Absence of Growth Retardation in Children With Perennial Allergic Rhinitis After One Year of Treatment With Mometasone Furoate Aquous Nasal Spray

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ABSTRACT. Objective. Intranasal corticosteroids are used widely for the treatment of allergic rhinitis because they are effective and well tolerated. However, their potential to suppress growth of pediatric subjects with allergic rhinitis continues to be a concern, particularly in light of reports of growth suppression after treatment with intranasal beclomethasone dipropionate or intranasal budesonide (see the article by Skoner et al in this month’s issue). To establish the absence of growth suppression associated with intranasal corticosteroids, we conducted a 1-year study of prepubertal patients between 3 and 9 years of age with perennial allergic rhinitis to assess the effects on growth of mometasone furoate aqueous nasal spray (MFNS), a new once-daily (QD) intranasal corticosteroid with negligible bioavailability.

Methods. This was a randomized, placebo-controlled, double-blind, multicenter study. Ninety-eight subjects were randomized to treatment with either MFNS 100 µg QD or placebo for 1 year. Each subject’s height was required to be between the 5th and 95th percentile at baseline, and skeletal age at screening was required to be within 2 years of chronological age, as determined by left wrist x-rays. Washout periods for medications that affect either childhood growth or allergic rhinitis symptoms were established based on estimated period of effect, and these medications were prohibited during the study. However, short courses of either oral prednisone lasting no longer than 7 days or low-potency topical dermatologic corticosteroids lasting no longer than 10 days were permitted if necessary.

Height was measured with a calibrated stadiometer at baseline and at 4, 8, 12, 26, 39, and 52 weeks, and the primary safety variable was the change in standing height from baseline using data from all visits of subjects (intent-to-treat principle). The change from baseline in standing height was analyzed by a 2-way analysis of variance that extracted sources of variation attributable to treatment, center, and treatment-by-center interaction.

Results. Demographic characteristics were similar at baseline. Eighty-two subjects completed the study (42 in the MFNS group and 40 in the placebo group), and 93% of subjects achieved at least 80% compliance with therapy. After 1 year of treatment, no suppression of growth was seen in subjects treated with MFNS, and mean standing heights were similar for both treatment groups at all time points. For the primary safety variable (change in height from baseline), both treatment groups were similar at all time points except for weeks 8 and 52. Subjects treated with MFNS had a slightly greater mean increase in height than subjects treated with placebo at these time points: the change in height was 6.95 cm versus 6.35 cm at the 1-year time point. However, the rate of growth (.018 cm/day) averaged for all time points over the course of the study was similar for both treatment groups. Additional analyses found that MFNS did not retard growth in any sex or age subgroup of subjects. The use of exogenous corticosteroids other than the study drug was also similar among the 2 treatment groups.

Results from cosyntropin stimulation testing confirmed the absence of systemic effects of MFNS. The change from baseline in the difference between pre-stimulation and poststimulation levels was similar for both treatment groups after 1 year of treatment, with no evidence of HPA-axis suppression in MFNS-treated subjects at any time point. Incidences of treatment-related adverse events were similar for both treatment groups, with 16% of MFNS-treated subjects reporting adverse events, compared with 22% of placebo-treated subjects.

Conclusions. In summary, 1 year of treatment with MFNS 100 µg QD was found to be well tolerated, with no evidence of retardation of growth or suppression of HPA-axis function in perennial allergic rhinitis subjects as young as 3 years of age. These findings may be particularly relevant for children with co-morbid atopic disorders, such as asthma and eczema. Such patients may be treated concurrently with inhaled and/or dermatologic corticosteroids, thereby increasing the risk of systemic adverse events, including growth suppression. The absence of systemic adverse events found in this study, combined with the established efficacy and safety profile of MFNS in children, indicates that it may be an appro-
Allergic rhinitis is a common disease, affecting up to 10% of children and 20% of adolescents and adults. The use of intranasal corticosteroids as a first-line therapy for children with persistent disease has gained acceptance in recent years because they are well tolerated and effective for treating nasal symptoms of allergic rhinitis.

As intranasal corticosteroids become more widely used for treatment of allergic rhinitis, their potential to cause systemic adverse events continues to be an issue. This potential is of special consideration for perennial allergic rhinitis (PAR) patients who receive concomitant treatment with inhaled corticosteroids for asthma for extended periods of time. Systemically active corticosteroids have the potential to cause a wide range of adverse events, including osteoporosis, glaucoma, cataracts, adrenal suppression, and, of particular concern in children, impaired growth. Systemic corticosteroids have been found to inhibit growth through a variety of mechanisms, including inhibition of growth hormone secretion, interference with nitrogen and mineral retention, and inhibition of osteoblast function.

Few previous studies have investigated the effects of either short- or long-term treatment with intranasal corticosteroids on growth in children, and the studies that have been published demonstrate conflicting results. A recent 1-year study of 100 patients with PAR between 6 and 9.5 years of age found that treatment with beclometasone dipropionate (BDP) aqueous nasal spray 168 µg twice daily (QD) statistically significantly suppressed growth, compared with placebo. At the end of 1 year, the BDP-treated patients had grown 0.9 cm less than the placebo-treated patients. However, an uncontrolled study investigating the effects of budesonide (BUD) 200 µg QD (50 µg per spray, 1 spray per nostril) to be the optimal dose of MFNS in children 6 to 11 years of age with SAR and comparable in efficacy to BDP 84 µg QD. The systemic safety of MFNS in doses up to 200 µg per day in children has been studied by Brannan et al. who found no evidence of HPA-axis suppression. In addition, plasma levels of mometasone furoate were too low to be detected in >99% of samples; therefore, the bioavailability of MFNS in children could not be calculated. The current study was undertaken to assess the effects of MFNS over 1 year on growth in pediatric patients with PAR.

METHODS

This was a phase 3, randomized, placebo-controlled, double-blind, multicenter, 12-month safety study to assess pediatric growth during treatment with MFNS. The study was approved by an institutional review board, and patients and their parent or guardian were required to provide written informed consent at the screening visit. Ninety-eight patients of either sex and of any race between 3 and 9 years of age with PAR were enrolled in the study. A 1-year treatment period was chosen to avoid possible seasonal variations in growth rate.

Patients were required to be no greater than stage 1 on the Tanner Classification of Sexual Maturity. To recruit a patient population that would remain prepubescent throughout the study, boys were required to have been <9 years and 6 months of age at baseline and girls were required to have been <9 years of age at baseline and premenarchal. The skeletal ages of the patients at screening were required to be within 2 years of their chronological age, as determined by a radiograph of the left wrist. In
addition, patients were required to have had at least a 1-year history of PAR that required treatment within the past 12 months and have a positive skin response to the relevant perennial allergen.

Each patient’s height was required to be within the 5th and 95th percentiles as measured by a Harpenden stadiometer (model 602, Holtain, Ltd, London, UK) at baseline and at least 1 other time during the period between 3 and 24 months before screening. If no stadiometer height measurement had been taken during this preliminary period, 2 nonstadiometer height measurements were required. Height at screening was required to be no 10 percentiles different from the stadiometer measurement obtained in the previous 3 to 24 months, or no 15 percentiles different from the 2 nonstadiometer measures taken over the same period.

Patients were excluded if they had asthma that required the chronic use of inhaled or systemic corticosteroids, or if they had taken inhaled corticosteroids for asthma for >2 months within the 12 months before enrollment. (Patients with mild to moderate asthma were allowed to enroll, so long as their asthma was well-controlled without the use of corticosteroids.) Patients also were excluded if they had a history or presence of abnormal growth or gross malnutrition, a history of multiple drug allergies, allergy to corticosteroids, posterior subcapsular cataracts or nasal structural abnormalities (including large nasal polyps and marked septal deviation), an upper respiratory infection or sinus infection requiring antibiotic therapy during the 2 weeks before screening, or if they had a viral upper respiratory infection during the 7 days before screening.

Washout periods were established for medications that may either have an effect on childhood growth or on rhinitis symptoms, based on the medication’s duration of efficacy or estimated period of effect on growth. Before baseline, patients were required to have taken inhaled, oral, intravenous, or rectal corticosteroids for 1 month, intranasal or intraocular corticosteroids for 3 months, intranasal or ocular corticosteroids for 2 weeks, or high-potency dermatologic corticosteroids for 1 month. In addition, nasal Cromolyn sodium, nedocromil, antihistamines, decongestants, ketotifen, nasal atropine, ipratropium, desmopressin acetate, nasal saline, immunotherapy, and any investigational drugs were prohibited for a specified time period ranging from 24 hours to 6 months, depending on the medication.

A complete medical history was taken at screening. Patients were given a thorough physical examination including assessments of sexual maturity, vital signs, 12-lead electrocardiography, and clinical laboratory determinations (complete blood cell count, blood chemistry, and urinalysis) at screening and at weeks 26 and 52. Vital signs were measured at each visit. Cosyntropin stimulation testing was performed at 4 of the 10 centers at screening and at weeks 26 and 52.

Treatments

A 7- to 14-day diary run-in phase was conducted between the screening and baseline visits, during which patients recorded rhinitis symptom scores, concomitant medications, and adverse events BID. At baseline, patients meeting the entry criteria were instructed in the proper use of the nasal spray devices and were randomized to treatment with either MFNS 100 µg QD, administered intranasally 1 spray (50 µg) per nostril in the morning on awakening or placebo for 52 weeks.

Any concomitant medications taken within 30 days before screening were recorded. The study physician prescribed concomitant treatment for relief of intolerable symptoms of allergic rhinitis as necessary. Patients were encouraged not to use any other medications; however, the use of other nonsteroidal allergy preparations was allowed. Medications that were prohibited included systemic or other topical corticosteroids, nasal Cromolyn sodium, nedocromil, atropine or ipratropium, desmopressin acetate, cyproheptadine, and methylphenidate hydrochloride. Exceptions could be made if oral corticosteroids or low-potency dermatologic corticosteroids were required during the study. Up to 2 courses of oral prednisone lasting no >7 days were allowed, but there must have been an interval of at least 4 weeks between the last dose and cosyntropin-stimulation testing (where applicable). Low-potency topical dermatologic corticosteroids (≤1% hydrocortisone) were permitted as long as no individual treatment episode lasted >10 days and the overall period of treatment during the study was <4 weeks. Concomitant treatment with immunotherapy was allowed if the patient had been on a stable maintenance schedule for at least 1 month before screening. Patients recorded in diaries any medications taken during the study, and their use was reviewed with the investigator at each visit.

Compliance was evaluated by asking patients and their parent or guardian about medication usage, reviewing study cards, and weighing the spray bottles at day 8 and weeks 4, 8, 12, 26, 39, and 52. The bottles were weighed without the knowledge of the patient and parent or guardian.

Primary Safety Assessment

The primary safety variable was the change in standing height as measured by stadiometer at baseline and at weeks 4, 8, 12, 26, 39 and 52. Height was recorded by trained study staff while patients were barefoot with their heads in the neutral position, and it was attempted to have each patient’s measurements occur at the same time of day. To reduce variability and measurement error that could occur if a patient was not positioned properly on the stadiometer (eg, if the patient was slouching), the greatest of 3 measurements was recorded. The rate of growth was evaluated for each subject as the slope of the change in standing height from baseline over time, using all visits as points from which to construct the curve for all patients with data from at least 2 visits. Investigators also assessed the occurrence of adverse events and rated them for severity (mild, moderate, severe, or life-threaten-

<table>
<thead>
<tr>
<th>TABLE 1. Demographic Characteristics at Baseline</th>
<th>MFNS 100 µg QD (n = 49)</th>
<th>Placebo (n = 49)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex (male/female)</strong></td>
<td>31/18</td>
<td>35/14</td>
</tr>
<tr>
<td><strong>Asthma (yes/no)</strong></td>
<td>16/33</td>
<td>13/36</td>
</tr>
<tr>
<td><strong>Comorbid SAR (yes/no)</strong></td>
<td>39/10</td>
<td>36/13</td>
</tr>
<tr>
<td><strong>Mean age (y; range)</strong></td>
<td>6.3 (3–9)</td>
<td>6.4 (3–9)</td>
</tr>
<tr>
<td><strong>Age category (n)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3–5 y</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>6–9 y</td>
<td>30</td>
<td>29</td>
</tr>
<tr>
<td><strong>Mean body weight (lbs; range)</strong></td>
<td>54.5 (29–84)</td>
<td>55.2 (29–100)</td>
</tr>
<tr>
<td><strong>Mean height (cm)</strong></td>
<td>120.2</td>
<td>120.9</td>
</tr>
</tbody>
</table>

Fig 1. Mean change in standing height from baseline measured by stadiometer over 1 year of treatment with MFNS 100 µg QD or placebo.
ing) and with respect to their relationship to treatment at all study visits.

Secondary Assessment
Cosyntropin Stimulation

To further investigate possible systemic effects of long-term treatment with MFNS, cosyntropin (synthetic adrenocorticotropic hormone, Cortrosyn, Organon, Inc, West Orange, NJ) stimulation testing was conducted at 4 of the 10 designated study sites at screening and at weeks 26 and 52. At each of these visits, pre-stimulation plasma cortisol concentrations were measured at 8:00 AM. Patients who did not have a normal cortisol concentration of at least 5 μg/100 mL at screening were excluded from the study. Pre-stimulation measurements were followed by an intravenous injection of .25 mg cosyntropin dissolved in 1 mL sodium chloride (9% United States Pharmacopeia). Post-stimulation cortisol concentrations were measured 30 minutes after the injection. Patients who did not have a post-stimulation cortisol concentration of at least 18 μg/100 mL at screening also were excluded from the study.

Statistical Analysis

The safety and efficacy analyses were based on all randomized patients (intent-to-treat principle). Any subject with missing data for a parameter was not included in any analysis of that parameter at that visit. For the analysis of endpoint, those subjects only having baseline data or only postbaseline data were not included. The change from baseline in standing height was analyzed by a 2-way analysis of variance (ANOVA) that extracted sources of variation attributable to treatment, center, and treatment-by-center interaction. The treatment comparison was based on the least square mean from the ANOVA, using a 5% (2-sided) significance level. In addition, the growth rate for each parameter was defined as the slope of a linear regression of the change in height by time. These rates were also analyzed with the 2-way ANOVA noted above. Data from the plasma cosyntropin-stimulation were analyzed by the same 2-way ANOVA used for the analysis of growth. All treated patients (pooled across all centers) in whom the relevant height values were available were included in these analyses.

RESULTS

Demographics

Ninety-eight patients 3 through 9 years of age with a history of PAR were randomized to treatment; 49 patients received MFNS 100 μg QD and 49 patients received placebo. The overwhelming majority of patients (93%) were at least 80% compliant with therapy, based on recordings in patient daily diaries, and rates of compliance were similar in both treatment groups. The remainder of patients achieved at least 60% compliance. All statistical analyses were based on all treated patients (intent-to-treat principle).

There were no significant differences between the 2 groups at baseline with respect to age, sex, race, or body weight, and both groups were similar with regard to the presence or absence of asthma and/or SAR. The mean baseline height for patients in the MFNS treatment group was 120.2 cm, compared with 120.9 cm in the placebo-treatment group. This difference was not statistically significant (P = .61). In addition, both treatment groups had similar numbers of patients who were in the 3 to 5 years of age category and the 6 to 9 years of age category (see Table 1). Approximately 90% of study participants were white. No evidence of Tanner stage progression was noted for any patient at the physical examinations performed at weeks 26 and 52. The percentages of patients who had both their baseline and endpoint height measurements performed at the same time were similar in both treatment groups.

At the final treatment visit, 82 patients were still enrolled (42 in the MFNS group and 40 in the placebo group). Among the MFNS-treated patients, 1 patient discontinued because of adverse events, 2 patients discontinued because of treatment failure, and 4 patients discontinued for other reasons (eg, noncompliance or failure to meet eligibility criteria). Among the placebo-treated patients, 1 patient discontinued because of an adverse event, 1 patient withdrew because of treatment failure, and 7 patients withdrew for other reasons. In addition, among the 82 patients, 1 MFNS-treated patient dropped out because of an adverse event at the final visit, and 1 placebo-treated patient discontinued because of noncompliance at the final visit. Because data were available for these 2 patients, they were included in the statistical analyses. In a reanalysis without these 2 patients, results for standing height and growth velocity remained unaffected.

Use of exogenous corticosteroids other than the study drug during the treatment phase of the study was similar in both treatment groups. Among the MFNS-treated patients, 1 received an intranasal corticosteroid in addition to the study drug from day 85 throughout the rest of the study and 4 received courses of oral corticosteroids lasting 3 to 12 days. Similarly among the placebo-treated patients, 2 pa-

| Table 2. Mean Increase in Height Over One Year in Patient Sex and Age Subgroups |
|-----------------------------|-----------------------------|-----------------------------|
| Subgroup         | MFNS 100 μg QD | Placebo |
| Age 3–5 y        | 7.65 cm (n = 12) | 7.26 cm (n = 11) |
| Age 6–9 y        | 6.67 cm (n = 30) | 6.00 cm (n = 29) |
| Female           | 6.73 cm (n = 15) | 6.25 cm (n = 12) |
| Male             | 7.07 cm (n = 27) | 6.39 cm (n = 28) |

Fig 2. Plasma cortisol response to cosyntropin stimulation during 1 year of treatment with either MFNS 100 μg or placebo.
patients received an intranasal corticosteroid in addition to the study drug, 4 patients received courses of oral corticosteroids, and 1 patient received an inhaled corticosteroid for 2 weeks.

**Primary Safety Analyses**

The mean heights were similar for both treatment groups at all time points ($P \geq .20$). For the primary safety variable (change in standing height from baseline), both treatment groups were similar at all time points except for weeks 8 and 52. At these 2 time points, the mean increase in height from baseline was significantly greater in the MFNS-treatment group. At week 52, the mean increase in height was 6.95 cm in MFNS-treated patients compared with 6.35 cm in placebo treated patients ($P = .02$; see Fig 1).

To ensure that slight difference in height at baseline had no effect on the results, a 2-way analysis of covariance was performed, which adjusted for baseline standing height. This analysis also found that the adjusted mean increase in height from baseline in the MFNS-treated patients (mean adjusted by the analysis of covariance) was significantly greater (6.85 cm) than in the placebo-treated patients (6.35 cm; $P = .04$) at week 52. However, analysis of the rate of growth (calculated using all visits as data points to construct the curve) found no significant difference between the 2 treatment groups, based either on all patients treated (mean growth of .018 cm/day for both groups; $P = .80$) or on the subset of patients who completed the study (mean growth of .018 cm/day for placebo-treated patients and .019 cm/day for MFNS-treated patients; $P = .22$). Additional analyses found that MFNS did not retard growth in any specific sex or age subgroup of patients (see Table 2). There were no significant differences in growth within or between treatments, between the age categories, or between male and female subjects.

**Secondary Analyses**

**HPA-Axis Function**

Thirty-eight patients were enrolled in the 4 treatment centers that participated in the cosyntropin stimulation portion of the study (17 in the MFNS-treatment group and 21 in the placebo-treatment group). Of these patients, 14 receiving MFNS and 17 receiving placebo remained in the study until week 52.

All patients had a normal response to cosyntropin stimulation at all time points. There was no evidence of HPA-axis suppression in the MFNS-treatment group at any time point. At week 26, the response to cosyntropin stimulation (ie, the change from the prestimulation to poststimulation plasma cortisol concentration) was actually greater in the MFNS-treatment group than the placebo group ($P = .02$); however, the basal plasma cortisol concentrations were similar in the 2 treatment groups ($P = .38$). At week 52, both the basal plasma cortisol concentrations and the response to cosyntropin stimulation were similar in the 2 groups ($P = .75$ and $P = .51$, respectively). The difference between the MFNS- and placebo-treatment groups in the response to cosyntropin stimulation from screening to week 52 also was not significant ($P = .48$; see Fig 2 and Table 3).

**Adverse Events**

The overall incidence of treatment-related adverse events was similar for both treatment groups: treatment-related adverse events occurred in 8 (16%) MFNS-treated patients and in 11 (22%) placebo-treated patients (see Table 4). The most common treatment-related adverse event was epistaxis, which occurred in 6 (12%) MFNS-treated patients and 4 (8%) placebo-treated patients. One of the 6 MFNS-treated patients experienced severe intermittent epistaxis on day 20 of the study; all other instances of epistaxis were of mild or moderate severity. One 6-year-old MFNS-treated patient and one 3-year-old placebo-treated patient discontinued treatment because of epistaxis that was considered to be treatment-related. No other patients withdrew from the study because of treatment-related adverse events.
DISCUSSION

In contrast to some previous studies that have found suppression of either short-term or long-term growth in children after administration of other intranasal corticosteroids, the results from this study indicate that intranasal MFNS 100 μg QD does not retard growth in children.

This study found that the mean height of MFNS-treated children was similar at all time points to that of the placebo-treated children. Although the change in height from baseline was significantly greater in MFNS-treated patients at weeks 8 and 52, the rate of growth over the entire year was similar in both treatment groups, as demonstrated by the supplementary analysis. The slight differences in the change in height at weeks 8 and 52 may have reflected seasonal variations in growth rate in the 2 treatment groups or may have reflected individual variations in growth rates.

A recent knemometry study provided additional support for the lack of growth suppression by MFNS in children. This study investigated longitudinal lower leg growth with knemometry in children 6 to 12 years of age with allergic rhinitis. No reduction in lower leg growth velocity was seen in children treated with MFNS for 2 weeks at a dosage of either 100 μg QD or 200 μg QD.

The assessments of HPA-axis function via cosyntropin stimulation in the current study were also unable to detect systemic activity of MFNS, in agreement with the absence of growth retardation. The effect of cosyntropin on cortisol secretion is among the measurements of HPA-axis function commonly used in clinical trials and has successfully detected systemic effects of corticosteroids. This study found no evidence of HPA-axis suppression, as measured by basal plasma cortisol concentrations or cosyntropin stimulation, in children as young as 3 years of age after 1 year of treatment with MFNS. These results were not surprising, because the FDA has indicated that childhood growth may be a more sensitive indicator of systemic corticosteroid effects than the HPA axis.

The cosyntropin stimulation results from this study are consistent with previous reports, which also have demonstrated a lack of effect of MFNS on the HPA axis in pediatric patients. Studies of MFNS in children have demonstrated a lack of systemic activity after treatment with 50, 100, or 200 μg/day for 7 days in 6- to 12-year-old children and for 14 days in 3- to 5-year-old children, as measured by urinary free-cortisol concentrations, plasma cortisol concentrations, and plasma cortisol response to cosyntropin stimulation. Mometasone furoate concentrations were assessed in the 6- to 12-year-old children. Although the change in height from baseline was significantly greater in MFNS-treated patients. Therefore, the bioavailability of MFNS in children 6 to 12 years of age was too low to be calculated.

These findings are consistent with 2 studies by Brannan et al. that found no evidence of HPA-axis suppression in healthy adult male volunteers after single intranasal doses of as high as MFNS 4000 μg (20 times the normal recommended dose for adults) or in PAR patients treated with MFNS 200 or 400 μg QD for 36 days. In contrast, intranasal formulations of both BUD and BDP have been found to have significant systemic effects. For instance, 4 days of treatment with either BDP 400 μg BID or BUD 200 μg BID has been found to significantly suppress urinary cortisol values.

The results of the current study show that 1 year of treatment with MFNS 100 μg QD did not retard growth and did not suppress the HPA axis in children 3 to 9 years of age, indicating a high level of systemic safety for MFNS. MFNS also was found to be well tolerated for the treatment of PAR in these children, with the overall incidence of adverse events similar in both treatment groups. These findings are in agreement with the results of other studies that have found that MFNS 100 μg QD is well tolerated for the treatment of SAR in children.

The absence of systemic effects of MFNS, as demonstrated by assessments of both growth and HPA-axis function, suggests that it may be particularly useful for the treatment of PAR in children. Furthermore, MFNS may provide a greater degree of systemic safety than other intranasal corticosteroids, such as BUD and BDP, which have been demonstrated to have significant systemic effects including growth suppression.

These findings may be particularly relevant for children with PAR who also suffer from co-morbid disorders such as asthma and atopic eczema. These patients are also at risk of systemic exposure to corticosteroids from inhaled or dermatological products, thereby increasing their risk for systemic adverse events including growth suppression. Therefore, especially when treating children with corticosteroids for multiple atopic disorders, physicians should consider the systemic safety profile of each medication and select those with high local potency and minimal systemic activity.

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