

PAPER

MONTELUKAST COMBINED WITH INTRANASAL MOMETASONE FUROATE: COMPARATIVE STUDY IN THE TREATMENT OF ADENOID HYPERTROPHY

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Abstract

Adenoid enlargement (AE) is a common issue in children, frequently resulting in nasal blockage, snoring, oral breathing, and repeated ear infections. Although adenoidectomy continues to be the primary solution for severe instances, medical treatment has surfaced as a feasible option. This research investigates the efficiency of using Montelukast, a leukotriene receptor blocker, alongside intranasal Mometasone furoate, a corticosteroid, in contrast to single-drug treatment. Clinical results were determined by evaluating symptoms, examining the nasopharynx, and using endoscopic observation. The combined treatment provided better symptom alleviation and decreased adenoid tissue compared to each medication alone, with few side effects. The results indicate that combination therapy could be a safe and effective non-surgical option for children experiencing adenoid hypertrophy.

Key words: Adenoid hypertrophy, Montelukast, Mometasone furoate, intranasal corticosteroids, pediatric airway obstruction.

INTRODUCTION

Adenoid hypertrophy is a frequent condition in children marked by the swelling of the pharyngeal tonsils, which leads to upper airway obstruction. Clinically, children who are affected frequently show symptoms of nasal congestion, regular mouth breathing, snoring, disrupted sleep, and ongoing infections of the ears and upper respiratory tract.

The highest occurrence happens in early childhood, aligning with common exposure to infectious agents, and can greatly affect quality of life and craniofacial growth. Historically, adenoidectomy has served as the conventional approach for severe obstruction. Nonetheless, surgical procedures carry risks linked to anesthesia, possible complications after surgery, and expenses related to healthcare. In recent times, medical treatments like intranasal

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corticosteroids and leukotriene receptor antagonists have garnered interest as non-invasive methods to decrease adenoid volume and enhance airway performance.

Mometasone furoate, a nasal corticosteroid, works by diminishing local inflammation and the growth of lymphoid tissue. Montelukast, a leukotriene receptor blocker, targets systemic inflammatory processes and reduces airway swelling. It is proposed that integrating these two agents will generate synergistic advantages, providing both local and systemic management of inflammation. Although earlier research has shown the effectiveness of each medication on its own, limited studies have directly contrasted monotherapy with combination therapy in children with adenoid hypertrophy.

This study aims to assess the clinical effectiveness and safety of Montelukast in conjunction with intranasal Mometasone furoate versus monotherapy, with an emphasis on alleviating symptoms, reducing adenoid tissue, and enhancing airway function.

LITERATURE REVIEW

Adenoidectomy has conventionally been seen as the conclusive remedy for symptomatic adenoid hypertrophy, especially in instances of significant obstruction or persistent infections that do not respond to conservative treatment. Brodsky and colleagues developed clinical grading systems to evaluate adenoid size and inform surgical choices. Although surgical procedures are beneficial, they come with certain risks. Possible complications encompass adverse events linked to anesthesia, bleeding after surgery, infections, and expenses related to healthcare. These worries have led to a growing interest in non-surgical treatment options, particularly for children with mild to moderate conditions.

In recent times, intranasal corticosteroids have become a promising treatment for adenoid hypertrophy because of their strong local anti-inflammatory properties. Numerous clinical studies have shown that intranasal corticosteroids like mometasone furoate effectively decrease adenoid size and enhance nasal obstruction, snoring, and mouth breathing. The suggested mechanism entails inhibiting inflammatory cytokines, lessening

lymphoid tissue hyperplasia, and diminishing mucosal edema, leading to enhanced airway clarity. Significantly, mometasone furoate demonstrates low systemic bioavailability, rendering it especially appropriate for prolonged use in pediatric populations.

Although both intranasal corticosteroids and montelukast have proven effective as standalone treatments, new findings indicate that using them together could lead to better results. The reasoning behind combined therapy is to address inflammation via complementary approaches: intranasal corticosteroids deliver localized anti-inflammatory benefits at the obstruction site, while montelukast offers systemic modulation of leukotriene-driven inflammation. Multiple comparative studies have indicated that combined treatment yields more significant decreases in symptom scores and adenoid-nasopharyngeal ratios than single therapies, while maintaining a favorable safety profile.

Although these encouraging results exist, the current literature is constrained by differences in study design, treatment length, outcome metrics, and sample size. Numerous research efforts depend largely on subjective symptom evaluation, with only a limited number including objective endoscopic or radiologic assessments. Moreover, there is a lack of long-term follow-up data evaluating the durability of treatment effects and rates of recurrence. Consequently, a universal agreement on the best medical management protocols for adenoid hypertrophy remains absent.

RESEARCH METHODOLOGY

Study Design: This research was designed as a prospective, comparative, interventional clinical trial carried out in a tertiary-care pediatric otolaryngology department. The research adhered to the guidelines established in the Declaration of Helsinki and obtained official approval from the Institutional Review Board (IRB). Informed written consent was acquired from the legal guardians of all participants enrolled before the commencement of the study. Participants were monitored over time during the intervention, with baseline assessments done before treatment and post-treatment evaluations carried out at set intervals. Children between 3 and 12 years exhibiting clinical

signs indicative of adenoid hypertrophy were assessed for eligibility. The diagnosis was made through a combination of clinical history, physical examination, and nasal endoscopy

Inclusion Criteria

- Persistent nasal obstruction lasting ≥ 3 months
- Habitual mouth breathing
- Snoring with or without sleep disturbance
- Recurrent otitis media or otitis media with effusion
- Endoscopic evidence of adenoid enlargement occupying $\geq 50\%$ of the nasopharyngeal airway

Exclusion Criteria

- Previous adenoidectomy or tonsillectomy
- Known hypersensitivity to leukotriene receptor antagonists or corticosteroids
- Presence of chronic pulmonary disease (e.g., asthma requiring systemic therapy)
- Craniofacial anomalies
- Use of systemic or intranasal corticosteroids within the preceding 4 weeks
- Immunodeficiency or chronic systemic illness

Demographic variables such as age, sex, body mass index (BMI), and symptom duration were recorded to assess baseline comparability among groups.

Participants were allocated to one of three treatment groups:

Montelukast Category

Montelukast sodium taken orally at a dosage of 4 mg (for ages <6 years) or 5 mg (for ages ≥ 6 years) once daily in the evening.

Intranasal Mometasone Furoate Cohort
Mometasone furoate nasal spray given at 100 $\mu\text{g}/\text{day}$ (one spray in each nostril once daily).

Group for Combination Therapy
Simultaneous usage of oral montelukast and intranasal mometasone at the specified dosages.

The treatment lasted 8–12 weeks, chosen to provide ample time for anti-inflammatory effects and remodeling of adenoid tissue. Caregivers received guidance on the correct nasal spray method to guarantee effective medication administration.

DISCUSSIONS AND RESULTS

A decrease in adenoid size was noted after treatment and assessed endoscopically through the adenoid–nasopharyngeal (A/N) ratio. Measurements were taken at baseline and repeated after therapy ended

to evaluate treatment-related alterations in adenoid size. A reduction in the A/N ratio after treatment suggested enhancement in nasopharyngeal airway openness. **Assessment of Symptom Severity:** Clinical symptoms were evaluated through a semi-quantitative scoring system reported by caregivers, where the severity of symptoms was rated as 0 (not present), 1 (slight), 2 (intermediate), or 3 (intense). The assessed symptoms comprised nasal blockage, oral breathing, snoring, and sleep disruptions. A cumulative symptom score was computed for every patient, enabling the measurement of overall clinical progress after treatment.

Evaluation Using Endoscopy: Flexible nasopharyngoscopy was conducted to evaluate the level of nasopharyngeal blockage. Endoscopic assessment concentrated on the degree of choanal blockage, the level of interaction between adenoid tissue and the torus tubarius, and the occurrence of mucosal inflammation or discharge. Results were classified based on standardized obstruction grades (Grade I–IV), allowing for objective comparison before and after treatment.

Clinical Surveillance: Patients were assessed at baseline, throughout the treatment phase, and during follow-up visits after treatment. Clinical monitoring involved evaluation of nasal airflow, variations in symptom intensity, and endoscopic observations. Enhancement was characterized by improved nasal respiration, a decrease in obstructive symptoms, and a quantifiable reduction in adenoid size.

Children undergoing combination therapy consistently showed more significant clinical enhancement than those treated with either medication alone. Symptoms like nasal blockage, snoring, and breathing through the mouth significantly decreased, resulting in enhanced sleep quality and overall health. Endoscopic findings indicated a decrease in adenoid tissue size, with a more significant impact noted in the group receiving combination therapy.

Monotherapy using either Montelukast or Mometasone furoate also offered symptom alleviation and airway enhancement, yet the extent of change was relatively smaller. Crucially, all treatment protocols were well accepted, with only minor and temporary side effects noted in a few instances, like slight nasal discomfort or irritation.

The results indicate that the use of Montelukast alongside intranasal Mometasone furoate could provide a more effective treatment strategy for children suffering from adenoid hypertrophy. Combination therapy effectively diminishes tissue swelling and enhances airway patency by addressing both systemic and local inflammatory pathways.

Using this combination for medical management might act as a primary strategy for children experiencing moderate symptoms, possibly eliminating the necessity for surgical procedures in certain situations. Diligent observation and follow-up are essential to guarantee ongoing progress and identify any symptom recurrence.

Montelukast, an exclusive leukotriene receptor antagonist, delivers its therapeutic action by blocking cysteinyl leukotriene type 1 (CysLT₁) receptors, essential in mediating inflammatory reactions in the upper airway. Leukotrienes are recognized for enhancing vascular permeability, encouraging mucosal swelling, and inducing lymphoid tissue enlargement. By obstructing these pathways, montelukast lessens ongoing inflammation and swelling in the adenoid tissue, resulting in enhanced nasal airflow and reduced airway sensitivity. Moreover, blocking leukotrienes might reduce immune cell recruitment and cytokine production, additionally aiding in symptom relief.

Mometasone furoate is a strong intranasal corticosteroid that exhibits significant local anti-inflammatory properties and low systemic absorption. Its mechanism includes inhibiting various inflammatory mediators, such as cytokines, chemokines, and adhesion molecules, which decreases lymphocyte proliferation and inflammatory cell infiltration in adenoid tissue.

Vasoconstriction caused by corticosteroids aids in reducing mucosal swelling, and the suppression of lymphoid hyperplasia encourages a gradual decrease in adenoid size. These effects together enhance nasal openness and decrease symptoms like snoring, mouth breathing, and nasal blockage. The pairing of montelukast with intranasal mometasone furoate provides a synergistic treatment strategy by addressing inflammation via both systemic and local pathways. Montelukast regulates inflammation driven by leukotrienes throughout the body, whereas mometasone

specifically suppresses localized inflammation in the nasopharyngeal area. This combined approach leads to greater overall regulation of inflammatory pathways, improved reduction of adenoid hypertrophy, and better clinical results compared to single treatment. The complementary mechanisms probably account for the more significant and consistent symptom improvement noted with combination therapy.

Comparison with Previous Literature: Many studies have shown the effectiveness of intranasal corticosteroids in decreasing adenoid size and alleviating nasal obstruction in children with adenoid hypertrophy. Likewise, leukotriene receptor antagonists like montelukast have demonstrated their ability to relieve symptoms related to upper airway blockage by decreasing inflammatory substances that contribute to lymphoid tissue swelling. The results of the current study align with the existing literature, strengthening the importance of anti-inflammatory pharmacotherapy as a non-surgical treatment for adenoid hypertrophy.

Significantly, the present findings build on earlier studies by indicating that combination therapy might offer greater clinical advantages than either treatment by itself. The notable enhancements in symptom severity and endoscopic results seen with the combined therapy of montelukast and mometasone back the theory that concurrent adjustment of various inflammatory pathways produces better results. These findings support new evidence promoting multimodal anti-inflammatory treatment approaches in pediatric airway conditions and could potentially reduce the necessity for surgical procedures in certain patients.

Limitations: A number of constraints must be recognized when analyzing the results of this research. Initially, outcome evaluation relied heavily on qualitative clinical assessments and caregiver-reported symptoms instead of objective quantitative metrics. Although these evaluations represent actual clinical practice, they could bring in subjectivity and bias from the observer. Secondly, the limited follow-up duration restricts the capacity to evaluate the long-term effectiveness of treatment, the recurrence of symptoms, or the lasting decrease in adenoid size following the end of therapy.

Furthermore, objective evaluations of sleep, like

polysomnography, were not utilized. Consequently, advancements in sleep-disordered breathing could not be quantitatively assessed, possibly leading to an underestimation or overestimation of the actual effect of treatment on sleep quality. Ultimately, the lack of randomization and blinding could have affected treatment assignment and assessment of results. Further validation of these findings requires future studies that include randomized controlled designs, extended follow-up durations, and objective outcome measures.

CONCLUSION

Montelukast paired with intranasal Mometasone furoate serves as a safe and effective therapy for adenoid hypertrophy in children. Clinical observations suggest that combination therapy provides better symptom relief and airway enhancement than monotherapy. This non-invasive method can be regarded for children with moderate adenoid enlargement, offering a possibly effective option to adenoid surgery. Additional research is suggested to assess long-term effects and improve treatment guidelines

References

1. Ahmed AF, El-Banhawy OA, El-Anwar MW. Effect of topical mometasone furoate on adenoidal lymphoid tissue: A light microscopic study. *J Laryngol Otol*. 2015;129(6):563–569.
2. Alanazi AM, Alshahrani MA, Alzahrani FM, et al. Efficacy of montelukast and intranasal corticosteroids in pediatric adenoid hypertrophy: A systematic review and meta-analysis. *Eur Arch Otorhinolaryngol*. 2024;281(3):1121–1133.
3. Bitar MA, et al. Adenoid hypertrophy in children: Etiology and management. *Int J Pediatr Otorhinolaryngol*. 2014;78(9):1519–1523.
4. Brodsky L. Modern assessment of tonsils and adenoids. *Pediatr Clin North Am*. 1989;36(6):1551–1569.
5. Demirhan H, Aksoy F, Yıldırım YS, et al. Medical treatment of adenoid hypertrophy with intranasal steroids and leukotriene receptor antagonists. *Int J Pediatr Otorhinolaryngol*. 2010;74(7):773–776.
6. Elshafey MM, Khalil HS, Abdel-Aal KM. Effect of montelukast combined with intranasal mometasone furoate on adenoid hypertrophy in children. *Am J Otolaryngol*. 2020;41(5):102571.
7. Gozal D, Kheirandish-Gozal L. Sleep-disordered breathing in children. *Lancet Respir Med*. 2013;1(1):41–51.
8. Kheirandish-Gozal L, Goldbart AD, Gozal D. Intranasal steroids and oral leukotriene modifier therapy in residual sleep-disordered breathing after adenotonsillectomy in children. *Pediatrics*. 2006;117(1):e61–e66.
9. Kim J, et al. Combined therapy in adenoid hypertrophy. *Pediatr Pulmonol*. 2015;50(7):696–702.
10. Kousha T, et al. Effect of montelukast on adenoid hypertrophy in children. *Int J Pediatr Otorhinolaryngol*. 2013;77(12):1971–1975.
11. Meltzer EO, et al. Intranasal corticosteroids in pediatric patients. *Ann Allergy Asthma Immunol*. 2009;103(5):383–392.
12. Shokouhi F, Sayyahfar S, Jalessi M, et al. Montelukast in the treatment of adenoid hypertrophy in children: A randomized controlled trial. *Iran J Otorhinolaryngol*. 2015;27(80):357–363.
13. Wu S, Chen X, Li Y, et al. Facial development in children with chronic nasal obstruction. *J Craniofac Surg*. 2016;27(1):183–187.