

# Are nasal steroids effective in children with adenoid hypertrophy?

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## ABSTRACT

**Objectives:** Chronic nasal obstruction is a common disease of childhood. Adenotonsillar hypertrophy plays an important role in obstructive sleep apnea. The topical use of the aerosolized forms of corticosteroids therefore seems the most appropriate route to decrease systemic side effects. The aim of our study is to demonstrate the effect of topical mometasone furoate especially on the adenoid volume in patients without any allergic story.

**Methods:** The study group consisting of 30 males and 25 females was administered topical nasal mometasone furoate steroid treatment. The 20 patients were in the control group where saline solution (0.9% NaCl) treatment was administered consisted of 12 males and 8 females. Nasopharyngeal X-rays before treatment revealed that 25 patients were Grade 2 and 30 patients were Grade 3 according to the Fujioka method.

**Results:** Flexible endoscopy performed before the treatment revealed that 20 patients were Grade 2, 11 patients were Grade 3 and 24 patients were Grade 4. Nasal endoscopies performed after 6 weeks of intranasal topical steroid therapy revealed that 45 patients were Grade 1 and 10 patients were Grade 2. A statistically significant difference was present between endoscopic grades before and after treatment ( $p < 0.0001$ ). Nasal endoscopies performed after 6 weeks in control group receiving saline solution treatment revealed Grade 2 in 7 patients, Grade 3 in 10 patients and Grade 4 in 3 patients. There was no statistically significant difference between in the prior and later grades of the control group ( $p = 0.3125$ ).

**Conclusions:** We believe that the use of intranasal steroids (mometasone furoate) for 6 weeks in patients with pediatric chronic nasal obstruction due to adenoid hypertrophy may be an effective treatment modality in alleviating symptoms and decreasing adenoid volume without causing systemic side effects.

**Keywords:** Nasal steroids, adenoid hypertrophy, flexible endoscopy, nasopharyngeal X-rays, Fujioka method

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Chronic nasal obstruction (CNO) is a common disease of childhood with an estimated prevalence of 2-3% in the healthy pediatric population [1]. Adenotonsillar hypertrophy plays an important role in obstructive sleep apnea (OSA) pathophysiology and

reaches its peak incidence between the ages of 2 and 8 years [2, 3]. OSA is the result of increased airway resistance during sleep and characteristically manifests as repeated arousals, hypercapnia, episodic snoring, and periods of oxyhemoglobin desaturation [4]. Ade-



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adenoid hypertrophy is the most common cause of OSA in preschool children [5]. Allergic exacerbations give rise to the hypertrophy of adenoid tissue but repeated upper respiratory tract infections (URTIs) may also be the cause.

The hypertrophied lymphoid tissue may cause many pathological conditions including snoring, sleep apnea, sinusitis otitis media with effusion and adenoid face [6]. If left untreated, OSA may cause many serious problems such as cognitive and behavioral disorders, systemic and pulmonary hypertension, enuresis, and developmental delay [7]. Adenoidectomy is the definitive treatment modality for upper airway obstruction caused by adenoid hypertrophy but decongestant nasal drops have also been used for its symptomatic management [8].

Structural and neuromuscular pathologies may also cause CNO but the main factor in the pathogenesis of pediatric CNO is the size of the tonsils and adenoid tissue making their surgical excision the primary choice of treatment [9, 10]. Postoperative residual OSAS is found in 20% of the children who have undergone adenotonsillectomy [11].

The nonsurgical treatment options for pediatric CNO are attracting increasing attention due to the potential complications of adenotonsillectomy. Anti-allergic drugs have been used from time to time despite the lack of adequate evidence to support their usage, as allergy is just one of the reasons for obstruction [12]. The efficacy of oral steroids in relieving the obstructive symptoms of adenoid hypertrophy is reported in the literature. They also reduce the size of the adenoid tissue significantly. However, their long-term use is limited due to the significant side effects [12]. The topical use of the aerosolized forms of corticosteroids therefore seems the most appropriate route to decrease systemic side effects, since there will be a minimal amount of systemic absorption from the upper airway [13]. The 6-week administration of triamcinolone acetonide aqueous nasal spray to children with allergic rhinitis aged 6 to 12 years has been reported to have no significant impact on adrenocortical function. Treatment with mometasone furoate aqueous nasal spray (200 micrograms once a day) for 14 days was shown to be safe and well-tolerated in children [14].

Local and systemic inflammatory markers and proinflammatory cytokines, which trigger lymphoid tissue proliferation, are increased in children with ade-

notonsillar hypertrophy. Systemic or topical anti-inflammatory agents have therefore been recommended to prevent the potential tonsillar hypertrophy in these patients [15, 16]. Topical nasal corticosteroids can alleviate CNO symptoms and nasal obstruction as well as reduce the size of the adenoid tissue [17, 18]. However, the optimal dose and duration of treatment with nasal steroid agents is not clearly defined yet.

The current inadequate evidence on the efficacy of intranasal steroids in children with adenoid hypertrophy and nasal obstruction led us to conduct this study. With this study we evaluated the efficacy of Mometazon furoate (topical nasal steroid) on adenoid hypertrophy in children.

## METHODS

### Patients

The study was conducted a randomized placebo-controlled children with adenoid hypertrophy who presented to Otolaryngology and Pediatrics outpatient clinics of our hospital. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The Institutional review board (IRB) was taken for the patients (Number: 66519339- 900-01/1304). Informed consent was obtained from all individual participants included in the study. Adenoidectomy operation under general anesthesia was suggested as the main treatment of adenoid hypertrophy to all parents or patients and they were informed about the possible complications of the operation. Mometazon furoate as topical nasal steroid was prescribed for the patients who rejected the surgery and for the relief of symptoms in case of preoperative severe symptoms. All the patients included in the study had a complaint of snoring, cessation of breathing and frequent arousals during sleep. They were aged between 6 to 12 years old.

The diagnosis of adenoid hypertrophy was based on endoscopic nasopharyngeal findings (the occlusion degree of the choana with adenoid tissue) and lateral cephalometric X-ray findings (the thickness of soft tissue density in nasopharyngeal airway) in patients

suspected of having adenoid hypertrophy (i.e., having nasal obstruction without septoconchal pathology, snoring, and/or nasal discharge).

*The inclusion criteria were as follows:*

1. Patients aged between 6 to 12 years old,
2. With a diagnosis of adenoid hypertrophy without tonsillar hypertrophy for a minimum of 12 months,
3. With a follow-up period of 2 months and per 2-week intervals,
4. No sign of improvement despite medical treatment with antibiotics under parental control.

*The exclusion criteria were as follows:*

1. Use of any nasal or systemic steroid within the past 1 year.
2. Use of any nasal decongestant or anti-allergic medication within the past 2 weeks.
3. History of upper respiratory tract infection within the past 2 weeks.
4. History of one or more of the following conditions: genetic craniofacial, or neuromuscular syndromes, chronic epistaxis, immune disease, asthma, nasal surgery, septal perforation, nasal trauma

within the last 3 months and hypersensitivity to mometazon furoat.

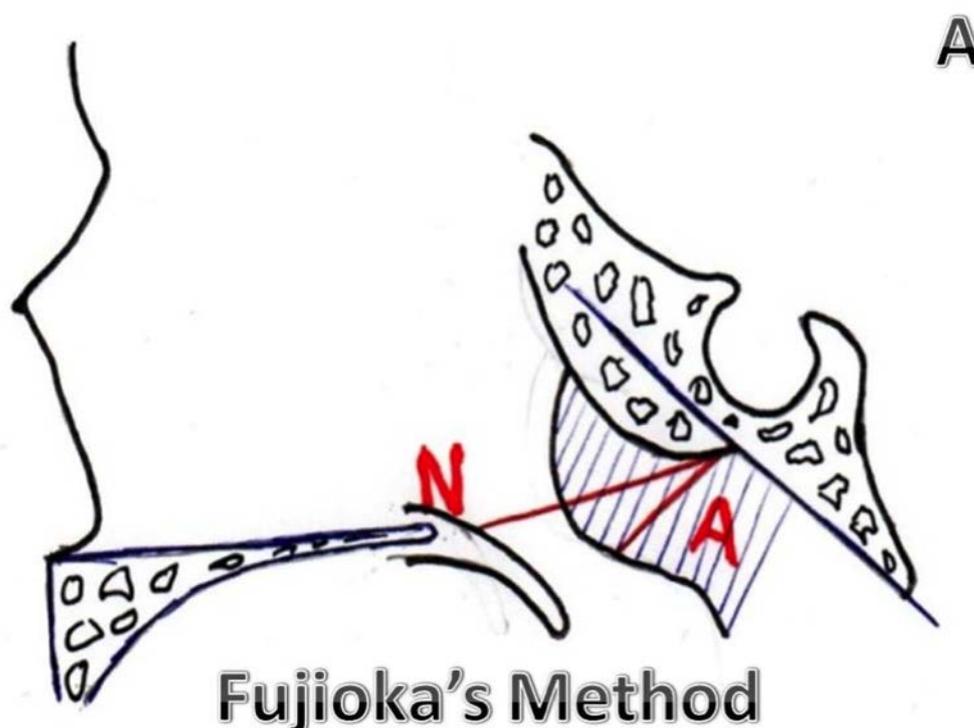
The patients with a diagnosis of adenoid hypertrophy who were candidates for surgery with a 6-weeks follow-up saline solution (3-5 drops, three times/day) treatment were included in the study as control group.

After taking a detailed medical history, the symptoms were graded according to the severity of patient's clinic. Pre-treatment a lateral cephalometric X-ray graph and a nasopharyngoscopic image ("MSI Flexible Nasopharyngoscope, Germany" attached to "Karl-Storz Telecam SL II, Tuttlingen, Germany" camera) were obtained from every patient. The hypertrophy of adenoid tissue was graded. Follow up examinations were based on nasopharyngoscopic records. Nasopharyngoscopic examination records were repeated after medical treatment. The difference between pretreatment and post treatment adenoid tissue enlargement were compared.

### Diagnostic Criteria

#### Symptoms

Nasal obstruction, snoring, and nasal discharge



**Figure A.** Adeno/nasopharyngeal ratio according to the Fujioka method.

were graded according to the frequency reported by the parents (Grade 0: never seen, Grade 1: seen during URTI, Grade 2: frequently seen, Grade 3: always occurs).

### Nasopharyngeal X-ray

Adenoid tissue enlargement was graded according to the Adenoidal-nasopharyngeal ratios (ANR). The distance of adenoid tissue density was measured along a line dropped perpendicularly from point of maximal convexity of adenoid tissue to its point of intersection with line drawn along the straight part of the anterior margin of the basiocciput. Then the nasopharyngeal space was measured as the distance between posterior superior edge of hard palate and the anterior inferior edge of the sphenoid basioccipital synchondrosis. The ANR was obtained by dividing the measurement for adenoid tissue density by the value for nasopharyngeal space in millimeters as described by Fujioka *et al.* [19]. It was rated as: Grade 1: > 6 mm, Grade 2: 4-6 mm, and Grade 3: < 3 mm. Nasal endoscopy: Performed with a Flexible Nasopharyngoscope following topical anesthesia with 4% lidocaine without any decongestant. Graded according to the rate of obstruction of the choanal aperture by the adenoid tissue as Grade 1: 25%, Grade 2: 50%, Grade 3: 75%, and Grade 4: 100% occlusion. Adeno/nasopharyngeal ratio according to the Fujioka method has been indicated in Figure A.

The children with a diagnosis of adenoidal hypertrophy were then prescribed topical mometasone furoate for 6 weeks, once a day, two puffs to each nostril (50 mcg/puff), comprising a daily total dose of 200 mcg. The nasal endoscopic evaluations were repeated 6 weeks after the initial diagnostic workup.

Operation was not recommended to the control and study groups with grade 2 adenoid hypertrophy, however medical treatment was given.

### Statistical Analysis

The data were analyzed with the SPSS for Windows v.16.0 software by IBM, USA, using the appropriate nonparametric tests for nominal and ordinal data. In all analytical evaluations,  $p < 0.05$  was the significance limit value.

## RESULTS

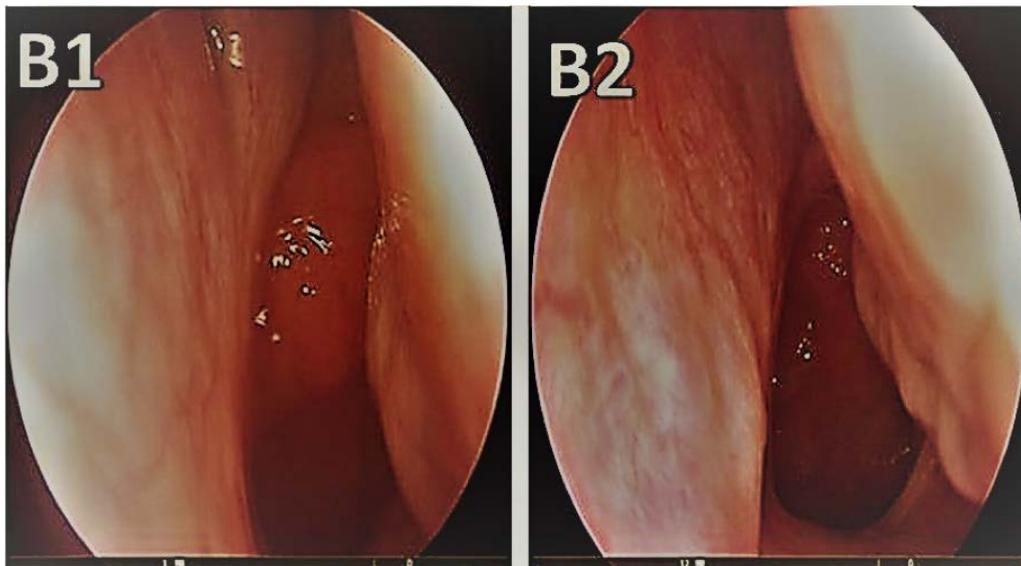
The study consisted of 75 patients (42 male, 33 female) with adenoid hypertrophy aged between 6 to 12 years old. Demographic characteristics and Body Mass Index (BMI) and Percentiles of the study group and the control group have been indicated in Table 1. Fifty-five patients (30 male, 25 female) who had topical nasal steroid accepted as study group and 20 patients (12 male, 8 female) who had preoperative follow up record for 6-weeks were accepted as control group. Endoscopic appearances before and after topical steroid treatment were indicated in Figures B, C and D.

The study group consisting of 30 males and 25 females was administered topical nasal Mometasone Furoate steroid treatment. Nasopharyngeal X-rays before treatment revealed that 25 patients were Grade 2 and 30 patients were Grade 3 grade according to the Fujioka method. Flexible endoscopy performed before the treatment revealed that 20 patients were Grade 2, 11 patients were Grade 3 and 24 patients were Grade 4. Nasal endoscopies performed after 6 weeks of

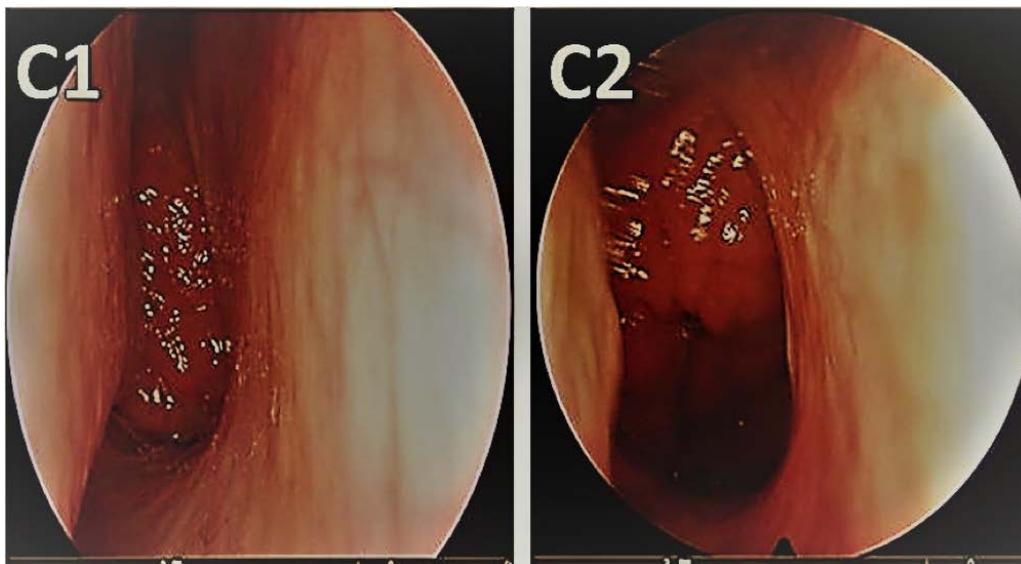
**Table 1.** Demographic characteristics and BMI and percentiles of the study group and the control group

	Study Group (n = 55)	Control Group (n = 20)
Age (year)	7.92 ± 1.81 (6-12)	7.80 ± 1.36 (6-11)
Gender (M/F)	30/25	12/08
Height (cm)	125.47 ± 11.30	123.15 ± 6.89
Weight (kg)	28.72 ± 7.09	27.55 ± 5.88
BMI	17.94 ± 1.58	17.95 ± 1.94
Percentile	77.89 ± 16.88	78.95 ± 13.83
BMI as a multiple of the mean	1.11 ± 0.07	1.10 ± 0.08
BMI		

BMI = Body Mass Index, F = Female, M = Male



**Figure B.** Endoscopic appearance before (B1) and after (B2) topical steroid treatment.



**Figure C.** Endoscopic appearance before (C1) and after (C2) topical steroid treatment.

intranasal topical steroid therapy revealed that 45 patients were Grade 1 and 10 patients were Grade 2. Nasopharyngeal X-rays were not requested for follow-up to avoid the additional harmful effects of X-rays. A statistically significant difference was present between endoscopic grades before and after treatment ( $p < 0.0001$ ) (Table 2). Non-severe epistaxis was observed in three patients who administered topical steroid therapy.

The 20 patients in the control group where saline solution (0.9 % NaCl) treatment was administered consisted of 12 males and 8 females. Nine patients was

Grade 2 and Grade 3 in 11 patients on nasopharyngeal X-rays at first presentation according to the Fujioka method in this group. Flexible nasal endoscopy performed simultaneously revealed that there were Grade 2 in 6 patients, Grade 3 in 9 patients and Grade 4 in 5 patients. Nasal endoscopies performed after 6 weeks in control group receiving saline solution treatment revealed Grade 2 in 7 patients, Grade 3 in 10 patients and Grade 4 in 3 patients. There was no statistically significant difference between in the prior and later grades of the control group ( $p = 0.3125$ ) (Table 2).



**Figure D.** Endoscopic appearance before (D1) and after (D2) topical steroid treatment.

**Table 2.** First and second endoscopic grades in the study group and in the control group

Grade n (%)	Study (n = 55)	Study (n = 55)	Control (n = 20)	Control (n = 20)
	First	Second	First	Second
Grade 1	0 (0%)	45 (81%)	0 (0%)	0 (0%)
Grade 2	20 (36%)	10 (18%)	6 (30%)	7 (35%)
Grade 3	11 (20%)	0 (0%)	9 (45%)	10 (50%)
Grade 4	24 (43%)	0 (0%)	5 (25%)	3 (15%)
Endoscopic grade (mean $\pm$ SD)	3.07 $\pm$ 0.89	1.18 $\pm$ 0.38	2.95 $\pm$ 0.75	2.80 $\pm$ 0.69

\*Wilcoxon test (paired samples).

## DISCUSSION

Demain and Goetz [20] used beclomethasone nasal spray for 8 weeks (338 microgm/day) followed by a lower daily dose (168 microgm/day) in the next 16 weeks for the treatment of adenoid hypertrophy and reported a decrease in adenoid size in all the study subjects. A history of atopy was reported in previous studies but they excluded those with such a history in this study [20]. We treated 55 children between the ages of 6 and 12 years with mometasone furoate nasal spray at a daily dose of 200 mcg for 4 weeks [20]. Lepcha *et al.* [21] reported no significant improvement in X-ray and endoscopy findings of subjects administered intranasal topical beclomethasone for 8 weeks while there was a 6% improvement in nasal congestion. However, there was an improvement in adenoidal obstruction and clinical findings when compared with placebo after 24 weeks of treatment [21]. In our patients, we also observed a decrease in the endoscopic findings of adenoid tissues.

Kheirandish-Gozal and Gozal [4] showed intranasal budesonide to decrease the severity of respiratory distress and the size of the adenoids, although mildly, with 6 weeks of use in children with mild OSA. Brouillette *et al.* [17] observed an improvement in the respiration of patients despite no noticeable change in the adenoid tissue mass after 6 weeks of treatment with intranasal fluticasone before T&A surgery in patients with moderate-to-heavy OSA. No significant change was found in the size of adenoids and tonsils and in the symptoms as reported by the parents but there was a significant reduction in apnea and hypopnea frequency among children treated with fluticasone.

Similar positive effects in OSA patients were reported in later studies. It is interesting that the effect on OSA symptoms continued even after the treatment was discontinued during 9 months of follow-up in the Alexopoulos *et al.* [18] study on 27 patients. The general results regarding OSA severity are strikingly similar despite differences in patient selection criteria

and treatment methods, indicating that nasal corticosteroids can be used as the first treatment option in pediatric OSA. However, there is no clear consensus on the appropriate steroid dosage and treatment duration at present [16, 22]. Junk *et al.* [23] have reported a significant improvement in loud snoring, breath holding, frequent awakening from sleep, breathing from the mouth, URTI frequency and nasal discharge after 4 weeks of intranasal mometasone furoate treatment in children. We also chose mometasone furoate among the various steroid nasal sprays commercially available for this study. The reason was the absence of reports on any negative effect of this spray on the nasal mucosa or side effects related to the hypothalamus-pituitary-adrenal axis and growth with long-term use [24]. The systemic effect of the drug is lower than other steroids after topical administration [25]. Berlucchi *et al.* [26] recently evaluated the effectiveness of 40 days of mometasone furoate treatment in adenoid hypertrophy and found symptomatic improvement in 77.7% of the patients. Fujioka *et al.* [19] described the A/N ratio as an indicator of adenoid hypertrophy in 1979 and the method has been used in many studies. Jung *et al.* [23] showed that the A/N ratio on lateral neck graphs decreased in 22 (71%) of 31 children after 4 weeks of treatment ( $p = 0.006$ ). Cengel and Akyol [27] reported shrinkage of adenoid tissue in 67.2% of the children after 6 weeks of treatment with intranasal mometasone furoate treatment in 2006. However, there is no proven mechanism explaining adenoid shrinkage. The presence of inflammation in the soft palate mucosal surface has been shown in OSA patients [28]. Jung *et al.* [23] thought that this type of upper respiratory tract inflammation could also involve the adenoid mucosa and that 4 weeks of local steroids could decrease this inflammation, causing the adenoids to shrink.

There are several methods to evaluate the size of the adenoids causing sleep-disordered breathing. The most commonly used techniques are lateral neck radiograph and direct videorhinoscopy. Mlynarek *et al.* [29] reported video rhinoscopy to be more useful for the evaluation of symptom severity in 2004. However, the nasopharyngeal examination of small children with fiberoptic devices can be difficult. The above results indicate that intranasal steroids can be used to reduce symptoms in children with sleep-disordered breathing, regardless of allergy or sinusitis.

The results of our study were also consistent with the literature. However, a limitation of our study is the lack nasal airway patency evaluation with objective methods such as acoustic rhinometry.

## CONCLUSION

We believe that the use of intranasal steroids (mometasone furoate) for 6 weeks in patients with pediatric chronic nasal obstruction due to adenoid hypertrophy may be an effective treatment modality in alleviating symptoms and decreasing adenoid volume without causing systemic side effects. Placebo-controlled studies are required to investigate the long-term effect of short-term steroid use in the treatment of pediatric sleep disorders in the future.

### *Conflict of interest*

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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The authors disclosed that they did not receive any grant during conduction or writing of this study.

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