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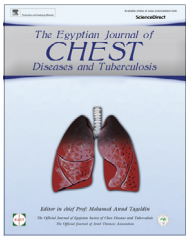

ORIGINAL ARTICLE

Assessment of eotaxin 1 in exhaled breath condensate of chronic obstructive pulmonary disease patients

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KEYWORDS

COPD;
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Abstract The aim of this work was to assess eotaxin 1 level in exhaled breath condensate of exacerbated and stable COPD patients in relation to normal subjects.

Subjects and methods: There were 16 COPD patients (during infective exacerbation), 16 stable COPD patients and 20 healthy volunteers as controls matched with them in age, sex and smoking history. EBC was collected and concentration of eotaxin 1 was measured by using Human Eotaxin 1 ELISA Kits.

Results: The mean eotaxin 1 concentration in exhaled breath condensate of studied groups was 962.5 ± 150 pg/ml in the exacerbated COPD group, 427.8 ± 186.6 pg/ml in the stable group and 89.2 ± 47.5 pg/ml in the control group. Age, smoking, FVC, FEV1, and FEV1/FVC, showed no significant correlation with eotaxin 1 levels among all the studied groups.

Conclusion: Eotaxin 1 levels in exhaled breath condensate of COPD patients during infective exacerbation was significantly higher than in stable COPD patients and both groups showed significant higher levels than the control group.

The prognosis for chronic obstructive pulmonary disease (COPD) is adversely affected by acute exacerbation, which increases mortality and morbidity and causes an irreversible decline of pulmonary functions [1]. It has been suggested that patients with more frequent exacerbations have an increase in airway inflammation and a higher baseline sputum interleukin (IL)-6 and IL 8 levels, and these cytokines may predict the frequency of future exacerbations [2]. It has also been reported that not only neutrophils but also eosinophils and lymphocytes may participate in the increased airway inflammation during acute exacerbation [3]. However, the details of the mechanism of increase in airway inflammation during exacerbation remain obscure.

Abbreviations: COPD, chronic obstructive pulmonary disease; EBC, exhaled breath condensate; IL, interleukin; Th, T-helper cells; FVC, forced vital capacity; FEV1, forced expiratory volume in first second; CCR3, c chemokine receptor 3

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It has been suggested that, among various causes of acute exacerbation, respiratory tract infection due to virus or bacteria may account for as much as, or even more than 80% of COPD exacerbations [4]. Infection of airway epithelial cells induces various cytokines and chemokines, such as eotaxin 1 which is a potent and selective chemoattractant for human eosinophils .The human eotaxin receptor, CCR3 is expressed on eosinophils, basophils and Th2 cells [5].

Eotaxin, in association with Th1-derived cytokine IL2 and Th2 – derived cytokine IL4 is an important T lymphocyte activator, stimulating the directional migration, adhesion, accumulation, and recruitment of T lymphocytes in parallel with the accumulation of eosinophils and basophils during the process of certain types of inflammation [6].

Exhaled breath condensate (EBC) is a promising source of biomarkers of lung disease. It is important to note that EBC is not a biomarker, but rather a matrix in which biomarkers may be identified, in that way equivalent to blood, sweat, tears, urine and saliva. EBC may be thought of either as a body fluid or as a condensate of exhaled gas [7].

Eotaxin 1 was assessed by many studies in patients with bronchial asthma. It was measured in serum and EBC of stable and unstable asthmatics and correlated with the level of control of asthma and monitoring of inflammatory process [8,9].

Many studies have investigated eotaxin 1 in serum and sputum of COPD patients, however, up to our knowledge no studies have investigated eotaxin 1 in exhaled breath condensate of COPD patients.

So, the aim of this work was to assess eotaxin 1 level in exhaled breath condensate of exacerbated and stable COPD patients in comparison to normal subjects.

Subjects and methods

This study was conducted in the chest department and outpatient clinic of the Ain Shams University hospital in the period between February and April 2013. Fifty-two individuals were included in the study; they were 32 COPD patients (diagnosed according to GOLD 2011 guidelines) and 20 controls. The candidates were classified into: 16 COPD patients (during infective exacerbation), 16 stable COPD patients and 20 healthy volunteers as controls matched with them in age, sex and smoking history.

Exclusion criteria

The following patients were excluded from the study:

- Patients with history of bronchial asthma.
- Patients with history of intake of inhaled or oral steroids.
- Patients with heart failure.
- Patients with pulmonary thromboembolism.

All candidates were subjected to:

- Full history taking.
- Thorough clinical examination.
- Chest X-ray.
- Routine laboratory investigations.
- Pulmonary spirometry to measure: FEV1, FVC and FEV1/FVC ratio. It was done using a Flowmate spirometer.
- EBC was collected by using a commercially available condenser (EcoScreen; Jaeger, Würzburg, Germany) according to the current ATS/ERS guidelines [10]. Patients were instructed to breathe tidally for 10 min with nose clip. The respiratory rate ranged from 15 to 20 breaths/min. Patients were asked to swallow their saliva periodically and to temporarily discontinue collection if they need to cough. At the end of collection 1.5–3.5 ml aliquots of condensate were transferred to Eppendorf tubes and immediately frozen. Samples were stored at −20 °C [11]. Concentration of eotaxin 1 was measured by using Human Eotaxin 1 ELISA Kits.

Statistical analysis

Continuous variables are expressed as mean and standard deviation. Categorical variables are expressed as frequencies and percents. A significance level of $P < 0.05$ was used in all tests. All statistical procedures were carried out using SPSS version 15 for Windows (SPSS Inc., Chicago, IL, USA).

Results

The current study was conducted on 52 male subjects, they were divided into: 16 exacerbated COPD patients, 16 stable COPD patients and 20 healthy volunteers as the control group. Their mean age was (59.5 ± 11.6, 56.9 ± 12.0 and 53.2 ± 5.2) years, respectively, with no statistical significant difference.

Among the exacerbated group 14 patients out of 16 (87.5%) were current smokers and two patients (12.5%) were ex-smoker with a mean smoking index 36.8 ± 16.8 pack year. While in the stable group 10 patients out of 16 (62.5%) were smokers, 5 (31.3%) patients were ex-smokers and one patient out of 16 (6.3%) was a non-smoker with a mean smoking index 42.4 ± 17.6 pack-year. In the control group there were 18 out of 20 (90%) current smokers, one ex-smoker (5%) and one non-smoker (5%). There was no statistical significant difference between the studied groups as regard smoking habit.

The spirometry showed no statistical significant difference between the exacerbated and stable COPD group as the mean FVC was 64.5% ± 21.8 and 64.2% ± 20.7 of the predicted in exacerbated and stable COPD patients, respectively. The mean FEV1 was 48.0% ± 19.8 and 47.6% ± 20.9 in exacerbated and stable groups, respectively. Also the mean FEV1/FVC ratio was 58.2% ± 10.6 and 58% ± 9.9 in exacerbated and stable groups, respectively.

As regard eotaxin 1 concentration in exhaled breath condensate of studied groups it was 962.5 ± 150 pg/ml in the exacerbated COPD group, 427.8 ± 186.6 pg/ml in the stable group and 89.2 ± 47.5 pg/ml in the control group.

Table 1 shows no significant difference between exacerbated and stable COPD cases as regard age, smoking habits, and spirometry. However, a highly significant difference between exacerbated and stable COPD cases was present as regard eotaxin 1 level with higher level among the exacerbated group.

Table 2 shows no significant difference between exacerbated and controls as regard age and smoking habits. However, a highly significant difference between exacerbated