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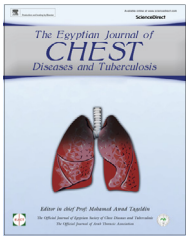

ORIGINAL ARTICLE

Adiponectin as inflammatory biomarker of chronic obstructive pulmonary disease

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KEYWORDS

COPD;
Adiponectin

Abstract *Background:* Systemic inflammation appears to be a major factor linking COPD to metabolic disorders as comorbidities. Obesity is considered a state of chronic, low-grade inflammation in which a greater production of pro-inflammatory cytokines overcomes the production of cytokines with anti-inflammatory properties.

Aim of the work: To measure serum adiponectin (APN) level in COPD (obese and non obese) during stable and acute exacerbation stage and to evaluate its role as a biomarker of systemic inflammation

Subjects and methods: This study was conducted on 88 subjects; 68 male smokers with COPD (34 stable and 34 exacerbated each group reclassified into [17 obese and 17 non-obese]) and 20 age and sex matched apparently healthy subjects as a control group. Serum APN level was measured with ELIZA. Ventilatory function testing and body mass index (BMI) were performed in all patients and control subjects.

Results: Serum APN level was approximately more than 3-fold higher in non-obese AECOPD and stable COPD cases than obese. However, more than 2-fold higher in non-obese control than obese. There was no correlation between adiponectin and FEV1, FEV1/FVC in COPD patients.

Conclusion: Serum adiponectin level was raised in non-obese COPD cases more than obese COPD cases and the rise is more during exacerbation. In addition, serum adiponectin level was raised in obese COPD cases during exacerbation and during stable conditions. A further rise in serum APN in exacerbation period denotes that APN may also be a biomarker of exacerbation and represents significant prognostic marker for COPD disease.

Abbreviations: COPD, chronic obstructive pulmonary diseases; APN, adiponectin; AECOPD, acute exacerbation COPD

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Introduction

Adipose tissue seems to interfere with systemic inflammation in COPD patients by producing a large number of proteins, known as ''adipocytokines'' or ''adipokines'' which are involved in various processes such as metabolism, immunity and inflammation. There is evidence that APN is an important modulator of inflammatory processes [1].

APN is a secretory protein hormone, synthesized by adipocytes and has important anti-inflammatory, anti-obesity and anti-atherosclerotic effects [2].

APN exerts anti-inflammatory effects, by reducing inflammatory cytokines and inducing anti-inflammatory ones through activation of all receptors. Several pathogenesis have been suggested, including direct actions on inflammatory cells' (inhibition of IL-6 production accompanied by induction of the anti-inflammatory cytokines IL-10 and IL-1 receptor antagonist [3], inhibition of endothelial Nuclear factor kappa beta (NF-Kß)) signaling by c-AMP proteinkinase A dependent pathway [4]. Its effect also extends to inhibit the production of the pro-inflammatory cytokine TNF-α [5]. The exact mechanism of APN participation in the COPD inflammation is still a point of debate [6].

Adiponectin also has an endocrine effect (as a circulating hormone acting on blood vessels, liver and skeletal muscles) and an auto/paracrine effect (differentiation of preadipocytes to adipocytes) [6].

Adiponectin and all of the known receptors for adiponectin such as AdipoR1, AdipoR2, T-cadherin and calreticulin are expressed on multiple cell types in the lung and APN has been isolated from BAL fluid [7–9].

Lower serum concentrations of APN may be associated with decreased lung function in humans. It is therefore possible that the lung is a target organ for APN signaling and consequently, adiponectin derangements may be associated with diseases of the lung [10].

Limited data are available on the role of APN in COPD, with the exception of an increase in its levels in underweight COPD patients and a marginal difference between stable phase and exacerbation [11].

Aim of the study

This study was carried out to measure serum adiponectin level in COPD (obese and non obese) patients during stable and acute exacerbation stage and to evaluate its role as a biomarker of systemic inflammatory response.

Subjects and methods

This case–control study was conducted at Chest Departments, Al-Zahraa University Hospital. The ethics committee of University Hospital institute approved the study. An informed written consent was obtained from all participants before their enrollment into the study. This study was conducted on 88 subjects. They are divided into two groups:

- *COPD group:* Including 68 male smokers, COPD patients (34 exacerbated and 34 stable). All of them had symptoms of chronic airflow limitation and fulfilled criteria set out by (**GOLD, 2011**) [12] for diagnosis of COPD. All COPD patients were smokers and had post-bronchodilator (FEV_1) less than 80% of the predicted value, along with an FEV_1/FVC not more than 70%. They had an increase in FEV_1 less than 200 ml or less than 12% of baseline value, 15–20 min after 2 puffs of inhaled salbutamol via a metered-dose inhaler [12]. Thirty-four patients of them had clinically stable COPD for at last 3 months and the other 34 patients had clinical symptoms of COPD exacerbation (increased dyspnea, sputum production or purulence) [12].
- *Control group:* includes 20 age and sex-matched apparently healthy nonsmoking volunteers.

Patients and control groups were reclassified according to body mass index (BMI) into non-obese (BMI < 25 and > 18) and obese (BMI > 30) as follows:

1. Thirty-four stable COPD (17 obese patients and 17 non-obese patients);
2. Thirty-four AECOPD (17 obese patients and 17 non-obese patients); and
3. Control group (10 obese subjects and 10 non-obese subjects).

- Overweight with BMI $\geqslant 25$ and $\leqslant 30$ were not included in this study [13].

Exclusion criteria

Patients with significant comorbidities, including other lung disease except from COPD, apparent heart failure, coronary artery disease, renal or liver impairment or failure, diabetes mellitus, history of cancer in any site, metabolic syndrome, collagen and vascular disorders were excluded [13].

Study design

All subjects were subjected to the following:

1. Detailed medical history and clinical examination, as well as history of any other co-morbidity that may rise the APN level.
2. Body mass index (BMI) was calculated as (kg/m^2).
3. Standard Laboratory tests (to exclude comorbid diseases), e.g. CBC, ESR, urine analysis, fasting and postprandial blood sugar, liver and renal function tests.
4. Plain chest radiography and ECG.
5. The ventilatory function tests were carried out using Spirosift spirometry 5000 FUKUDa NENSHI before and after bronchodilatation. Spirometric–indices were calculated using best out of three technically satisfactory trials according to the recommendations of the ATS (1987) [14]. The following parameters were recorded: FVC%, FEV_1%, FEV_1/FVC%.
6. Blood samples for adiponectin were drawn prior to the initiation of treatment for COPD during exacerbation, stable condition and for control group. Blood samples were collected by vein puncture into plain tubes. Sera were obtained by centrifugation at 1000*g*. for 5 min at room temperature; the samples were stored at -70 until analysis.