



SUMMARY
AND
CONCLUSION



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SUMMARY

COPD is a syndrome rather than a single disease because there are two major phenotypes in COPD. One is the emphysema-dominant type, so-called “pink puffers,” who are frequently cachexic. Another is the airway disease-dominant type, so-called “blue bloaters,” who are frequently obese. It was demonstrated that BMI might be one of the determinants of COPD phenotype.

The adipose tissue is an important contributor to systemic manifestations of COPD. Indeed the inflammatory/anti inflammatory effects of adipokines highlight the fact that adipose tissue is more than energy storage organ, they also highlight the importance of body composition in the pathogenesis of COPD.

Adiponectin is an adipocyte-specific protein secreted by visceral fat tissue that has anti-inflammatory as well as anti-obesity effects. In patients with metabolic syndrome, adiponectin levels in plasma decreased in proportion to the increase in body weight.

Although systemic manifestations as a result of low grade systemic inflammatory process are recognized by many researchers there is little knowledge on the alteration of adiponectin in COPD patients.

The present study was conducted in Elmehlla chest hospital and the chest department in Benha University Hospital in the period between July 2010 and July 2011.

The aim of this work was to assess the levels of serum adiponectin in Chronic Obstructive Pulmonary Disease patients (COPD) during acute exacerbation and in stable conditions and to determine whether changes in its levels correlate with changes in the ventilatory functions.

40 patients with COPD in exacerbation divided into (25 nonobese and 15 obese) and 15 controls with matched age divided into (8 nonobese and 7 obese) were included in this study.

All patients received medical treatment for acute exacerbation.

All subjects were submitted to:

1. Full history taking.
2. Full clinical examination.
3. Body mass index.
4. Plain x-ray chest (postero-anterior and left lateral views).
5. Spirometry pre and post bronchodilator.
6. Estimation of serum adiponectin during a cute exacerbation and in stable state.

Exclusion criteria included:

- 1- By history and examination any disease that may result in elevation of Serum adiponectin level such as (malignancy, infection, cardiac failure, severe endocrine disorder, hepatic or renal disease, systemic autoimmune or connective tissue disorder and recent surgery).
- 2- By spirometry: reversibility in post bronchodilator FEV1 (%pred) more than 12% or 200 ml.

The results were tabulated and statistically analyzed:

It was found that:

In nonobese subjects: serum adiponectin level was higher in COPD cases during exacerbation as compared to the control group and to stable COPD cases. Also serum adiponectin level was higher in stable COPD cases as compared to control group.

In obese subjects: serum adiponectin level was higher in COPD cases during exacerbation as compared to the control group. Also serum adiponectin level was higher in stable COPD cases as compared to the control group.

There was no any correlation between changes in serum adiponectin from exacerbation to stable conditions with changes in ventilatory functions (FVC (%pred), FEV1 (%pred), FEV1 / FVC and FEF25-75(%pred) from exacerbation to stable conditions.

Serum adiponectin level showed a negative correlation with BMI in COPD cases (nonobese and obese) and in controls (nonobese and obese).

CONCLUSIONS

From this study, it was concluded that:

1. Serum adiponectin level is raised in nonobese COPD cases and the rise is more during exacerbation.
2. Serum adiponectin level is raised in obese COPD cases during exacerbation and during stable conditions.
3. Serum adiponectin level is raised in nonobese COPD cases more than obese COPD cases.
4. Serum adiponectin level negatively correlated with the body mass index.