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Evaluation of the effects of montelukast, mometasone furoate, and combined therapy on adenoid size: a randomized, prospective, clinical trial with objective data

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Background/aim: The incidence of adenoid hypertrophy is 2%–3% in children. Adenoidectomy is a commonly performed procedure in children that may cause complications such as early or late bleeding (4%–5%), recurrence of adenoid tissue (10%–20%), and postoperative respiratory problems (27%). Therefore, medical therapy alternatives to adenoidectomy are important and must be tried before surgery. In this study, we investigated the efficacy of mometasone furoate, montelukast, and a combination of these drugs in pediatric patients with adenoid hypertrophy who were scheduled for reduction with medical therapy after not being recommended for surgery.

Materials and methods: The study included 120 children with adenoid hypertrophy aged between 4 and 10 years. The patients were randomized into 4 separate groups, with 30 in each group. Group 1 received 100 µg of mometasone furoate per day, group 2 received 4/5 mg (for age) montelukast per day, and group 3 received mometasone furoate + montelukast. Medical therapy continued for 3 months in the treatment groups. Group 4, which comprised patients with mild symptoms, received no treatment and was the control group. The pre- and posttreatment adenoid tissue ratios in lateral neck radiographs were recorded in the four groups.

Results: When radiologic measurements of adenoid-to-air passage were calculated, an improvement of 21.76% was observed in group 1 after treatment. The rate of improvement was 22.51% in group 2. There was a 21.79% reduction in adenoid size in group 3 after 3 months' treatment and 12.46% in the control group. There were statistically significant differences between pre- and posttreatment values in every single group administered corticosteroids, montelukast, and combined therapy ($P < 0.05$).

Conclusion: According to our results, both montelukast and mometasone furoate therapies were similarly successful in treating adenoid hypertrophy. Combined therapy has no superiority over single-therapy treatment.

Key words: Adenoid, montelukast, mometasone furoate

1. Introduction

Adenoids are pyramid-shaped enlarged masses of lymphoid tissue in the nasopharynx (1), and they form an important part of Waldeyer's tonsillar ring. Although the adenoids are very small at birth, they may exhibit growth in the first 4 years of life, depending on the development of the immune system (2).

The incidence of adenoid hypertrophy is 2%–3% in children (2). Untreated adenoid hypertrophy may lead to difficulty in breathing through the nose, snoring, sleep apnea, nasal speech, ear problems, growth retardation, and craniofacial anomalies (1,2). Various methods are used for the diagnosis of adenoid hypertrophy including lateral head radiography, videofluoroscopy, palpation, and nasal endoscopy; the most recent methods are lateral radiographs and nasal endoscopy (3).

Adenoidectomy is a commonly performed procedure in children that may cause complications such as early or late bleeding (4%–5%), adenoid tissue recurrence (10%–20%), and postoperative respiratory problems (27%) (2). In addition, anesthesia risks are also among the factors that should be taken into account (4). As such, medical therapy alternatives to adenoidectomy are important and must be tried before surgery.

Leukotrienes are inflammatory mediators found in the respiratory system and these play a role in several allergic childhood diseases (5). Cysteinyl leukotriene receptor-1 mediates the inflammatory pathway and has been found in high rates in postoperative adenotonsillar tissues in pediatric patients with obstructive sleep apnea (6–8). Montelukast is an effective and reliable cysteinyl leukotriene receptor antagonist that can be used in oral form and has high bioavailability. It was approved by the United States Food and Drug Administration (FDA) for preventive therapy against the inflammatory component in asthma and allergic rhinitis in children aged over 1 year.

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Furthermore, montelukast has not induced tolerance in long-term studies (9). It is used to inhibit the inflammatory component in the disease group, which is triggered by allergic reactions (5).

Topical nasal steroids are the most effective medical treatment option in controlling symptoms related to allergic rhinitis, most commonly nasal congestion (10,11). The reliability of nasal administration of steroids in children has been reported in a number of studies (12–14). They penetrate the bloodstream at a low rate (0.1%) and bind corticosteroid receptors in tissues at a high rate (15,16). Among these medications, mometasone furoate has been demonstrated to be the most reliable drug in children aged over 2 years (16,17). Only a few adverse effects have been reported, but whether this was due to poor reporting or fewer adverse effects is not clearly known (18).

In the present study, we investigated the efficacy of mometasone furoate, montelukast, and a combination of these drugs in pediatric patients with adenoid hypertrophy who were scheduled to receive reduction using medical therapy after not being recommended for surgery.

2. Materials and methods

The study included 120 patients who presented to the Ear, Nose, and Throat (ENT) outpatient clinic of Adana Numune Training and Research Hospital between January 2016 and July 2016 and were diagnosed followed up for adenoid hypertrophy. The patients were diagnosed as having adenoid hypertrophy in the first 3 months, and in the following months we monitored their symptoms and nasal obstruction. Although prick tests were negative in our patients, we preferred the months before spring in order to minimize allergic reactions. The study was approved by the local ethics committee of Adana Numune Training and Research Hospital (26.04.2016/ ANEAH. EK.2016/85). Patients aged 4–10 years of both sexes with grades 3 and 4 adenoid hypertrophy according to the Cassano classification were enrolled in the study (19). Snoring, mouth breathing, and recurrent sinusitis related to adenoiditis were the main symptoms in varying degrees. These symptoms were scored using a scale between 0 and 10 in order to objectivize, according to information obtained from the parents. The treatment groups were selected randomly, and we formed a control group of patients with mild symptoms. The same symptom scale was used after treatment.

Patients underwent anterior rhinoscopy and flexible nasopharyngoscopy. Children with infections, systemic disease, craniofacial abnormalities, or genetic diseases; those who had previously used anti-allergic drugs for asthma or other allergic diseases; those with (+) prick tests; those receiving immunosuppression therapy for any reason; those recommended for surgery because of advanced-stage nasal obstruction; those who had previously undergone adenoidectomy; and those with mometasone furoate hypersensitivity were excluded from the study. We also excluded children who developed upper respiratory tract infections during follow-up because we know that infection can possibly increase adenoid size, which would affect our study.

Grading of adenoidal hypertrophy was performed as described by Cassano et al. According to this classification, patients with 50%–75% obstruction of adenoid tissue in the endoscopic view are grade 3 and total choanal obstruction with adenoid tissue is grade 4. We excluded the <25% obstruction as grade 1 and 25%–50% as grade 2 (19). We excluded patients who had severe symptoms such as obstructive sleep apnea that required surgery. Lateral head radiographs were performed and assessed in all patients included in the study. Radiographic evaluation of the nasopharynx has been established as a simple method for determining the size, shape, and position of the adenoids. Evaluation of the airway was performed using lateral neck radiographs with standard techniques in the radiology department of our hospital. The neck was extended, and patients were asked to breathe through their nose. The adenoidal/nasopharyngeal ratio was measured according to the method described by Fujioka et al. (20,21). Adenoid tissue and the distance between the sphenobasiooccput and the posterior end of the hard palate were measured and proportioned (Figure 1). Lateral neck radiographs were performed before and after 3 months of follow-up.

This study was performed as a randomized and prospective clinical trial. Patients were divided into 4 groups, with 30 in each group. Group 1 received 1 puff of mometasone furoate nasal spray in each nostril (50 μg/ puff) once a day, group 2 was given 4 or 5 mg (according to age) of oral montelukast per day, group 3 was given mometasone furoate + montelukast therapies, and group 4 received no medicine and was the control group. In the treatment groups, medical therapy was continued for 3 months. Patients receiving treatment were called for follow-up once per month. Patients were checked at the each visit for treatment adherence and any adverse effects were questioned. Lateral head radiographs were repeated after 3 months and the adenoid tissue/air passage ratio was measured. Pre- and posttreatment values were recorded in all groups.

Data obtained in this study were analyzed using SPSS 20. The Shapiro–Wilk test was used to investigate the normality of the variables due to the unit numbers. When the results were interpreted, significance level was set at 0.05 with P values < 0.05 indicating variables with nonnormal distribution, and P values > 0.05 showed variables with normal distribution. The Kruskal–Wallis–H test was used to analyze differences between the groups

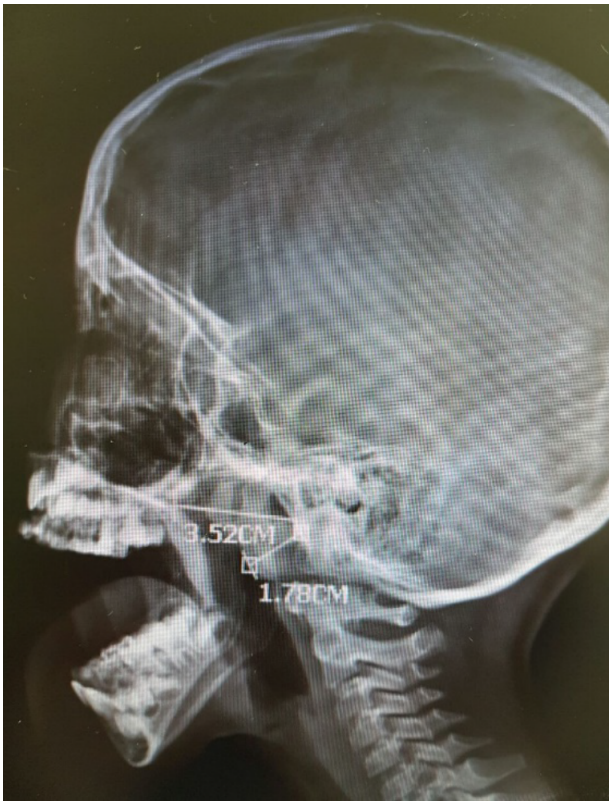

Figure 1. Lateral neck radiograph measuring the ratio between adenoid tissue and the distance between the sphenobasioocciput and the posterior end of the hard palate.

because the variables did not show normal distribution. In the event that significant differences were obtained from the Kruskal–Wallis–H test, the post-hoc multiple comparison test was used to determine the groups causing the differences. Chi-square analysis was performed to examine the correlations of nominal variables between the groups. Fisher's exact test was used in the case of cells with insufficient volumes in 2 × 2 tables, and Pearson's chi-square analysis was used through Monte Carlo simulation in R × C tables. The Wilcoxon test was used to analyze differences between two dependent variables because the variables did not show normal distribution.

When the results were interpreted, significance level was set at 0.05 with P values < 0.05 indicating significant difference; P > 0.05 showed no significant difference.

3. Results

A total of 120 patients comprising 57 girls and 63 boys who presented to the ENT outpatient clinic of Adana Numune Training and Research Hospital were included in the study. The ages of the patients ranged between 4 and 10 years. The mean age (±SD) was 6.97 ± 2.01 years. The mean ages of groups 1, 2, 3, and 4 were 6.83 ± 2.05 years, 6.73 ± 2.20 years, 7.37 ± 1.85 years, and 6.93 ± 1.96 years. There were no statistical differences between the mean ages of all four groups. Informed written consent was obtained for all patients. No withdrawal or treatment-related adverse effects were observed. All patients presented (–) prick tests.

There were statistically significant differences between the pre- and posttreatment values in the groups administered corticosteroids, montelukast, and combined therapy (P < 0.05). However, when compared with each other, no statistically significant difference was found among the three groups (Table 1; Figure 2).

There was a significant difference between the groups regarding the change in obstruction rate (P < 0.05). The percentage of reduction in the treatment groups was higher than in the placebo group.

When the radiologic adenoid-to-air passage measurement was calculated, an improvement of 21.76% was observed in group 1 and of 22.51% in group 2 after treatment. There was a reduction of 21.79% in adenoid size in group 3 after 3 months' treatment. We found the least recovery in the untreated group at 12.46% (Table 2; Figure 3).

Table 1. Pre- and posttreatment obstruction values.

		Group						Wilcoxon test		
		n	Mean	Median	Min.	Max.	SD	Mean rank	z	P
Corticosteroid	0-month obstruction ratio (%)	30	77.43	80	50	90	14.2	15.5	–4.825	0.001
	3-month obstruction ratio (%)	30	60.4	60	40	80	12.83	0		
Montelukast	0-month obstruction ratio (%)	30	68.3	70	50	81	11.15	15.5	–4.807	0.001
	3-month obstruction ratio (%)	30	53.53	50	20	70	13.47	0		
Corticosteroid + montelukast	0-month obstruction ratio (%)	30	82.33	90	50	90	11.12	15	–4.775	0.001
	3-month obstruction ratio (%)	30	64.8	67.5	35	90	13.59	0		
Nontreatment group	0-month obstruction ratio (%)	30	71.87	70	50	90	13	14	–4.578	0.001
	3-month obstruction ratio (%)	30	63.07	65	40	90	13.53	0		

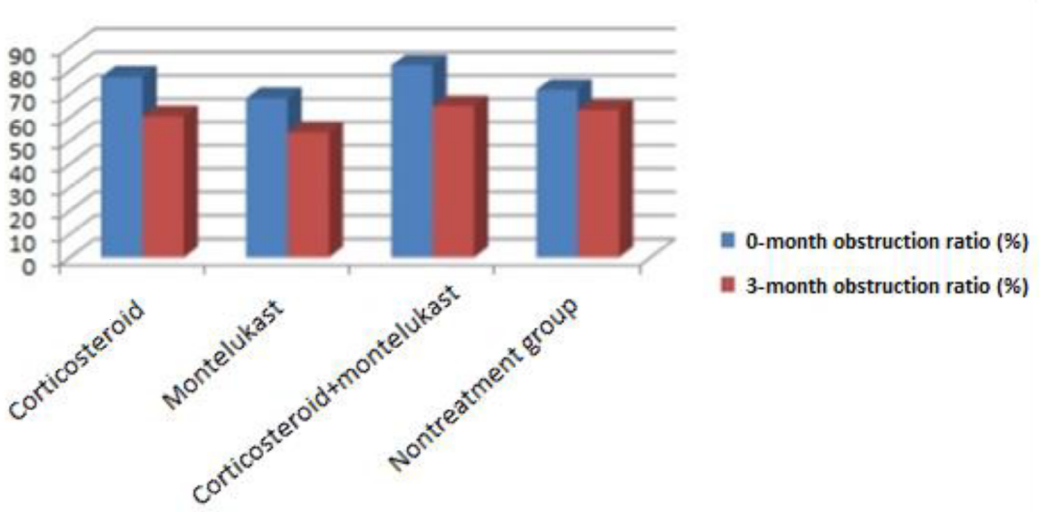

Figure 2. Degrees of obstruction before and after treatments in each group.

Table 2. Kruskal–Wallis–H test results regarding reduction.

n		Group						Kruskal–Wallis–H test		
		Mean	Median	Min.	Max.	SD	Average	H	P	
Change in obstruction rate-% variation	Corticosteroid	30	−21.76	−24.65	−33.33	−9.21	9.52	52.92	20.327	0.001
	Montelukast	30	−22.51	−20.63	−60	−12.5	10.41	51.08		
	Corticosteroid + montelukast	30	−21.79	−22.22	−50	0	9.8	52.78		
	Placebo (control)	30	−12.46	−12.5	−28.57	0	7.84	85.22		
	Total	120	−19.62	−20	−60	0	10.21	2–4 3–4 1–4		
Change of obstruction rate	Corticosteroid	30	−17.03	−17	−30	−5	8.66	52.53	25.184	0.001
	Montelukast	30	−14.77	−13	−30	−10	5.59	56.92		
	Corticosteroid + montelukast	30	−17.54	−20	−40	0	7.71	45.97		
	Placebo (control)	30	−8.8	−10	−20	0	5.65	86.58		
	Total	120	−14.53	−12	−40	0	7.77	3–4 1–4 2–4		

We observed recovery after treatment in all treatment groups, but in the combined therapy group, the recovery in symptom scores was statistically significant compared with the control group (Table 3). We observed clinical recovery from symptoms as reported by parents in all groups, but significance only appeared in the combined therapy group.

4. Discussion

Adenoid hypertrophy is a common disorder of childhood. The adenoids or nasopharyngeal tonsils are lymphoepithelial organs situated in a critical anatomic position in the roof of the nasopharynx. Normal adenoids attain their maximum size between the ages of 3 and 7 years and then regress. Hyperplasia usually follows upper respiratory tract infection. Chronic or recurrent infections are the two most common manifestations of pathologic and physiologic changes in the adenoids. If infection does not occur, adenoid tissue can regress in the process of time (22). It leads to symptoms including nasal obstruction, oral breathing, sleep disturbances, infections in the ear and sinuses, and reduced quality of life. Despite definitive treatment with adenoidectomy, adenoid tissue may grow after infections or chronic allergic reactions, and owing to its complications (hemorrhage, infections, palate dysfunction, emotional stress patients and families, and the risks of general anesthesia), alternative treatments have emerged over time. Ren et al. reported hypernasality

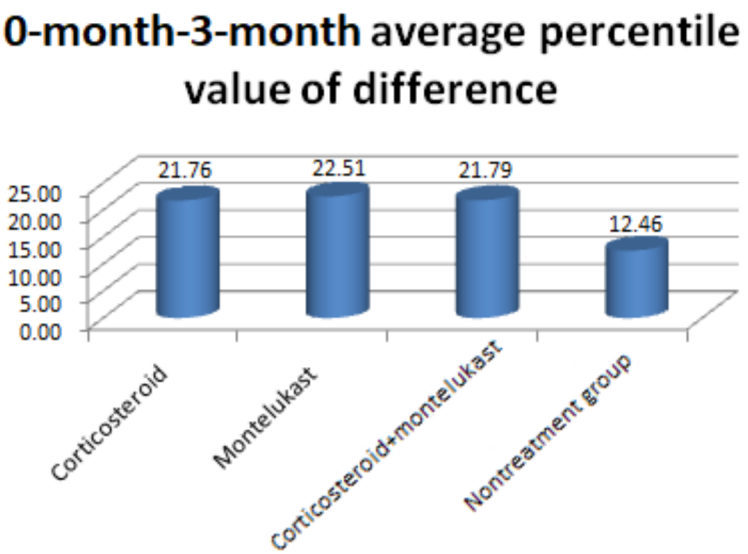

Figure 3. Difference reduction depending on treatment.

Table 3. Comparison of recovery in symptom scores after treatment between groups.

Multiple Comparisons							
Measure:MEASURE_1							
	Group	Group	Mean difference	Std. error	Sig.	95% confidence interval	
						Lower bound	Upper bound
Bonferroni	Corticosteroid	Montelukast	0.6500	0.28639	0.150	−0.1188	1.4188
		CS + montelukast	−0.5667	0.28639	0.301	−1.3354	0.2021
		Placebo	0.6500	0.28639	0.150	−0.1188	1.4188
	Montelukast	Corticosteroid	−0.6500	0.28639	0.150	−1.4188	0.1188
		CS + montelukast	−1.2167*	0.28639	0.000	−1.9854	−0.4479
		Placebo	0.0000	0.28639	1.000	−0.7688	0.7688
	CS + montelukast	Corticosteroid	0.5667	0.28639	0.301	−0.2021	1.3354
		Montelukast	1.2167*	0.28639	0.000	0.4479	1.9854
		Placebo	1.2167*	0.28639	0.000	0.4479	1.9854
	Placebo	Corticosteroid	−0.6500	0.28639	0.150	−1.4188	0.1188
		Montelukast	0.0000	0.28639	1.000	−0.7688	0.7688
		CS + montelukast	−1.2167*	0.28639	0.000	−1.9854	−0.4479
Dunnett t (2-sided)[a]	Corticosteroid	Placebo	0.6500	0.28639	0.065	−0.0317	1.3317
	Montelukast	Placebo	0.0000	0.28639	1.000	−0.6817	0.6817
	CS + montelukast	Placebo	1.2167*	0.28639	0.000	0.5350	1.8983

Based on observed means.

The error term is Mean Square (Error) = 1.230.

*. The mean difference is significant at the 0.05 level.

a. Dunnett t-tests treat one group as a control, and compare all other groups against it.

that developed due to adenoidectomy in 16 patients who had no predisposing factors such as palatal defects (23). In a study by Abdel-Aziz et al., 18 patients developed velopharyngeal insufficiency after adenoidectomy. The authors applied speech therapy to these patients for 3 months, but had to perform pharyngoplasty in 10 patients who had no response to the speech therapy (24). Furthermore, adenoid size may increase again in children who have undergone adenoidectomy. As with other clinics, we sometimes encounter these complications after performing adenoidectomies, and so what can we use as an alternative to surgical treatment, which has so much complications?

Intranasal corticosteroid use is one of the most commonly published methods in the literature on this subject. In their studies, Zhang et al. demonstrated that intranasal corticosteroids were quite efficient in reducing adenoid size (10). In a meta-analysis of 87 studies conducted in children with adenoid hypertrophy, Chohan et al. evaluated intranasal mometasone furoate in terms of nasal symptoms, adenoid size or adenoid/choana ratio, and improvement of otitis media with effusion and obstructive sleep apnea, and the authors stated that it was effective (18). Bhargava et al. used intranasal mometasone furoate in 62 patients and evaluated pre- and posttreatment adenoid size, improvement of otitis media with effusion, which might develop according to adenoid hypertrophy, and they demonstrated statistical effectiveness of intranasal steroids therapy (1). Similarly, in our study, the results of the group that received intranasal steroid therapy indicated that it was effective in reducing adenoid size or the adenoid choana ratio. In a randomized, prospective study by Yılmaz et al. of 28 adolescents aged 12–18 years, mometasone furoate was found to be effective in reducing symptoms, but it did not make a difference in adenoid size (25).

There are different views in the literature about the mechanism of intranasal steroids, which provide a reduction in adenoid hypertrophy. Demain et al. presented a view of the regression in adenoid tissue stating that it might be provided by intranasal steroids through a direct lympholytic pathway, antiinflammatory impact, or inhibition of reservoir infection (26). We suppose that these three mechanisms may have influenced the regression of adenoid size in our study.

Leukotrienes are inflammatory mediators active in childhood diseases related to the respiratory system and are among factors considered to have a role in the pathogenesis of adenoid hypertrophy. Based on this, leukotriene receptor antagonists can also be considered among the alternative treatments to adenoidectomy. In their study in children with obstructive sleep apnea, Goldbart et al. demonstrated that a 16-week treatment period with montelukast provided a statistically significant reduction in adenoid size (5). In our study, we also found regression in adenoid size in the group receiving montelukast. Again, in another study in pediatric patients (n = 60) with adenoid hypertrophy causing more than 75% choanal obstruction, Shokouhi et al. observed a reduction of 76% in the group receiving montelukast, whereas this rate was only 3% in the placebo group (2).

Although the effectiveness of intranasal steroids or oral montelukast has been separately investigated in a number of studies, no study was found in the literature comparing these two medicines on adenoid hypertrophy. Vuralkan et al. compared intranasal mometasone furoate and montelukast in patients with nasal polyposis and reported no statistically significant difference between either preparation in the reduction of symptoms, although they found intranasal steroids were more efficient in the prevention of polyp recurrence (27). In their study of patients with seasonal allergic rhinitis, Martin et al. reported that the effectiveness of intranasal steroids in symptom reduction was statistically significant compared with montelukast (28). In the present study, both leukotriene antagonists and intranasal steroids separately provided reduction in adenoid size, but we could not demonstrate statistical superiority of one over the other. At this point, the question could arise as to whether combined administration of both these medications would contribute to improvement. Friedmann and Goldman administered combined therapy in their 4-year-old patient whose relatives had rejected surgery. They observed that the combined therapy was effective and reported that surgery might be replaced by antiinflammatory therapy in patients with mild obstructive sleep apnea (OSAS) or postoperative residual OSA (29). In a study by Khirandish with 22 patients, a combination of budesonide and montelukast was administered for 12 weeks for OSAS due to residual adenoid tissue following adenotonsillectomy and a significant improvement in the apnea hypopnea index was found (30). According to our results, the combination therapy is effective at reducing adenoid size, but superiority over montelukast or steroid alone could not be established.

According to our results, both montelukast and mometasone furoate therapies were similarly successful in the treatment of adenoid hypertrophy. Both treatment methods may separately be an alternative option to surgery depending on treatment adherence by the patients. Larger studies are warranted for dosage and duration of use.

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