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Serum leptin level in children with atopic dermatitis-treated topical steroids

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Leptin, the obese gene product, is a 16-kDa peptide hormone secreted by adiposities. Systemic administration of exogenous glucocorticoids has been found to increase circulating leptin levels. In this study, we aimed to assess serum leptin in children with atopic dermatitis (AD)-treated with local steroids. Twenty children with AD were included during the 2001–2002 time period. The study was conducted prospectively. Atopy was defined as the presence of at least one aeroallergen-specific immunoglobulin E (IgE) antibody. Serum leptin was determined using a commercially available radioimmunoassay kit with 3.4–8.3% intra-assay and 3.0–6.2% interassay coefficients of variation, and 0.5 ng/ml sensitivity. Fourteen boys and six girls with AD, the mean age of the patients was 3.1 ± 2.2. Forty-three percentage of the family histories for atopy were positive, 60% of the cases passive smoking histories were positive. In seven patients the aeroallergen-specific IgE were positive. All 20 patients treated clobetasone 17-butirate (0.05%). There was no significant difference in serum leptin between patients (mean ± s.d.: 4.6 ± 3.8), and controls (mean ± s.d.: 6.2 ± 3.6) ($p > 0.05$). Local steroid does not influence circulating leptin levels, suggesting that regulation of body weight is unaffected.

İlknur Bostanci[1], Özlem Atli[2], Nermin Çelebı[3], Ayşin Taşar[2], Esra Alpkarakoç[4] and Yıldız Dallar[5]
Departments of [1]Pediatric Allergy, [2]Pediatrics, [3]Biochemistry, [4]Microbiology and [5]Chief of Pediatrics, Ankara Education and Research Hospital, Ankara, Turkey

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İlknur Bostanci, Department of Pediatric Allergy, Ankara Education and Research Hospital, Ankara, Turkey
E-mail: ilknurbirol@hotmail.com

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Since the discovery of leptin in 1994 as an important factor influencing appetite and energy expenditure in animal (1, 2). Leptin, the obese gene product, is a 16-kDa peptide hormone secreted by adipocytes. Systemic administration of exogenous glucocorticoids has been found to increase circulating leptin levels in adults (3) and in children (4). Infants and young children with atopic dermatitis (AD) are at great risk of developing respiratory allergy later in life with rhinitis, eye symptoms, and sometimes asthma (5). AD is a common skin disease with a complex immunopathology that has a significant impact on the quality of life of patients and their families. Although topical corticosteroids have been the cornerstone of therapy, safety concerns, especially in children, with potent preparations or with chronic use have prompted treatment with various adjunctive therapies (6). Since potent of topical steroid may cause systemically detectable effects. But to this point there have been few studies investigating the effect of topical steroid therapy in children on leptin levels. This is the first study that shows AD patients have not change serum leptin level with treated topical steroid in children.

In this study, we aimed to assess serum leptin in children with AD-treated with topical steroid.

Subjects and methods

We performed an open, prospective study for among 20 children with AD, aged between 1 and 8 years. AD was defined as pruritic, chronic, or chronically relapsing dermatitis, with typical features and distribution. This study recruited children who had been diagnosed with AD according to the criteria proposed by the Hanifin and Rajka (7). A score was used to evaluate the severity and extent of involvement (graded 0–3 each) (8). All

patients were followed up in our Pediatric Allergy Department. Age-matched control children were enrolled from those managed in the same hospital department of general pediatric Outpatient for non-allergic and non-immunologic diseases. None of these children had been treated with topical steroids for atopic eczema or received systemic steroids for more than 3 days during the previous 6 months before enrollment into the study. All the children with AD were given medium-dose clobetasone 17-butirate (0.05%, GlaxoSmithKline, Istanbul, Turkey) twice daily. Only antihistaminic (cetrizine) was used for relief of symptoms when need. Compliance was measured by weighing the tubes before and after trial. Compliance was considered acceptable if there was > 90% reduction in the weight of the tubes. The clobetasone 17-butirate (0.05%, GlaxoSmithKline) was applied on average 8% of total body surface area in patients.

Children were seen as outpatients at 2 weeks during the treatment period.

After detailed physical examination blood was with-drawn after 12 hours of overnight fasting, at 08.30 am for fasting leptin and specific immunoglobulin E (IgE) levels. Subjects or their parents gave informed written consent, and this study were approved by the local ethics committee.

Total and aeroallergen-specific IgE concentrations

Peripheral venous blood was collected from our subjects for the measurement of serum total IgE concentration using nephelometry (Immage Immunochemistry system, Beckman Coulter, CA, USA). The results of which are presented following logarithmic transformation (IgE_{log}). Serum concentrations of specific IgE antibodies to *Dermatophagoides* p antigen (Der p 1), cat, cockroaches, molds, grass, egg-yellow, milk, and casein were measured by liquid-phase enzyme immunoassay method (DPC AloSTAT System, Diagnostic Products Corporation, Los Angeles, CA, USA) The grading of specific IgE into classes 0–4 was made according to manufacturer's instructions, with a specific IgE concentration of 0.35 kIU/l being positive. Atopy was defined as the presence of at least one aeroallergen-specific IgE antibody.

Serum leptin

A portion of the fasting serum was frozen and kept at −20°C until assay. Serum leptin was determined using a commercially available radioimmunoassay kit (human Leptin RIA kit; Linco Research Inc., St Louis, MO, USA) with 3.4–8.3% intra-assay and 3.0–6.2% interassay coefficients of variation, and 0.5 ng/ml sensitivity.

Statistical analysis

The data were analyzed using SPSS version 10.0. The demographic data and serum total IgE and serum leptin level in patients and controls were analyzed using the Student's *t*-test or the chi-square test. p-Values of < 0.05 were considered to be significant.

Results

Twenty, AD children and 20 control subjects were enrolled between November 2002 and January 2003. Their respective mean ages were 3.1 (s.d.: 2.2) years patients and 2.7 (s.d.: 2) years control ($p = 0.9$). Fourteen (70%) patients and 11(55%) control subjects were male ($p = 0.51$). On admission to the study, the mean eczema score was 4.9 ± 1.2 (range 2–8, median 5). The other clinical characteristics of children in the AD are summarized in Table 1. The family history for atopia was positive in 43%, passive smoking history was positive in 60% of the cases. In seven patients the aeroallergen-specific IgE were positive. All 20 patients treated clobetasone 17-butirate (0.05%). There was no significant difference in serum leptin between patients (mean ± s.d.: 4.6 ± 3.8), and controls (mean ± s.d.: 6.2 ± 3.6) ($p > 0.05$) (Table 2).

Table 1. Characteristics and clinical indicators of atopic dermatitis in our patients

Characteristics	Results*
Demographic data	
Age (years)	3.1 ± 2.2
Age at assessment (years)	1.2 ± 0.8
Gender (male/female)	14/6
Coexisting physician-diagnosed allergic rhinitis, n (%)	3 (15)
Coexisting physician-diagnosed asthma, n (%)	6 (30)
Coexisting physician-diagnosed food allergy, n (%)	3 (15)
Smoke exposure, n (%)	12 (60
Family history, n (%)	8 (42)
Disease severity score	4.9 ± 1.2
Details on antiatopic dermatitis treatment	
Number of patients receiving local corticosteroids, n (%)	20 (100)

*Expressed as mean values.

Table 2. Summary of laboratory investigations on serum total, aeroallergen-specific immunoglobulin E (IgE) and serum leptin level concentrations

Characteristics*	Patients	Controls	p-Value
Serum IgE_{log} in mean (s.d.), kIU/l	307 ± 79	45 ± 20	0.000
Positive serum-specific IgE levels to aeroallergens for mite, n (%)	7 (35)	0	0.02
Serum leptin level (μg/l)	4.6 ± 3.8	6.2 ± 3.6	0.6

*Values are mean ± s.d.

Discussion

Leptin is a pleiotropic hormone that is involved in the regulation of food intake and body weight (1). One of the most troublesome features of AD is its chronic relapsing nature, and there is a lack of published evidence on the best treatment strategy for long-term management of the disease. Ng et al. show that the effect of postnatal systemic dexamethosone on serum leptin, insulin and hormones of the hypothalamic-pituitary-adrenal axis in preterm, very low birth weight infants (9). The administration of systemic corticosteroid resulted in significant increases in serum leptin and insulin (6). Cleare Aj et al. showed that increases in plasma leptin levels flowing low dose hydrocortisone therapy in adults with chronic fatigue syndrome (10). But to this point there have been few studies investigating the effect of topical steroid therapy in children Heuck et al. reported similar data as ours (11). They showed that inhaled budesonide 800 μg/day does not influence circulating leptin levels. In our study, at the end of the second week of topical steroid administration, circulating leptin is not influenced.

In conclusion, data obtained in our study show that serum leptin level is not influenced by the presence of AD with topical steroid treatment. Further studies are needed to clarify the relation between leptin, and children treated with steroid therapy.

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