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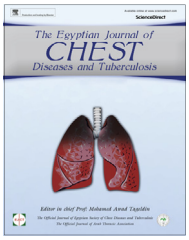

ORIGINAL ARTICLE

The relation between the blood osteopontin levels and body fat percentage in asthmatic women

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KEYWORDS

Obesity;
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Abstract *Introduction:* Obesity and asthma prevalence have been increasing over the past decade. Osteopontin (OPN) is a cytokine, with suggested diverse roles in tissue remodeling, fibrosis, immunomodulation, inflammation, and tumor metastasis.

Aim of the work: To assess the relation between serum osteopontin, immunoglobulin E (IgE) and body fat percentage in obese and non-obese asthmatic women in addition, to determine whether correlations exist between these parameters and asthma control.

Patients and methods: This study was conducted on 40 women after taking informed written consents. They were divided into 4 groups (10 each): healthy non-obese non-asthmatic (NO/NA), obese non-asthmatic (O/NA), non-obese asthmatic (NO/A) and obese asthmatic (O/A). All were subjected to full history taking, spirometry to non-asthmatic, asthma control questionnaire (ACQ) to asthmatic, determination of body fat percentage and serum levels of osteopontin and IgE.

Results: Body fat percentage was positively correlated to serum OPN levels. Body fat percentage was positively correlated to concentrations of IgE. In addition, the correlation between serum OPN levels and serum IgE levels was significantly positive. The improvement (presented by difference between ACQ before and after treatment (Δ ACQ)) was significantly superior in non-obese asthmatic. A negative correlation was detected between Δ ACQ and body fat percentage, serum OPN and IgE concentration.

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In conclusion: Because the multiple roles of OPN action potentially contribute to inflammation in obesity, it is suggested that, in addition to weight reduction, interference with OPN action could become a therapeutic strategy in the treatment of obesity worsening disorders like bronchial asthma.

Introduction

Asthma is characterized by eosinophilic inflammation of the conducting airways, and is regarded as a typical type-2 T-helper cell (Th2) associated allergic disease [1]. Airway inflammation in asthma is a multicellular process that is associated with structural alterations of the airway components, a process known as airway remodeling. The prominent role of airway inflammation and remodeling in the pathogenesis and clinical presentation of asthma has led to the current focus on mediators potentially involved in both processes [2].

Obesity and asthma prevalence have been increasing over the past decade. Several studies have identified an association between obesity and asthma and demonstrated that obesity results in an increased risk of developing bronchial asthma. Even modest levels of increased weight increase asthma risk. Moreover, obese asthmatic patients demonstrate increased asthma severity, as indicated by increased exacerbations and worse asthma control [3–5]. Most of these studies restricted their measurement of excess body weight to body mass index (BMI); however, other measures, such as total body fat or body fat percentage are more precise in order to determine the relative importance of obesity in the pathophysiology of bronchial asthma. It has been suggested that the association between obesity and asthma prevalence is stronger in women than men. A cross-sectional study found that a one-unit increase of BMI was associated with a 6% increase in asthma risk in women and 3% increase in men [6].

Published data suggest that obese asthma patients may represent a distinct phenotype of asthma [7,8]. Obese asthma patients demonstrate increased asthma severity, as indicated by increased exacerbations and decreased responses to conventional asthma therapies, specifically, relative corticosteroid resistance [9]. Small studies suggest improvements in the disease with weight loss in obese asthma patients. It is possible that altered lung mechanics associated with obesity could increase severity and worsen asthma control [10]. However, it is unclear and controversial whether the effects of obesity on asthma are mechanically mediated through restrictive effects on chest wall expansion or mediated through other mechanisms that are specific to the obese state. These suggested mechanisms include: (1) the presence of co morbidities, such as gastroesophageal reflux disease and sleep disordered breathing, (2) systemic and/or airway inflammation associated with obesity (with elevated levels of circulating inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), and (3) increased oxidative stress in obesity [8].

Multiple studies investigated the relationship between serum levels of hormones related to adipose tissue, such as adiponectin, leptin, and resistin and bronchial asthma. Although the data are sometimes conflicting, it seems that these adipocytokines may modulate airway inflammation and bronchial hyperreactivity. However, the relationship between serum levels of leptin/adiponectin and the presence of asthma is not sufficient to explain the relationship between asthma and obesity [11,12].

Osteopontin (OPN) is a cytokine, with suggested diverse roles in tissue remodeling, fibrosis, immunomodulation, inflammation, and tumor metastasis. It was originally described as a structural component of the extracellular matrix having the ability to bind to proteins and most types of collagen [13]. Although OPN is synthesized at the highest levels in the bone, it is produced by most cells of the immune system, including T-cells, B-cells, macrophages, neutrophils, eosinophils, natural killer cells and mast cells, as well as structural cells, including fibroblasts and smooth muscle and epithelial cells. In humans, increased OPN expression has been observed in a number of type-1 T-helper cells (Th1)-mediated lung diseases, including granulomatous diseases and pulmonary fibrosis [14]. There is now, also, emerging evidence to support an active role for OPN in type-2 T-helper cell (Th2)-linked inflammation and remodeling. OPN expression is upregulated in nasal tissue samples taken from asthmatic patients with chronic rhinosinusitis, and in the tear fluids of patients with allergic ocular diseases [15,16]. It has previously been demonstrated that OPN plays a crucial role in allergic airway inflammation [17–19]. Endobronchial biopsies from asthmatic individuals have shown increased osteopontin expression in bronchial epithelial cells and subepithelial inflammatory cells suggesting that eosinophils may be a cellular source of OPN [20]. Moreover, OPN is expressed in peripheral blood eosinophils of atopic human subjects, and acts as a chemoattractant for eosinophils in vitro [17,18]. It also may participate in the regulation of serum immunoglobulin E (IgE) levels in both asthmatic and non-asthmatic subjects [20].

Experimental studies on murine animal models of allergic airway disease demonstrated that OPN levels are increased in allergen-induced chronic airway remodeling, whereas, osteopontin deficiency, either through administration of blocking antibody or genetic deficiency, is protective against airway hyper-responsiveness (AHR) and airway remodeling [21,22].

Gómez-Ambrosi and his co-workers found that, plasma OPN levels and OPN expression in omental adipose tissue are increased in obese patients with high values of body fat percentage. It was suggested that measurement of OPN might be useful for evaluating the outcomes of obesity-related cardiovascular diseases [23].

Aim of the work

The aim of this study was to assess the relation between serum osteopontin, IgE levels and body fat percentage in obese and non-obese asthmatic women in addition, to determine whether correlations exist between these parameters and asthma control in these patients.