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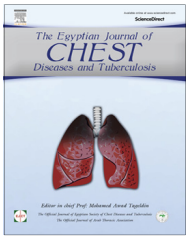

Serum resistin as an asthma marker and predictor of inhaled corticosteroid response in bronchial asthma in children

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KEYWORDS

Asthma;
Inhaled corticosteroids;
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Abstract Adipokines are factors produced by adipose tissue, that may be proinflammatory (such as leptin and resistin) or anti-inflammatory (such as adiponectin). Effects of these adipokines on the lungs have the potential to evoke or exacerbate asthma.

Aim: Our aim was to assess if serum resistin level is changed in obese and non obese asthmatic children and if it can predict their responsiveness to inhaled glucocorticoids.

Methods: Serum levels of resistin, were measured in 60 asthmatic children (30 obese and 30 non obese asthmatic children), and in 30 age and sex matched healthy controls. The measurements were repeated in all asthmatics after 8 weeks of treatment with inhaled corticosteroids.

Results: Serum resistin levels were found to be significantly elevated in all asthmatic children than control group and it was significantly elevated in obese children, compared with asthmatic non obese and control children 35 ± 0.2 ng/mL vs 20 ± 0.25 vs 10 ± 0.11 ng/mL respectively ($F < 0.005$). There was negative correlation between asthma severity as detected by FEV1 and serum resistin levels. Serum resistin levels in all asthmatic children had no correlation with duration of asthma in years. Serum resistin level was significantly reduced in all asthmatic children after inhaled corticosteroids for 8 weeks. Also asthmatic children with good response to inhaled corticosteroids had high initial resistin levels compared to corticosteroids non-responder.

Conclusions: From these results we can conclude that resistin can be considered as a marker of asthma and its severity and high resistin levels can predict favourable anti-inflammatory effect of inhaled glucocorticoids suggesting that resistin may be a marker of steroid-sensitive genotype in asthma in children.

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Introduction

Asthma is a chronic inflammatory airway disease characterized by cough, chest tightness and wheezing, and it is associated with reversible or variable airway obstruction. However, the diagnosis and follow-up of the disease are currently based on symptoms and lung function measurements rather than on assessing the underlying inflammatory process [1]. Several asthma phenotypes with different inflammatory mechanisms have been described suggesting that asthma is not a single disease entity but a syndrome with different underlying causes and mechanisms [2]. Adipokines like leptin, adiponectin, resistin and adipsin are protein mediators secreted by adipocytes and macrophages within the adipose tissue [3]. Leptin and resistin are usually pro-inflammatory, while adiponectin has mainly anti-inflammatory properties [3]. There are some evidence suggesting connections between adipokines and asthma. However, further studies are needed to understand the role of adipokines in the pathogenesis of, and more importantly, in predicting treatment responses in different phenotypes of asthma. The efficacy of treatment with inhaled glucocorticoids seems to vary between asthmatic phenotypes, and phenotype-specific predictors of treatment response are needed [2]. The aim of the present study was to assess serum levels of resistin in obese and non obese asthmatic children and to assess if it can predict the responsiveness to inhaled corticosteroids in asthmatic children.

Subjects

Sixty asthmatic children (mean age 8 years, range 5–15 years) diagnosed according to GINA guide lines and followed up in asthma clinic in Pediatric department of Tanta University Hospital were enrolled in this study Asthmatic children were classified into 2 groups. Obese asthmatic children (group 1) and non obese asthmatics (group 2), in addition to 30 age and sex matched healthy children as control group (group 3).

Exclusion criteria

Children with chronic heart or pulmonary or endocrine disease and children who received inhaled glucocorticoids in the last 6 weeks before the study were excluded.

Methods

All children were subjected to full history taking, thorough clinical examination, BMI, pulmonary function test including FEV_1. Lung function, asthma symptom score, and serum levels of resistin were measured in all asthmatics and in controls. The asthmatics also filled in an asthma symptom questionnaire. The same measurements were repeated in all asthmatic children after 8 weeks of treatment with inhaled glucocorticoids 500 μg b.i.d. during weeks 1–4, and 250 μg b.i.d. during weeks 5–8). The study was approved by the ethics committee of Tanta University Hospital and parents of all subjects gave written informed consent.

Sample collection

Venous blood was collected for the assessment of serum levels of resistin determined by enzyme-immuno-assay (EIA) by using commercial reagents (DuoSet ELISA, R&D Systems Europe Ltd, Abindgon, U.K. Netherlands) [4].

Asthma symptoms questionnaire

Asthma symptoms were recorded by using written symptom questionnaire. Cough, chest tightness, wheezing and nocturnal asthma symptoms were each scored from 0 to 3 yielding a total score from 0 to 12 points [5].

Statistics

Differences in resistin levels between asthmatics and controls were analysed with *t*-test, where appropriate. Pearson correlation coefficient was used to analyse correlations between resistin levels and lung function indices. Changes in serum levels of resistin before and after inhaled glucocorticoids treatment were analysed with a paired *t*-test. Results were presented as mean ± SD, and *P*-value < 0.05 was considered as significant. SPSS 15.0.1 software

Results

The data base of all groups is given in Table 1. There were no statistical significant differences as regards age, sex percentages between all asthmatics and controls. BMI was statistically sig-

Table 1 The demographic s of the studied groups.

	Obese asthmatics	Non-obese asthmatics	Controls	*F* Value
Age (Years) Mean ± SD	8.25 ± 1.5	8.20 ± 1.5	8.3 ± 1.25	$F > 0.5$
Sex				
M	12	12	12	$F > 0.5$
F	18	18	18	
Duration of asthma (years) mean ± SD	5.5 ± 2.5	5.00 ± 2.00	–	$F > 0.5$
BMI Mean ± SD	34.2 ± 2.1	22 ± 1.1	22 ± 1.4	$F < 0.05$
Eosinophils (Cell/ml) Mean ± SD	400 ± 20	380 ± 40	80 ± 10	$F < 0.05$
IgE Mean ± SD	280 ± 10	270 ± 15	50 ± 15	$F < 0.05$

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