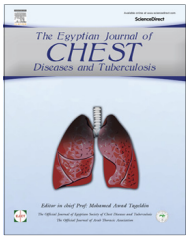
Egyptian Journal of Chest Diseases and Tuberculosis (2015) 64, 529–533

HOSTED BY

The Egyptian Society of Chest Diseases and Tuberculosis

Egyptian Journal of Chest Diseases and Tuberculosis

www.elsevier.com/locate/ejcdt
www.sciencedirect.com



ORIGINAL ARTICLE

Leptin and adiponectin are valuable serum markers explaining obesity/bronchial asthma interrelationship

Adel Salah, Mostafa Ragab, Waleed Mansour *, Mohammed Taher

Chest Department, Faculty of Medicine, Zagazig University, Egypt

Received 26 December 2014; accepted 18 February 2015
Available online 26 March 2015

KEYWORDS

Bronchial asthma;
Obesity;
Leptin;
Adiponectin;
Body mass index (BMI)

Abstract *Background:* Asthma in the obese represents a growing epidemic of pulmonary disease, and these patients are distinct from non obese asthmatics. Accordingly, studies on the pathogenesis of asthma in the obese are critical to guide our understanding of this disease process; such studies will ultimately guide the development of new therapies to treat the obese asthmatic population.

Patients and methods: Eighty (80) subjects were classified according to BMI into 4 groups: Group 1 (20 subjects): control none obese, they were apparently healthy subjects with BMI 22.9 ± 0.68 kg/m^2. Group 2 (20 subjects): control obese, they were apparently healthy subjects with BMI 36.16 ± 3.15 kg/m^2. Group 3 (20 patients): they were none obese asthmatic patients with BMI 22.97 ± 1.13 kg/m^2. Group 4 (20 patients): they were obese asthmatic patients with BMI 34.9 ± 2.4 kg/m^2.

Results: There was a higher leptin serum level in obese control (34.81 ± 2.32 pg/ml) compared to none obese control (9.73 ± 0.78 pg/ml) ($p < 0.01$). Moreover, there was a higher leptin serum level in obese asthmatic patients (39.74 ± 3.26 pg/ml) compared to none obese asthmatic patients (23.58 ± 1.99 pg/ml) ($P < 0.01$). There was a lower adiponectin serum level in obese control (4.95 ± 1.32) than non obese control (7.74 ± 3.13) ($P < 0.05$) and in obese asthmatic patients (3.3 ± 1.4) than non obese asthmatic patients (5.99 ± 1.5) ($P < 0.01$).

Conclusion: There is a strong association between asthma and obesity regarding serum level of leptin and adiponectin.

Introduction

There is a worldwide epidemic of obesity. In the USA, the prevalence of obesity, defined as a body mass index (BMI) $\geqslant$30 kg/m^{-2}, has increased among adults aged 20–74 years from, 15% in the late 1970 to, 35% in 2010. The obesity epidemic has impacted both developed and developing nations

* Corresponding author. Tel.: +20 1002868120.
E-mail addresses: dradelsalah1@gmail.com (A. Salah), mstrgb@yahoo.co (M. Ragab), waleedmansour123@gmail.com (W. Mansour), dr_mohamedsurgeon@yaho.com (M. Taher).

Peer review under responsibility of The Egyptian Society of Chest Diseases and Tuberculosis.

http://dx.doi.org/10.1016/j.ejcdt.2015.02.012

throughout the world. The World Health Organization estimates that worldwide, 2 billion people are either obese or overweight [1]. Not only is obesity a risk factor for asthma, but asthma in the obese has distinct features compared to disease in the non-obese. Obese asthmatics tend to have more severe disease [2,3] respond less well to standard controller therapy, [4] and have evidence of cellular glucocorticoid resistance [5]. This despite the fact they do not appear to have worsened airway inflammation as measured by either sputum eosinophils or neutrophils [6]. Asthma is associated with airway inflammation and reversible airflow obstruction. Obese asthma patients have more severe disease with increased asthma exacerbations, decreased asthma control, and decreased steroid responsiveness [7].

Asthma in the obese represents a growing epidemic of pulmonary disease, and these patients are distinct from non obese asthmatics. Accordingly, studies on the pathogenesis of asthma in the obese are critical to guide our understanding of this disease process; such studies will ultimately guide the development of new therapies to treat the obese asthmatic population [8].

Patients and methods

This study was carried out at Chest Department, Outpatient Clinics at Zagazig university hospitals from April 2013 to October 2013.

Study design: Prospective case control comparative study.

Type of selection: none randomized.

Subjects

The study included 80 subjects who were classified into 4 groups according to BMI and GINA 2012 [9]. Group 1 (20 subjects): control none obese, they were apparently healthy subjects. Group 2 (20 subjects): control obese, they were apparently healthy subjects. Group 3 (20 patients): they were none obese asthmatic patients. Group 4 (20 patients): they were obese asthmatic patients. All Asthmatic persons were diagnosed according to (GINA. 2012) [9].

Inclusion criteria

(1) Asthmatic patients either males or females, non-smokers or ex-smokers for at least 3 months well controlled on inhaled corticosteroids (ICS) with or without long acting β 2 agonist (LABA) [9]. (2) Obese BMI $\geqslant$ 30.3) Non obese have BMI (24.9–20) [10].

Exclusion criteria

Individuals with one or more of the following: known infectious disease, cardiovascular, rheumatic, malignancy, liver and kidney disorders, breast-feeding and pregnant women, and individuals with obesity due to secondary factors were excluded from the study [11]. Bronchial asthma patients who were receiving systemic steroids in the preceding 4 weeks were also excluded. [12].

Methods

All persons were subjected to:

(1) Full medical history: Including history of asthma symptoms especially; breathlessness, chest tightness, wheezing and cough, family history of asthma or atopic diseases, co-morbid disorders eg, DM, HTN, heart failure, liver cell failure and renal failure. (2) Full clinical examination (general examination and local chest examination): 1 – Weight which was measured in kgm, height which was measured in meters and accordingly BMI was calculated. (3) Plain chest X-ray: (postero-anterior view and lateral view) and HRCT chest if needed. (4) Full conventional laboratory investigation eg, CBC, ESR, RBS, liver function tests. (5) Spirometric ventilatory function was done by (Minispir S/N C00215): FEV1/FVC ratio <0.75–0.8 suggests airflow limitation. The degree of reversibility in FEV1 which indicates a diagnosis of asthma is generally accepted as 12% and 200 ml from the pre-bronchodilator value 15 min after inhalation of 200 μg of salbutamol [9]. (6) Measurement of inflammatory markers serum level: Fasting venous blood samples were collected at 09.00 h. After centrifuging at 4 °C, the blood was stored at −70 °C until analyzed [11]:

(A) Leptin: measurement of leptin serum level was done for all cases and control group by ELISA ''Enzyme – linked immunosorbent assay technique''. By (Bio-Rad, Hercules, CA, USA) in accordance with the manufacturer's guidelines. The minimum measurable level of leptin is 7.8 pg/ml.

(B) Adiponectin: measurement of adiponectin serum level was done for all cases and control group by ELISA ''Enzyme – linked immunosorbent assay technique''. 'ELISA KIT' by (anti Biotech OY, Orgenium Laboratories Business Unit, Finland).

Statistical analysis

Statistical analysis was performed with Epi Info™ version 7 and the SPSS version 19 statistical software package (SPSS Inc., Chicago, IL, USA). Data are presented as mean ± SD. For time point differences, a two-sample t test was used. P value <0.05 was considered significant.

Results

Demographic data of the studied population are demonstrated in Table 1 where, group 1 includes 20 subjects (10 males and 10 females) with mean age 33.4 ± 7.6 years, group 2 includes 20 subjects (11 males and 9 females) with mean age 33.6 ± 9.6 years, group 3 includes 20 asthmatic patients (11 males and 9 females) with mean age 31.9 ± 6.2 years, group 4 includes 20 obese asthmatic patients (9 males and 11 females with mean age 34.7 ± 8.08 with $P > 0.05$.

Table 2 shows the mean value of BMI in the studied population with highly statistical differences between group 1 with BMI 22.9 ± 0.68 kg/m^2, and group 2 with BMI 36.16 ± 3.15 kg/m^2, and between group 3 with BMI