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The Role of Mometasone Furoate Aqueous Nasal Spray in the Treatment of Adenoidal Hypertrophy in the Pediatric Age Group: Preliminary Results of a Prospective, Randomized Study

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ABSTRACT

OBJECTIVE. We evaluated the efficacy of mometasone furoate aqueous nasal spray in decreasing adenoid size and reducing the severity of chronic nasal obstruction symptoms in children affected by adenoidal hypertrophy.

METHODS. Sixty children were recruited in a 2-stage, randomized, placebo-controlled trial. All patients complained of chronic nasal obstruction symptoms, and nasal endoscopy showed >75% choanal obstruction attributable to adenoid pads. In the first stage, 30 patients (group A) underwent mometasone treatment (50 μ g per nostril per day) for 40 days, and 30 children (group B) received placebo. In the second stage, at the end of the first 40-day treatment period, patients in group A who showed subjective and objective clinical improvement were divided into 2 subgroups; group A1 (11 children) received topical intranasal steroid treatment on alternate days for the first 2 weeks per month, whereas group A2 (10 children) continued daily mometasone treatment for the first 2 weeks per month. After 3 months, all children were reassessed.

RESULTS. Fifty-seven children completed the study according to the protocol. After the first treatment period, the severity of symptoms and adenoid size decreased for 21 patients (77.7%) in group A. No improvement was observed in the placebo group. After 3 months of additional therapy, group A2 patients demonstrated a more-pronounced reduction in adenoid size compared with group A1 patients. No statistically significant change in symptoms was identified. Mometasone treatment was well tolerated by all patients.

CONCLUSIONS. Mometasone furoate aqueous nasal spray may be considered useful in decreasing adenoid pad size and the severity of symptoms related to adenoidal hypertrophy. Children with adenoidal hypertrophy that is not associated with tonsillar hypertrophy should be considered for intranasal mometasone treatment before surgery is planned.

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Key Words

adenoidal hypertrophy, adenoid pad, topical steroids, nasal steroids, mometasone furoate

Abbreviations

AH—adenoidal hypertrophy
OSAS—obstructive sleep apnea syndrome
MF—mometasone furoate

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THE ADENOIDS (PHARYNGEAL or Luschka's tonsil) are a single, pyramid-shaped aggregation of lymphoid tissue with the apex pointed toward the nasal septum and the base at the level of the roof and posterior wall of the nasopharynx. Present at birth, this lymphoid structure undergoes hypertrophy until 7 years of age, usually reaching a maximal size around the age of 4 years; later, it begins to atrophy until it almost invariably disappears in adulthood. The pharyngeal tonsil is a part of Waldeyer's ring, which is the first anatomic immunocompetent formation, situated at the entrance of the aerodigestive tract and composed of the adenoid pad in addition to the palatine, tubal, and lingual tonsils.

Adenoidal hypertrophy (AH), a common disorder in the pediatric population, presents with several signs and symptoms, ranging from nasal obstruction to obstructive sleep apnea syndrome (OSAS). Adenoidectomy is currently considered the treatment of choice for children with severe symptoms caused by AH, although more-conservative treatments using topical, intranasally administered, steroid preparations have been reported.¹⁻⁴ Herein we report the results of a prospective, randomized, placebo-controlled trial examining the effects of mometasone furoate (MF) aqueous nasal spray in the treatment of AH in pediatric patients.

METHODS

Study Subjects

Between February and July 2004, 60 children affected by AH and referred for exclusive adenoidectomy were recruited at the Department of Pediatric Otorhinolaryngology, Spedali Civili of Brescia (Brescia, Italy). At enrollment, patients needed to meet the following inclusion criteria: (1) adenoid pad occluding $\geq 75\%$ of the nasopharynx, as determined with nasal endoscopy; (2) age between 3 and 7 years; (3) symptoms consistent with AH lasting ≥ 12 months; and (4) no previous adenoidectomy. Children with concomitant tonsillar hypertrophy, positive history of allergy or atopy, upper respiratory infection within the past 2 weeks, nasal anatomic anomalies (eg, nasal septum deviation) or sinonasal diseases such as hypertrophy of inferior turbinates and/or nasal polyposis, craniofacial malformations including labiopalatal clefts, genetic diseases (eg, Down syndrome), neurologic disorders, cardiovascular diseases, immunodeficiency, history of epistaxis, hypersensitivity to steroids, or intranasal, topical, or systemic steroid or antibiotic treatment within the past 4 weeks were excluded.

Study Design

The design of the study and the informed consent form for this 2-stage, prospective, placebo-controlled, randomized study were approved by the institutional review board of the Spedali Civili of Brescia (Brescia, Italy). Before initiation of treatment, informed consent for

participation in the study was obtained from the parents or legal guardian of each child recruited. In the first stage, after a primary clinical evaluation and nasal endoscopy (time 0), children were assigned randomly to receive a single intranasal administration in each nostril of either MF aqueous nasal spray (50 μg) (group A) or a placebo saline solution nasal spray (group B) for 40 days.

At the end of the first 40-day period (time 1), both groups were reassessed, to evaluate the efficacy of treatment. Patients in group A who showed improvements in clinical findings and decreases in adenoid pad size, such that adenoidectomy could be avoided, were considered responders. In the second stage, responders underwent "maintenance therapy" for 3 months and were divided randomly into 2 subgroups; patients in group A1 received intranasal MF treatment on alternate days for the first 2 weeks per month, whereas those in group A2 continued daily topical steroid treatment for the first 2 weeks per month. Nonresponders and placebo-treated patients, for whom neither subjective nor objective improvement was apparent, were scheduled for adenoidectomy. After 3 months (time 2), the 2 subgroups were reassessed.

Assessment and Patient Treatment

At baseline, each child underwent physical evaluation and nasal endoscopy; clinical history was obtained from the parents with a questionnaire. Patient history included age, gender, weight, history and family history of atopy or allergy, and use of drugs. Symptoms such as nasal obstruction, rhinorrhea, cough, snoring, and obstructive sleep apnea were also evaluated at recruitment and at each subsequent visit, by using a clinical scoring system ranging from 0 to 3 (0 = absent; 1 = occasional; 2 = frequent; 3 = daytime and nighttime symptoms). In this way, we assigned to each aforementioned symptom a score related to severity. All scores were summed to obtain an overall symptom score for each patient. During follow-up visits, parents reported on their children's symptoms and eventual adverse effects (eg, nasal bleeding).

Nasal endoscopy was performed to estimate adenoid pad size and to identify other sinonasal disorders. After local anesthesia administration and decongestion of the nasal mucosa for 10 minutes with cotton pledgets soaked in phenylephrine and mepivacaine hydrochloride, endoscopic examination was conducted by using a rigid (2.7-mm diameter) or flexible endoscope, according to the compliance of the child. All nasal endoscopies, which were performed when the patient was performing quiet nasal breathing, were recorded by using a Karl Storz camera (Karl Storz, Tuttlingen, Germany). Pictures of both choanae were transferred to a computer. On each picture, the degree of adenoid obstruction was estimated as a percentage of the posterior choanal area occupied by

adenoid tissue. Children underwent endoscopic evaluation at time 0, time 1, and time 2.

Compliance with drug administration was assessed biweekly in telephone interviews with parents. Moreover, the occurrence of any disease and related therapy were recorded; the use of systemic steroid therapy resulted in exclusion of the patient from the study.

Statistical Methods

Baseline characteristics are presented as median and interquartile range or percentile, according to the type of variables analyzed. Possible baseline imbalance between the 2 groups was analyzed by means of the Wilcoxon exact test or Fisher's exact test. The primary end point of the first stage (reduction of AH) and the effects of treatment on secondary end points (score variations for chronic nasal obstruction symptoms) were analyzed with the Kruskal test or Wilcoxon exact test. Prognostic factors for responsiveness were studied with logistic regression. Potential differences at the end of the second stage were evaluated with the Kruskal test or Wilcoxon exact test. A Bonferroni correction for multiple comparisons was also applied. A statistical value of $P \leq .05$ was considered significant in all comparisons.

RESULTS

Sixty children were enrolled in the study and during the first stage were assigned randomly to receive MF (group A; $n = 30$, 18 male subjects and 12 female subjects; median age: 5 years) or placebo (group B; $n = 30$, 13 male subjects and 17 female subjects; median age: 4 years). No patient had a personal or family history of allergy or atopy, had undergone previous surgery, had received any drugs in the past 4 weeks, or had immunodeficiencies.

All children in group B and 27 patients in group A completed the study. Among the 3 patients in the latter group who did not complete the study, 1 was withdrawn for having received systemic steroid therapy for acute ethmoiditis, 1 was lost to follow-up monitoring, and 1 dropped out of the study.

At time 0, there were no significant differences between study groups with regard to demographic features or symptoms such as rhinorrhea, cough, and snoring, whereas nasal obstruction and obstructive sleep apnea were significantly more severe in group A. The mean overall symptom scores were 11 for group A (range: 6–15) and 10 for group B (range: 4–15). Nasal endoscopy was well tolerated by all children. No complication was observed during the initial diagnostic evaluation or after nasal endoscopies. Neither anatomic sinonasal abnormalities nor inflammatory changes of nasal mucosa except for AH were found with rhinoscopy. At recruitment, the mean choanal obstruction was 88.5% in group A (range: 75%–100%) (Fig 1) and 76.5% in group B (range: 75%–100%) (Table 1).



FIGURE 1
Endoscopic view of AH occluding 90% of the nasopharynx before MF treatment.

After the first treatment period (time 1), 21 (77.7%) of 27 patients who received MF were classified as responders, whereas 6 (22.3%) of 27 were nonresponders. Symptoms attributable to chronic nasal obstruction improved significantly in group A (mean overall symptom score: 3), whereas they were substantially unchanged in group B (mean overall symptom score: 9). The adenoid size also decreased significantly (Fig 2) in patients given MF (mean choanal obstruction: 64%) (Table 2). Only 1 patient in the steroid group reported episodic epistaxis, which resolved spontaneously without treatment. No other adverse effects were reported. At time 0, the only statistically significant difference between responders and nonresponders was related to obstructive sleep apnea (Table 3).

In the second stage, 11 of 21 responders were assigned randomly to group A1 and 10 to group A2. All children completed this stage of the study and, after 3 months of maintenance therapy, no significant differences in symptom scores were observed between the 2 subgroups, whereas the mean choanal obstruction in group A2 was less than that in group A1 (56% vs 65.5%; $P < .001$). Compliance with therapy during the last 3-month period was satisfactory in both subarms, and no adverse effects were observed.

DISCUSSION

AH is probably the most frequent pathologic condition occurring in the pediatric age group. It leads to different clinical manifestations according to adenoid size. Bilat-

TABLE 1 Baseline Demographic, Symptom, and Choanal Obstruction Data

	Group A (N = 27)	Group B (N = 30)	P
Age, median (interquartile range), y	5.0 (3.5–5.0)	4.0 (3.0–5.0)	.388
Male, n	16	13	.23
Weight, median (interquartile range), kg	18 (16–20)	19 (15–20)	.975
Nasal obstruction score, median (interquartile range)	3.0 (2.5–3.0)	2.0 (2.0–3.0)	.0202
Rhinorrhea score, median (interquartile range)	2 (1–3)	2 (1–2)	.468
Obstructive sleep apnea score, median (interquartile range)	1 (0–2)	0 (0–1)	.0113
Cough score, median (interquartile range)	1.00 (1.00–3.00)	1.00 (0.25–2.00)	.0973
Snoring score, median (interquartile range)	3.0 (2.0–3.0)	2.0 (1.3–3.0)	.350
Choanal obstruction, median (interquartile range), %	90 (85–90)	80 (75–90)	.13

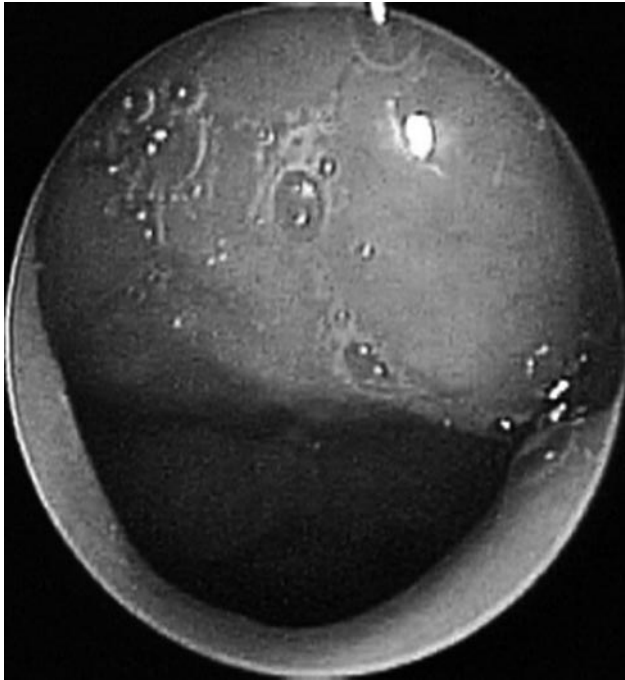


FIGURE 2
Endoscopic view of residual adenoid pad obstructing 50% of the left choana after intranasal steroid therapy.

eral nasal obstruction is a primary complaint that can be associated with different sleep disorders, ranging from snoring to OSAS.⁵ In such a situation, which is often observed when palatine tonsillar hypertrophy is also present, patients typically complain of both nighttime and daytime behavioral illnesses (ie, intermittent sleep, sleepwalking, morning headaches, difficulty concentrating, sleepiness, enuresis, slow feeding, and poor growth), which may lead to cardiorespiratory syndromes such as cor pulmonale in extreme cases. Rhinorrhea, mouth-breathing, hyponasal speech, and cough can also be observed in patients with AH. Moreover, nasal blockage and auricular secretions attributable to the adenoid pad relationship with nasal fossae and eustachian tubes may be present and represent a source of infection. In addition, AH seems to favor the occurrence of recurrent and effusive otitis media and recurrent and chronic rhinosinusitis.

Finger palpation, transoral mirror adenoid examination, and baseline lateral soft-tissue radiographs of the nasopharynx commonly have been used to assess adenoid size. However, these techniques are now considered inaccurate,⁶ and nasal endoscopy has been established as the method of choice for assessment of AH.^{6–9} In recent decades, technologic advances have led to the development of small-diameter (2.7-mm), flexible and rigid endoscopes that allow accurate nasal endoscopic examinations with no complications. As observed in the present study, rhinoscopy performed after positioning of cotton nasal pledgets soaked in topical anesthetic can be considered a thorough, safe, well-tolerated, reproducible procedure for assessment of adenoid pads. Furthermore, it provides information about the nasal mucosa, nasopharyngeal secretions, and the presence of other sinonasal anatomic abnormalities or diseases.

At present, AH is one of the most frequent indications for surgery in childhood, and adenoidectomy commonly is considered definitive treatment for nasopharyngeal obstruction. Nevertheless, this surgical technique has been the subject of some criticism. Paulussen et al¹⁰ hypothesized that the removal of adenoid lymphatic tissue could have a negative impact on the systemic immunologic system. Moreover, immediate postoperative or late bleeding is observed in ~1% of children who undergo adenoidectomy. Furthermore, it is well demonstrated that adenoids may recur after surgery in 10% to 20% of cases.^{11,12}

In the past decade, several authors have proposed the use of topical nasal steroids to decrease AH, with the intent to preserve immunologically active tissue and to avoid the anesthesiologic and surgical risks inherent in adenoidectomy.^{1–4} In 1995, Demain and Goetz¹ described an adenoid reduction using an aqueous nasal beclomethasone solution for 17 children with AH. All patients complained of classic symptoms of chronic nasal obstruction and had an estimated $\geq 90\%$ adenoid obstruction of the choanae, as determined with primary nasal endoscopy. In that study, patients were divided randomly into 2 groups; the first group received 4 weeks of intranasal aqueous beclomethasone (336 $\mu\text{g}/\text{day}$) nasal spray treatment, followed by 4 weeks of placebo

TABLE 2 Chronic Nasal Symptoms and Choanal Obstruction After the First 40 Days of Treatment

	Median (Interquartile Range)		P
	Group A (N = 27)	Group B (N = 30)	
Nasal obstruction score	1.000 (0.000–2.000)	0.000 (0.000–0.000)	.00005
Rhinorrhea score	1.00 (0.00–1.50)	0.00 (0.00–1.00)	.041
Obstructive sleep apnea score	0.000 (0.000–1.000)	0.000 (0.000–0.000)	.00034
Cough score	1.00 (0.00–2.50)	0.00 (0.00–1.00)	.0033
Snoring score	2.00 (0.00–2.00)	0.00 (0.00–0.00)	.00000005
Choanal obstruction, %	20.0 (12.5–32.5)	0.0 (0.0–0.0)	.007

TABLE 3 Demographic, Symptom, and Choanal Obstruction Data for Nonresponders Versus Responders at Time 0

	Nonresponders (N = 6)	Responders (N = 21)	P
Age, median (interquartile range), y	4.0 (3.3–4.8)	5.0 (4.0–5.0)	.282
Male, n	3	13	.601
Weight, median (interquartile range), kg	17 (14–20)	18 (16–20)	.491
Nasal obstruction score, median (interquartile range)	3 (3–3)	3 (2–3)	.108
Rhinorrhea score, median (interquartile range)	3.0 (1.5–3.0)	2.0 (1.0–2.0)	.103
Obstructive sleep apnea score, median (interquartile range)	2 (2–2)	0 (0–2)	.0361
Cough score, median (interquartile range)	2.5 (1.3–3.0)	1.0 (0.0–3.0)	.217
Snoring score, median (interquartile range)	3 (3–3)	3 (2–3)	.288
Choanal obstruction, median (interquartile range), %	90 (86–90)	90 (85–95)	.633

treatment, whereas the second group underwent the same treatments in the reverse order. After the first 8-week period, all children continued topical nasal administration of beclomethasone (168 $\mu\text{g}/\text{day}$) for 4 months. At the end of the study, significant improvements in adenoid obstruction and symptoms were observed for all patients. Of the 17 subjects, however, 7 had a clinical history of atopy (asthma, allergic rhinitis, or atopic dermatitis) and 12 had a family history of atopy. In our opinion, the characteristics of those patients could have influenced the final outcomes.

In 2001, Brouillette et al² tested the efficacy of another intranasal steroid treatment for OSAS in a randomized, triple-blind, placebo-controlled, parallel-group trial investigating the use of fluticasone propionate nasal spray versus placebo for 25 children affected by OSAS, as demonstrated with polysomnography. Thirteen of 25 patients underwent topical intranasal fluticasone therapy (50 μg of active drug) with 1 spray per nostril twice daily for the first 7 days and then once daily for an additional 5 weeks. The remaining 12 children received placebo. After treatment, the mixed/obstructive apnea/hypopnea index, frequency of hemoglobin desaturation, and respiratory movement/arousals decreased more in the fluticasone-treated group, compared with the placebo-treated group. Moreover, improvements in symptom scores were observed for 69% of children who received fluticasone.

In 2003, Criscuoli et al,³ studying 53 children, reinforced the conclusions reached by Demain and Goetz.¹ For the first time, they reported on the long-term outcomes of treatment with aqueous nasal beclomethasone

for patients with adenotonsillar hypertrophy. Twenty-four patients exhibited improvement after 2 weeks of steroid treatment, and an additional 24 weeks of therapy at a lower steroid dose maintained clinical improvement at 52 and 100 weeks for 45.8% of those patients.

Recently, Cengel and Akyol⁴ assessed the efficacy of MF in the treatment of AH, in a prospective, controlled, randomized, clinical trial. Of 122 patients enrolled, 67 received intranasal MF (100 $\mu\text{g}/\text{day}$) therapy for 6 weeks, whereas 55 patients were assigned to the control group. After treatment, a significant decrease of the adenoid mass was observed for 67.2% of the study group, whereas the clinical situation was unchanged in the control group. It is noteworthy that 8.9% of MF-treated patients had a positive history and prick-test results for atopy.

Among several commercially available steroid nasal sprays (beclomethasone dipropionate, budesonide, flunisolide, fluticasone propionate, MF, and triamcinolone acetonide), we chose to test MF for 4 reasons, namely, (1) the drug had been reported previously not to cause any adverse tissue changes in the nasal mucosa of patients treated for long periods,¹³ (2) it has no effects on growth in children,¹⁴ (3) it has no effects on the hypothalamic-pituitary-adrenal axis,¹⁵ and (4) the systemic availability of the drug after topical administration is lower than that of other steroids.¹⁶ To date, no standard indications regarding dosage and duration of topical intranasal steroid therapy for the treatment of AH have been established. Compared with the aforementioned trials, we chose to administer a lower daily steroid dose in each nostril but for a longer time (first treatment

period: 40 days). A single low dose of intranasally administered steroid was well accepted by parents/legal guardians and children, thereby increasing compliance. Moreover, only 1 complication (episodic epistaxis) was observed, which demonstrates the safety of intranasal MF administration.

Although group A had more-severe clinical findings than group B at time 0, 77.7% of children treated with MF showed significant improvements in symptoms and significant reductions in adenoid size after the first 40 days of treatment, thus avoiding adenoidectomy. Interestingly, among the analyzed factors (ie, age, gender, weight, symptoms, and choanal obstruction), only obstructive sleep apnea was statistically significant in discriminating between responders and nonresponders. However, it is difficult to attribute the true value to these results because of the small number of patients studied.

Although at our knowledge there are no publications in the literature supporting or criticizing a low topical steroid dosage for the treatment of AH, we modified the treatment schedule after the first treatment period, with the intent to maintain the outcomes while decreasing the dosage of the intranasal MF aqueous nasal spray further. Because we could not modify the steroid dose delivered in each puff, we decided to modify the duration of treatment, thus increasing the compliance of the children and their families. For this reason, responders were divided into 2 subgroups and underwent 2 different maintenance therapies. At the end of the second stage of treatment, group A2 showed significant reduction of AH, compared with group A1, whereas symptoms were comparable. Therefore, daily administration of MF for 2 weeks per month, in addition to maintaining successful clinical results, seems to decrease AH further.

It is critical to highlight that we obtained these successful results for children with only AH. By obstructing the postnasal space, adenoids prevent steroids from acting on the palatine tonsils. In the study by Demain and Goetz,¹ no evident tonsillar changes were observed for 7 children who showed, besides AH, moderate tonsillar hypertrophy. Because the steroid nasal spray acts especially in the nasal fossa and nasopharynx, we tested the effects of intranasal MF therapy on patients affected exclusively by AH, with no tonsillar hypertrophy. Several mechanisms, such as direct lympholytic action, inhibition of inflammation, and alteration of adenoid bacterial flora, have been suggested to explain how steroids decrease adenoid pad volume and improve symptoms of AH, although none has yet achieved widespread acceptance.^{1,17}

CONCLUSIONS

We report a prospective, randomized, placebo-controlled trial on the efficacy of MF aqueous nasal spray versus placebo for the treatment of AH in children. Topical intranasal MF therapy can be considered a good therapeutic option to decrease AH. Nasal administration of

this steroid is safe, reproducible, easily performed, and well tolerated by pediatric patients. Daily use for 2 weeks per month after an initial 40-day-period treatment seems to be the ideal maintenance schedule. Obviously, the indications for adenoidectomy remain unchanged for nonresponders. Future studies addressing the long-term (>1-year) efficacy of MF therapy and the identification of factors that could be used to select nonresponders are warranted.

REFERENCES

1. Demain JG, Goetz DW. Pediatric adenoidal hypertrophy and nasal airway obstruction: reduction with aqueous nasal beclomethasone. *Pediatrics*. 1995;95:355-364
2. Brouillette RT, Manoukian JJ, Ducharme FM, et al. Efficacy of fluticasone nasal spray for pediatric obstructive sleep apnea. *J Pediatr*. 2001;138:838-844
3. Criscuoli G, D'Amora S, Ripa G, et al. Frequency of surgery among children who have adenotonsillar hypertrophy and improve after treatment with nasal beclomethasone. *Pediatrics*. 2003;111:236-238
4. Cengel S, Akyol MU. The role of topical nasal steroids in the treatment of children with otitis media with effusion and/or adenoid hypertrophy. *Int J Pediatr Otorhinolaryngol*. 2006;70:639-645
5. Craig TJ, Teets S, Lehman EB, Chinchilli VM, Zwillich C. Nasal congestion secondary to allergic rhinitis as a cause of sleep disturbance and daytime fatigue and the response to topical nasal corticosteroids. *J Allergy Clin Immunol*. 1998;101:633-637
6. Chisholm EJ, Lew-Gor S, Hajioff D, Caulfield H. Adenoid size assessment: a comparison of palpation, nasendoscopy and mirror examination. *Clin Otolaryngol*. 2005;30:39-41
7. Wang DY, Clement P, Kaufman L, Derde MP. Fiberoptic examination of the nasal cavity and nasopharynx in children. *Acta Otorhinolaryngol Belg*. 1991;45:323-329
8. Kubba K, Bingham BJG. Endoscopy in the assessment of children with nasal obstruction. *J Laryngol Otol*. 2001;115:380-384
9. Cassano P, Gelardi M, Cassano M, Fiorella MC, Fiorella R. Adenoid tissue rhinopharyngeal obstruction grading based on fibroendoscopic findings: a novel approach to therapeutic management. *Int J Pediatr Otorhinolaryngol*. 2003;61:1303-1309
10. Paulussen C, Claes J, Claes G, Jorissen M. Adenoids and tonsils, indications for surgery and immunological consequences of surgery. *Acta Otorhinolaryngol Belg*. 2000;54:403-408
11. Kovaleva LM. Repeated adenoidectomy and prevention of the recurrence of adenoid hypertrophy [in Russian]. *Vestn Otorinolaryngol*. 1994;1:18-21
12. Buchinsky FJ, Lawry MA, Isaacson G. Do adenoids regrow after excision? *Otolaryngol Head Neck Surg*. 2000;123:576-581
13. Minshall E, Ghaffar O, Cameron L, O'Brein F, Quinn H, Rowe-Jones J. Assessment by nasal biopsy of long-term use of mometasone furoate aqueous nasal spray (Nasonex) in the treatment of perennial rhinitis. *Otolaryngol Head Neck Surg*. 1998;118:648-654
14. Schenkel EJ, Skoner DP, Bronsky EA, et al. Absence of growth retardation in children with perennial allergic rhinitis after one year of treatment with mometasone furoate aqueous nasal spray. *Pediatrics*. 2000;105(2). Available at: www.pediatrics.org/cgi/content/full/105/2/e22
15. Boner AL. Effects of intranasal corticosteroids on the hypothalamic-pituitary-adrenal axis in children. *J Allergy Clin Immunol*. 2001;108(suppl):S32-S39
16. Szeffler SJ. Pharmacokinetics of intranasal corticosteroids. *J Allergy Clin Immunol*. 2001;108(suppl):S26-S31
17. Barnes PJ, Pedersen S. Efficacy and safety of inhaled corticosteroids in asthma: report of a workshop held in Eze, France, October 1992. *Am Rev Respir Dis*. 1993;148:S1-S26

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