Vitamin D Level in Children Is Correlated With Severity of Atopic Dermatitis but Only in Patients With Allergic Sensitizations


PURPOSE OF THE STUDY. To determine the effect of vitamin D on atop dermatitis (AD) severity in children with and without allergic sensitization.

STUDY POPULATION. Children with AD, followed in the pediatric allergy department of a Turkish tertiary care hospital, were enrolled in this study. Exclusion criteria were use of topical or systemic steroid treatment in the past month and using vitamin supplementation in the past 6 months.

METHODS. Subjects were designated as having mild, moderate, or severe AD based on SCORing Atopic Dermatitis index. Skin prick testing and specific immunoglobulin E testing to foods and aeroallergens allergens were used to determine allergic sensitization. Peripheral eosinophil counts, 25-hydroxy vitamin D levels, and total immunoglobulin E were measured. Patients were grouped according to allergic sensitization.

RESULTS. Seventy-three pediatric AD patients, median age 33 months, were enrolled in the study; 33 of 73 were found to have allergic sensitization. Vitamin D levels of participants with moderate and severe AD were significantly lower than those with mild disease ($P = .01$). In the sensitized group, vitamin D levels of participants with moderate and severe disease were also significantly lower than those of participants with mild severity ($P = .01$). In those not sensitized, vitamin D levels did not differ among those with mild, moderate, and severe AD. There was a negative correlation between SCORing Atopic Dermatitis score and serum vitamin D level in those with allergic sensitization ($P = .047$, $r = -0.349$). There was no correlation in the group without sensitization. Vitamin D was not correlated with eosinophil count or total immunoglobulin E in either AD group.

CONCLUSIONS. In participants with AD and allergic sensitization, those with lower vitamin D levels had more severe AD.

REVIEWER COMMENTS. This study helps set the groundwork for future studies investigating the efficacy of vitamin D supplementation in allergic individuals with moderate to severe AD.

Grass Pollen Counts, Air Pollution Levels and Allergic Rhinitis Severity


PURPOSE OF THE STUDY. To assess the association between pollen count and severe seasonal allergic rhinitis (SAR) after controlling for air pollution levels and other confounders.

STUDY POPULATION. A nationwide sample of 36 397 patients in France who were suffering from SAR as defined by Symptomatic Global Score in the upper third quartile and were consulting a physician (general practitioner, ear-nose-throat specialist, pediatrician, or allergist) between May and August 2004. Patients were untreated and had uncomplicated SAR. Patients from all age groups were included.

METHODS. A multilevel model relating severity of SAR as the dependent variable and pollen counts and air pollution levels as independent variables was used. To understand the respective roles of pollen counts and air pollutants, 2 models were used: (1) a model taking into account only daily airborne pollen counts and (2) a model with both daily pollen counts and air pollution concentration.

RESULTS. A rise of 60 grass pollen grains per cubic meter increased the risk of severe SAR by 8% after adjusting for confounders (age, gender, address) and air pollution levels.

CONCLUSIONS. Grass pollen count aggravated SAR in terms of nasal and ocular symptoms in the nationwide sample, and the relationship between severity of SAR and grass pollen counts was not modulated by air pollution.

REVIEWER COMMENTS. This is a large study that quantifies the magnitude of change in SAR severity in relation to measured grass pollen counts. Because air pollution levels did not significantly modify this relationship, monitoring of pollen counts can be informative for the management of SAR regardless of the air quality.
This study investigated the association of objectively determined OME at age 6 with asthma, eczema, allergic and nonallergic rhinitis, nasal inflammation, and eosinophilia.

STUDY POPULATION. A single-center birth cohort of 262 children born to asthmatic mothers from the Copenhagen Prospective Study on Asthma in Childhood study were evaluated during their sixth year of life for OME.

METHODS. OME was diagnosed on the basis of otoscopic findings and tympanometry. Nasal patency was assessed twice, in and out of the pollen season, using wideband nasal acoustic rhinometry. Nasal scrapings were obtained and stained for eosinophils.

Atopic comorbidity was assessed by using standardized guidelines for asthma, allergic rhinitis, nonallergic rhinitis, and eczema. Allergic rhinitis was suspected via a history of sneezing or a blocked or runny nose after exposure to relevant allergens. Allergic sensitization was confirmed by serum-specific immunoglobulin E levels $\geq 0.35$ kU/L as determined by ImmunoCAP (Pharmacia Diagnostics, Uppsala, Sweden) for any of the common allergens (cat, dog, horse, birch, grass, mugwort, dust mite, and molds). Potential confounders were identified including pet exposure, parental atopy, household income, tobacco smoke exposure at birth, siblings, and gender.

RESULTS. OME was identified in 39% (102 of 262) of patients. OME was associated with concomitant allergic rhinitis (adjusted odds ratio = 3.36, $P = .02$) but not with nasal mucosal swelling, nasal eosinophilia, nonallergic rhinitis, asthma, or eczema. There was no correlation between pollen season and OME ($P = .48$).

CONCLUSIONS. OME is common in children born to asthmatic mothers. The presence of allergic rhinitis in the subjects significantly increased the risk of OME.

REVIEWER COMMENTS. Previous studies that have looked for an association between atopy and middle ear disease have been criticized for inconsistent definitions of OME and allergy. Many of these used questionnaires or physician subjective diagnoses and were therefore subject to bias. This study’s major strength lies in its objective measurements for OME and allergic rhinitis. The authors conclude that allergic rhinitis significantly increases the risk of having OME. This study suggests that obstruction of the eustachian tubes is not the primary mechanism leading to the development of OME; rather, the effusion appears to result from allergic inflammation of the respiratory epithelium, including the middle ear mucosa. This suggests that additional studies are needed to determine if “antiinflammatory agents” used for allergic diseases could play a role in the treatment or prevention of middle ear effusion.

Seasonal Variability in Paediatric Obstructive Sleep Apnoea


PURPOSE OF THE STUDY. To examine the seasonal effect on the incidence and severity of obstructive sleep apnea (OSA) in children. Pediatric obstructive sleep apnea is primarily due to adenotonsillar hypertrophy. Allergy and viral respiratory infections may contribute to OSA by promoting adenotonsillar growth.

STUDY POPULATION. Two hundred fifty-seven Australian children aged 3 to 12 years were referred for assessment of suspected OSA. All underwent overnight polysomnography (PSG).

METHODS. Clinical analysis of the PSG data were scored according to standard criteria. The obstructive apnea hypopnea index (OAHI) was defined as the total number of obstructive apneas, mixed apneas, and obstructive hypopneas per hour of total sleep time. Children were divided into 3 groups by OSA severity: primary snoring (OAHI <1), mild OSA (OAHI 1–5), and moderate/severe OSA (OAHI >5). Data from each subject was grouped into the season in which it was obtained; summer: December to February; autumn: March to May; winter: June to August; spring: September to November.

RESULTS. Although the summer season had the fewest number of PSG performed because the unit was closed for the summer holidays, there was not a statistical difference in the average number of PSGs performed. OAHI values were significantly higher during winter ($5.1 \pm 0.8$ events per hour) and spring ($4.6 \pm 0.9$ events per hour) compared with autumn ($2.4 \pm 0.8$ events per hour; $P < .01$ and $P < .05$, respectively) and summer ($2.0 \pm 0.5$ events per hour; $P < .05$ for both). A significantly higher portion of children were categorized with moderate/severe OSA during winter compared with autumn ($P < .05$).

CONCLUSIONS. The authors point out that seasonal variation may play a role in OSA severity. They noted that OAHI values were higher when PSG was performed during winter and spring season compared with values obtained during autumn and summer. The severity of OSA may be affected by the season in which PSG is performed. For those patients with borderline results when PSG was obtained during spring of autumn, their OSA symptoms may be more severe if PSG was performed during the winter months. The authors speculate that viral illnesses, which are more common during the winter season, contribute to adenotonsillar hypertrophy and would lead to more severe symptoms of OSA.
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