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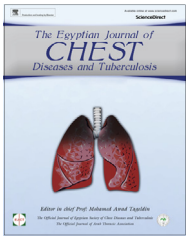

ORIGINAL ARTICLE

Osteopontin in chronic obstructive pulmonary disease: Smokers and ex-smokers

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KEYWORDS

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Abstract *Background:* COPD is a widely distributed disease with high morbidity and mortality, associated with different pathologies. The link between osteopontin, smoking and COPD needs to be clarified.

Objectives: To study the osteopontin serum levels in smoker COPD patients and ex-smoker COPD patients.

Methods: Serum levels of osteopontin were measured in 38 male COPD patients divided into smoker and ex-smoker groups using human osteopontin ELISA kits.

Results: There was a significant difference between smokers and ex-smokers regarding their serum osteopontin levels. There was no significant difference in serum osteopontin level between COPD patients regarding severity in both groups. There was a significant difference in serum osteopontin level between the smokers group regarding their pack year index of smoking. There was no significant difference in serum osteopontin level between ex-smokers group regarding the duration of cessation.

Conclusion: Serum level of osteopontin is increased in COPD patients and decreases with cessation of smoking. It has no relation to disease severity.

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].

Cigarette smoking is the major risk factor associated with the development of COPD; Heavy smokers are at greater risk of developing COPD than moderate smokers [2].

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Osteopontin is a secreted phosphorylated glycoprotein expressed in mineralized tissues and damaged renal tissues; it is also expressed by immune cells during inflammation and enhances the proinflammatory T-helper 1 cell response and inhibits the T-helper 2 cell response [3].

Aim of the work

The aim of the current study was to study the osteopontin serum levels in smoker COPD patients and ex-smoker COPD patients.

Patients and methods

This work was carried out on 38 male patients with COPD diagnosed and classified according to GOLD 2010 [1], referred to chest hospital in Shebin El-Kom. The study also included ten healthy male controls; their ages were matched with the patients.

Subjects were divided into:

Group A: included 19 smoker patients with COPD and were subdivided into: 14 patients with moderate COPD and 5 patients with severe COPD.
Group B: included 19 ex-smoker patients with COPD and were subdivided into: 12 patients with moderate COPD and 7 patients with severe COPD.
Group C: included 10 healthy volunteers who had no symptoms or signs of any chest disease and had normal ventilatory function tests as a control group.

After having a written consent; each patient underwent:

1. Full history taking and clinical examination.
2. Chest X-ray.
3. Pulmonary function tests.

Morning spirometry was done and all patients had FEV_1/FVC of less than 70% and post bronchodilator spirometry was performed after giving the patient a bronchodilator, such as an inhaled beta-agonist e.g. salbutamol 400 μg) [4].

The following parameters were measured:

(a) Forced expiratory volume in the first second (FEV_1) pre and post bronchodilator.
(b) Forced vital capacity (FVC).
(c) FEV_1/FVC ratio.
(d) Peak expiratory flow rate (PEFR).

Patients were classified according to GOLD staging system (GOLD 2010) [1] into:

Stage I: mild COPD (FEV_1/FVC < 70%; post bronchodilator $FEV_1 \geqslant 80\%$ predicted).
Stage II: moderate COPD (FEV_1/FVC < 70%, $50\% \leqslant$ post bronchodilator $FEV_1 \leqslant 80\%$ predicted).
Stage III: severe COPD (FEV_1/FVC < 70%, $30\% \leqslant$ post bronchodilator $FEV_1 \leqslant 50\%$ predicted).
Stage IV: very Severe COPD (FEV_1/FVC < 70%, post bronchodilator $FEV_1 \leqslant 30\%$ predicted or post bronchodilator $FEV_1 \leqslant 50\%$ predicted plus chronic respiratory failure).

Serum osteopontin level was measured for all subjects using human osteopontin ELISA kit.

Results

Table 1 shows the distribution of the studied groups regarding GOLD stages of patients in each group (classified according to GOLD 2010), and there was:

From 19 smoker patients in group (A), 14 (73.7%) had moderate COPD, 5 (26.3%) had severe COPD. While from 19 ex-smoker patients in group (B), 12 (63.2%) had moderate COPD, 7 (36.8%) had severe COPD. There was a non significant difference between smokers and ex-smokers groups as regards the GOLD stage (P-value > 0.05).

Table 2 shows that the mean age of patients in group A was 55.89 ± 8.17 years. In group B; the mean age of patients was 54.73 ± 6.37 years. Lastly, in the control group (C); the mean age of patients was 53.40 ± 9.45 years. There were insignificant differences between the patients of both groups and between them and the control group as regards age (P-value > 0.05).

Table 3 shows a significant difference between group (A) and group (B) regarding their serum osteopontin level (P-value < 0.01). There was also a significant difference between group (A) and the control group (C) (P-value < 0.01). However, there was a non significant difference between group (B) and group (C) regarding their serum osteopontin level (P-value > 0.05).

Table 4 shows that there was no significant difference between moderate and severe COPD smokers as regards serum osteopontin level (P-value 0.853).

Table 5 shows the non significant difference between moderate and severe COPD ex-smokers as regards serum osteopontin level (P-value 0.673).

Table 6 shows that there was no significant difference in mean serum osteopontin level between ex-smokers group regarding their duration of cessation (P-value 0.514).

Discussion

COPD is an inflammatory disease caused mainly by smoking [5]. Osteopontin as a mediator of inflammation may be implicated in the pathogenesis of a variety of disease states including COPD and its complications [6].

This work was carried out on 38 patients with stable COPD diagnosed clinically and by spirometry and classified into two groups: Smokers (Group A) and Ex-smokers (Group B). In addition to 10 healthy non smoker subjects as a control group were included. Serum OPN was measured for all subjects.

In the present study, the results showed a significant difference (P < 0.01) between smokers and ex-smokers regarding their serum OPN level (higher in smokers), there was also a significant difference between group A and the control group (P-value < 0.01). There was a non significant difference between group B and the control group regarding their serum osteopontin level (Table 3). These results were matched with the results of the studies carried out by Bishop et al. [7] who