Egyptian Journal of Chest Diseases and Tuberculosis (2015) 64, 557–565

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 hormone in obese and non-obese stable and exacerbated cases of chronic obstructive pulmonary disease☆

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Received 27 January 2015; accepted 5 March 2015
Available online 30 March 2015

KEYWORDS

Serum leptin;
Stable COPD;
COPD exacerbation;
Obese;
Non-obese;
BMI

Abstract *Objective:* The aim of this study was to assess the level of serum leptin hormone in chronic obstructive pulmonary disease patients during acute exacerbation and in stable conditions and also, to determine if these changes correlate with changes in the ventilatory functions.

Methods: Sixty cases were included in this prospective study (40 COPD patients and 20 age related smokers without symptoms or signs of COPD and within normal pulmonary functions as a control). Patients and control were divided according to their BMI into obese (BMI $\geqslant$ 30) and non-obese (BMI = 18.5–25). Subjects were submitted to full history taking, thorough physical examination, plain chest X-ray, complete blood count, erythrocyte sedimentation rate, liver and kidney functions, fasting and post prandial blood sugar, ventilatory functions, and serum leptin level measurement.

Results: Serum leptin level (ng/ml) was significantly higher ($P < 0.001$) in stable obese COPD (mean ± SD = 23.85 ± 4.47) patients than obese controls (mean ± SD = 20.9 ± 2.7) and stable non-obese COPD (mean ± SD = 5.63 ± 1.05) and stable non-obese COPD cases had significantly higher ($P < 0.05$) serum leptin level than non-obese controls (mean ± SD = 6.53 ± 1.19). Serum leptin level was significantly higher ($P < 0.001$) in obese COPD cases during exacerbation (mean ± SD = 67.59 ± 9.8) than in non-obese COPD cases during exacerbation

Abbreviations: BMI, body mass index; COPD, chronic obstructive pulmonary disease; SI, smoking index; PFT, pulmonary function tests; FEV1, forced expiratory volume in first second; FVC, forced vital capacity

☆ This work was primarily carried out in: Benha University Hospitals, Benha, Egypt.

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

http://dx.doi.org/10.1016/j.ejcdt.2015.03.011

(mean ± SD = 18.14 ± 4.15). Significant positive correlation between serum leptin and BMI (kg/m^2) of different groups ($P < 0.01$) [obese control ($r = 0.945$), non-obese control ($r = 0.970$), obese COPD in exacerbation ($r = 0.812$), obese COPD in stable state ($r = 0.774$), non-obese COPD exacerbation ($r = 0.876$) and non-obese COPD in stable state ($r = 0.799$)].

Conclusion: Serum leptin hormone level (ng/ml) was significantly higher in obese COPD cases than in controls and non-obese cases and during exacerbation than in stability which indicates that leptin plays a role in the systemic inflammatory process. Serum leptin hormone level positively correlated with BMI (kg/m^2).

Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in individual patients, its pulmonary component is characterized by airflow limitation that is not fully reversible, the airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases [1].

Tobacco smoking is a common risk factor for multiple co-morbidities, including coronary heart disease, heart failure and lung cancer. Co-morbidities such as pulmonary artery disease and malnutrition are directly caused by COPD. These extrapulmonary manifestations are mostly caused by chronic systemic inflammation. All of these co-morbidities potentiate the impact of COPD on health and lead to increased hospitalizations and healthcare costs [2–5].

Obesity is defined as a body mass index (BMI) greater than 30 kg/m^2; Over 1.6 billion adults worldwide are overweight, of which 400 million are obese. The World Health Organization predicted that 10% of the global population will be obese by 2015; In the United States of America, the annual health care costs are 36% greater for an obese patient compared with a patient with a normal BMI [6].

Leptin is a 167 amino acid peptide made exclusively in adipose tissue in a wide range of animal species, including humans. The (ob) gene encoding leptin is located on the mouse chromosome and the human homolog of the ob gene has been mapped to chromosome (7q31). Northern blot or RT-PCR analysis of the messenger ribonucleic acid (mRNA) for the ob gene showed that it was expressed only in adipose tissue [7–9].

Aim of the work

The aim of this study was to assess the level of serum leptin hormone in chronic obstructive pulmonary disease patients during acute exacerbation and in stable conditions and also, to determine if these changes correlate with changes in the ventilatory functions.

Methods

Sixty cases were included in this prospective study (40 COPD patients and 20 age related smokers without symptoms or signs of COPD and within normal pulmonary functions as a control). All COPD cases were in acute exacerbation and were admitted in El-Fayuom Chest hospital and Chest Department in Benha University from October 2010 to October 2011 and the diagnosis of COPD was established on the basis of Global Initiative for Chronic Obstructive Lung Disease [1]. All participants were divided according to their body mass index (BMI) values into non-obese (BMI $< 25 > 18.5$) and obese (BMI $\geqslant 30$). Participants were classified into 4 groups: group-1 (G1) contained 20 obese COPD cases, Group-2 (G2) contained 20 non-obese COPD cases, control-1 (C1) contained 10 control subjects and control-2 (C2) contained 10 control subjects.

Exclusion criteria for participants:

The presence of a co-morbidity can affect the levels of leptin hormone in cardiovascular disease, cerebral vascular diseases, Diabetes mellitus, arthritis, liver cirrhosis, end-stage renal disease, Tuberculosis, bronchiectasis, malignancy and connective tissue disorders [10].

All participants were subjected to the following:

1. Thorough history taking and clinical examination including:
   - Measuring of smoking index (number of packs/day x number of years smoking).
   - History of co-morbidities that may affect the level of the leptin hormone as ischemic heart diseases, hypertension, diabetes mellitus, tuberculosis, malignancy, end-stage renal disease, rheumatoid arthritis and any systemic infection or inflammation [10].
2. Body mass index (BMI): was calculated for all cases (the weight in kg divided by height^2).
3. Laboratory investigations: complete blood picture, erythrocyte sedimentation rate, fasting and 2 h post-prandial blood glucose, kidney and liver function tests.
4. Radiological examination: plain postero-anterior and lateral chest x-rays were done to exclude any chest lesion if present.
5. Pulmonary function tests (spirometry): before and after bronchodilatation using Sensor-medics V max series, 2130 Spirometer, V6200 Autobox, 6200DL. Ambient temperature and pressure were entered with the patient data (age in years, weight in kilograms, height in centimeters and sex) so that all results were calculated as percent of predicted (% predicted) except for FEV_1/FVC. Pulmonary functions were done pre and post bronchodilatation during acute exacerbation of the disease and pre and post bronchodilatation after management of cases during stability.
6. Serum leptin hormone measurement: