically the Th2 phenotype), and macrophages. This cellular influx is orchestrated by a complex cytokine and chemokine network, ultimately culminating in chronic, recalcitrant nasal congestion and mucosal hyperreactivity. [2-3]

Given this dual-phase inflammatory cascade, pharmacological interventions must be critically evaluated for their capacity to modulate both acute and chronic pathophysiological mechanisms. Historically, first-generation antihistamines were limited by severe central nervous system (CNS) sedation and anticholinergic effects. The advent of novel, second-generation oral antihistamines (e.g., Bilastine, Rupatadine, Levocetirizine) has revolutionized early-phase symptom management by providing highly selective, non-sedating H1 receptor inverse agonism. [3-5]

Simultaneously, advancements in intranasal corticosteroids (INCS) have yielded molecules with immense glucocorticoid receptor affinity and negligible systemic bioavailability (e.g., Fluticasone Furoate, Mometasone Furoate). INCS exert broad-spectrum anti-inflammatory effects through complex genomic mechanisms, theoretically providing superior suppression of both early and late-phase mediators. [4-5]

Despite clear guidelines from the Allergic Rhinitis and its Impact on Asthma (ARIA) initiative advocating for INCS as first-line therapy in moderate-to-severe disease, real-world prescribing practices often lean toward novel oral antihistamines due to patient preference and perceived ease of administration. This discrepancy highlights the necessity for rigorous, comparative clinical data.

Therefore, this study, developed as a collaborative initiative between clinical Otorhinolaryngology and academic Pharmacology, aims to objectively quantify and compare the clinical efficacy, objective airflow improvement, and safety profiles of Fluticasone Furoate (INCS) versus Bilastine (NAH) in a cohort of adult patients with persistent allergic rhinitis.

MATERIALS AND METHODS

Study Design and Setting

This was an 8-week, prospective, randomized, parallel-group clinical study. The study was conducted at Department of Otorhinolaryngology, 'Maheshwara Medical College and Hospital'. Ethical clearance was obtained from the Institutional Ethics Committee, and the study adhered strictly to the principles outlined in the Declaration of Helsinki. All participants provided written informed consent prior to enrollment.

Participants

Inclusion Criteria:

- Male and female patients aged 18 to 55 years.
- Documented clinical history of moderate-to-severe persistent AR for at least one year, defined according to ARIA guidelines (symptoms present >4 days/week and for >4 consecutive weeks).
- A baseline TNSS of ≥6 (out of a maximum 12) at the screening visit.
- Positive skin prick test (SPT) to at least one common regional aeroallergen (e.g., House Dust Mite, specific pollens).

Exclusion Criteria:

- Presence of rhinitis medicamentosa, severe nasal septal deviation, nasal polyposis, or concurrent acute sinusitis.
- History of asthma requiring daily inhaled corticosteroids (intermittent, mild asthma requiring only PRN bronchodilators was permitted).
- Systemic or topical corticosteroid use within 4 weeks prior to screening.
- Use of oral or topical antihistamines within 2 weeks prior to screening.
- Pregnant or lactating females.

Randomization

Patients were randomly assigned in a 1:1 ratio to either Group A or Group B using a computer-generated block randomization sequence (block size of 4).

Group A (INCS): Received active Fluticasone Furoate nasal spray (110 mcg once daily, 2 sprays per nostril) + Placebo oral tablet once daily.

Group B (NAH): Received active Bilastine oral tablet (20 mg once daily) + Placebo nasal spray once daily. The placebo spray was formulated to match the sensory characteristics (scent and viscosity) of the active INCS.

Clinical Assessments and Outcome Measures

The study spanned 8 weeks, with evaluation visits scheduled at Baseline (Day 0), Week 2, Week 4, and Week 8.

Primary Endpoint: The primary efficacy variable was the mean change from baseline in the reflective Total Nasal Symptom Score (rTNSS) over the 8-week period. The TNSS evaluates four symptoms: nasal congestion, rhinorrhea, nasal pruritus, and sneezing. Each symptom was graded by the patient on a 4-point Likert scale (0 = None, 1 = Mild, 2 = Moderate, 3 = Severe), yielding a maximum total score of 12.

Secondary Endpoints:

1. Peak Nasal Inspiratory Flow (PNIF): An objective measure of nasal airway patency, assessed using a portable Youlten peak flow meter. The best of three forced maximal inspirations (L/min) was recorded at each clinical visit.

2. Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ): A validated 28-item questionnaire assessing the impact of AR on daily activities, sleep, and emotional well-being over the preceding week. Lower scores indicate better QoL.
3. Safety and Tolerability: Spontaneously reported adverse events (AEs) and findings from targeted nasal endoscopies (assessing for mucosal atrophy or epistaxis) were recorded at every visit.

Statistical Analysis

Data were tabulated and analyzed using SPSS Statistics Version 28.0. Continuous variables were expressed as mean ± standard deviation (SD). The assumption of normality was verified using the Shapiro-Wilk test. Intra-group comparisons (baseline vs. post-treatment) were analyzed using paired Student's t-tests. Inter-group comparisons of mean changes were analyzed using an independent samples t-test. For non-parametric data, the Mann-Whitney U test was applied. A two-tailed p-value of < 0.05 was considered statistically significant.

RESULTS

Patient Disposition and Demographics

Of the 152 patients screened, 140 met all eligibility criteria and were randomized. A total of 128 patients (65 in Group A; 63 in Group B) successfully completed the 8-week trial. Withdrawals were primarily due to geographic relocation or loss to follow-up, with no dropouts attributed to severe adverse events. Baseline demographic and clinical characteristics were well-matched between the two cohorts, with no statistically significant differences (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Group A (INCS, n=65)	Group B (NAH, n=63)	p-value
Age (years), Mean ± SD	31.4 ± 8.2	30.9 ±7.9	0.73
Gender (Male/Female)	33 / 32	31 / 32	0.88
Duration of AR (years)	4.2 ± 1.5	4.5 ± 1.8	0.31
Baseline TNSS (0-12)	9.4 ± 1.1	9.2 ± 1.3	0.35
Baseline PNIF (L/min)	88.5 ± 15.2	90.1 ± 14.8	0.55
Baseline RQLQ Score	3.8 ± 0.6	3.9 ± 0.7	0.41

INCS, intranasal corticosteroids; NAH, non-sedating oral antihistamines; TNSS, Total Nasal Symptom Score; PNIF, peak nasal inspiratory flow; RQLQ, Rhinoconjunctivitis Quality of Life Questionnaire; SD, standard deviation.

Primary Efficacy Endpoint: Total Nasal Symptom Score (TNSS)

Both treatment modalities demonstrated a rapid and statistically significant reduction in TNSS from baseline by Week 2 ($p < 0.001$ for both). However, longitudinal analysis revealed a divergent trajectory of efficacy. By Week 8, Group A (INCS) exhibited a statistically superior reduction in the mean TNSS compared to Group B (NAH).

Specifically, the mean reduction in TNSS at Week 8 was 7.1 ± 1.2 for the INCS group, compared to 4.8 ± 1.4 for the NAH group ($p = 0.012$).

When dissecting the individual symptom components of the TNSS, distinct pharmacological profiles emerged. Bilastine (Group B) was highly effective in mitigating sneezing and nasal pruritus, achieving parity with Fluticasone Furoate (Group A) in these specific domains. However, Fluticasone Furoate demonstrated overwhelming superiority in resolving nasal congestion. At Week 8, the congestion sub-score in Group A dropped from a baseline of 2.8 to 0.6 (a 78% reduction), whereas Group B only achieved a reduction from 2.7 to 1.8 (a 34% reduction).

Table 2: Changes in TNSS and Sub-scores over 8 Weeks

Variable	Treatment group	Baseline	Week 2	Week 4	Week 8	Between-group p-value*
Total TNSS	INCS	9.4 ± 1.1	5.2 ± 1.0	3.5 ± 0.8	2.3 ± 0.6	<0.001
	NAH	9.2 ± 1.3	6.0 ± 1.2	5.1 ± 1.1	4.4 ± 1.0	
Congestion sub-score	INCS	2.8 ± 0.4	1.9 ± 0.5	1.1 ± 0.3	0.6 ± 0.2	<0.001
	NAH	2.7 ± 0.5	2.2 ± 0.4	2.0 ± 0.4	1.8 ± 0.3	
Sneezing sub-score	INCS	2.5 ± 0.5	1.1 ± 0.3	0.8 ± 0.2	0.5 ± 0.1	0.XXX
	NAH	2.4 ± 0.6	1.0 ± 0.2	0.9 ± 0.3	0.6 ± 0.2	

***Values are presented as mean ± SD.** *Between-group comparisons should ideally be based on a repeated-measures model or linear mixed-effects model, with adjustment for baseline values where appropriate. The p-values should correspond to the specified statistical analysis.*

Secondary Efficacy Endpoints

Peak Nasal Inspiratory Flow (PNIF): Objective measurements of nasal patency via PNIF strongly correlated with the subjective congestion sub-scores. Both groups improved, but Group A exhibited profound, continuous increases in inspiratory flow. At Week 8, Group A showed a mean increase of 42.5 ± 8.2 L/min from baseline, whereas Group B showed a modest increase of 15.3 ± 6.1 L/min ($p < 0.001$).

Quality of Life (RQLQ): Both pharmacological interventions significantly improved patient quality of life. The global RQLQ score dropped significantly in both cohorts. However, mirroring the TNSS data, Group A achieved a statistically greater improvement in overall QoL at Week 8 compared to Group B. Analysis of the RQLQ sub-domains revealed that patients in Group A reported substantially better sleep quality and reduced daytime fatigue, likely secondary to the superior restoration of nocturnal nasal breathing.

Safety and Tolerability Profiles

Both Fluticasone Furoate and Bilastine were exceptionally well-tolerated. No serious adverse events (SAEs) occurred during the 8-week study period.

In Group A (INCS), the most frequently reported adverse event was mild, transient epistaxis (7.6%, n=5) and localized nasal irritation/dryness (4.6%, n=3). Anterior rhinoscopy confirmed that these events were minor mucosal abrasions without septal perforation, resolving without treatment interruption.

In Group B (NAH), the incidence of somnolence was remarkably low (3.1%, n=2), confirming the non-sedating profile of Bilastine. Mild headache was reported equally across both groups (Group A: 3%, Group B: 4.7%). There were no reports of anticholinergic side effects (e.g., dry mouth, urinary retention) in the antihistamine group

DISCUSSION

The optimal pharmacological management of moderate-to-severe persistent allergic rhinitis (AR) remains a critical focus for both otorhinolaryngologists and pharmacologists. The therapeutic goal is to aggressively suppress the multi-cellular inflammatory cascade that drives the disease, thereby restoring normal nasal function and mitigating the downstream impacts on patient quality of life.[6-8] This prospective, randomized study definitively demonstrated that while a novel oral antihistamine (Bilastine) is highly efficacious for early phase histamine-driven symptoms, an intranasal corticosteroid (Fluticasone Furoate) provides statistically superior overall clinical symptom resolution, primarily via the unparalleled alleviation of nasal congestion.

Mechanistic Divergence

From a pharmacological perspective, the mechanisms underpinning these clinical outcomes are profoundly distinct, reflecting the bifurcated pathophysiology of AR. The early-phase reaction is triggered by mast cell degranulation, flooding the local mucosa with histamine. Bilastine functions as a highly selective inverse agonist at the peripheral H1 receptor, stabilizing the inactive receptor conformation. Due to its unique pharmacokinetic profile combining high lipophilicity with active P-glycoprotein efflux at the blood-brain barrier Bilastine achieves potent peripheral H1 blockade without central nervous system sedation. Our clinical data accurately reflects this mechanism; patients in the Bilastine cohort achieved rapid and significant reductions in early-phase symptoms, namely paroxysmal sneezing and intense pruritus, within the first two weeks of therapy.

However, histamine merely initiates the allergic cascade. The late-phase reaction is predominantly mediated by leukotrienes, eosinophilic basic proteins, and kinins, which orchestrate profound mucosal edema, structural remodeling, and severe congestion. Isolated H1 receptor blockade is mechanistically insufficient to counter this cellular influx. Conversely, the clinical superiority of Fluticasone Furoate is rooted in its genomic pharmacodynamics. By binding to the cytosolic glucocorticoid receptor with exceptionally high affinity, Fluticasone Furoate induces the transrepression of critical pro-inflammatory transcription factors, notably Nuclear Factor-kappa B (NF-KB). This genomic interference effectively shuts down the synthesis of cytokines (IL-4, IL-5, IL-13) and endothelial adhesion molecules, physically reversing mucosal edema and restoring airway patency. [7-10]

Comparison with Similar Clinical Studies

Our findings are highly concordant with the broader landscape of rhinitis literature, yet they provide crucial modern updates by utilizing the latest generation of therapeutic agents.

Efficacy in Symptom Resolution:
The superiority of INCS over oral antihistamines in overall TNSS reduction aligns with a pivotal 2024 meta-analysis by MI Torres et al., which evaluated across multiple RCTs concluding that intranasal treatments are more effective than oral treatments at improving symptoms and quality of life in seasonal allergic rhinitis.[11] While older trials frequently compared first-generation INCS (like beclomethasone) to earlier antihistamines (like cetirizine); our study confirms that the efficacy gap remains wide even when utilizing a highly advanced, non-sedating agent like Bilastine. Similarly, a 2024 comparative trial by Ghanbari et al evaluating desloratadine versus mometasone furoate reported nearly identical trajectories: it tested mometasone furoate in all arms with daily desloratadine, twice-daily desloratadine, montelukast, or desloratadine plus montelukast in 64 children with persistent moderate-to-severe allergic rhinitis.[12] Our data confirms this chronological divergence, with Bilastine plateauing in efficacy against congestion while Fluticasone Furoate continued to drive symptom scores downward.

Objective Airflow Measurements (PNIF):
Subjective symptom scores are notoriously vulnerable to the placebo effect, particularly in rhinitis trials. Therefore, our inclusion of Peak Nasal Inspiratory Flow (PNIF) provides vital objective validation. The robust increase in inspiratory flow observed in the Fluticasone Furoate group (42.5 ± 8.2) strongly mirrors the results published by H.Kaiser wherein perennial allergic rhinitis, fluticasone furoate nasal spray improved daily PNIF more than placebo over 6 weeks. [13] Our PNIF data firmly corroborates that the subjective sensation of "unblocking" reported by INCS users correlates directly with measurable, physical reductions in turbinate hypertrophy.

Quality of Life and Sleep Architecture:
Perhaps the most critical clinical implication of our study lies in the Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ) outcomes. The profound improvement in sleep parameters reported by the INCS cohort underscores that nasal blockage is the primary driver of AR-induced sleep fragmentation. This supports various studies which broader conclusion that nasal congestion drives sleep impairment and daytime somnolence in allergic rhinitis, and that intranasal corticosteroids improve congestion, sleep-related quality of life, and overall rhinitis quality of life more than oral antihistamines. [14-16]

Clinical Implications and Guideline Alignment
These outcomes strongly reinforce the current treatment paradigms established by the Allergic Rhinitis and its Impact on Asthma (ARIA) and European Academy of Allergy and Clinical Immunology (EAACI) guidelines. While patient preference often gravitates toward the convenience of a once-daily oral tablet, clinicians must proactively educate patients with moderate-to-severe, congested phenotypes that oral antihistamines are therapeutically insufficient as monotherapy. INCS must remain the foundational pillar of management for persistent disease.[17]

Limitations of the Study
Despite the rigorous double-dummy design, this study possesses certain limitations. First, the 8-week duration, while sufficient to capture steady-state efficacy, does not fully encapsulate the long-term adherence challenges or potential localized tachyphylaxis associated with year-round nasal spray usage. Second, the study design evaluated these agents strictly as monotherapies. The current vanguard of AR management heavily favors fixed-dose combination therapies (e.g., Azelastine-Fluticasone nasal sprays), which synergize topical antihistaminic action with corticosteroid efficacy. By excluding a combination therapy arm, this trial cannot comment on how Bilastine or Fluticasone Furoate performs against the absolute latest standard of severe disease management. Finally, being a single-center study, the aeroallergen profile driving the patients' AR was heavily localized, which may slightly limit the geographical generalizability of the findings. Future multi-center research should prioritize comparing targeted INCS monotherapy against fixed-dose combination sprays in persistent AR phenotypes.

CONCLUSION
This randomized controlled trial substantiates the pharmacological and clinical superiority of Intranasal Corticosteroids over novel oral antihistamines in the management of moderate-to-severe persistent allergic rhinitis. While novel antihistamines like Bilastine offer rapid, non-sedating relief of acute pruritus and sneezing, they lack the broad-spectrum anti-inflammatory potency required to resolve established mucosal edema. Fluticasone Furoate demonstrated unmatched efficacy in resolving nasal congestion, confirmed by significant objective improvements in Peak Nasal Inspiratory Flow, leading to superior overall symptom control and enhanced patient quality of life. Consequently, clinical prescribing practices for persistent, severe AR should adhere closely to current guidelines, prioritizing the early and consistent initiation of topical corticosteroid therapy.

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