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Plasma brain natriuretic peptide levels in COPD without pulmonary hypertension

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ABSTRACT

Introduction: It has long been known that COPD causes elevation in BNP level caused by hypoxia present in cases of pulmonary hypertension in COPD.
The aim of this study: This study was carried out to evaluate the changes in BNP level in COPD patients without pulmonary hypertension or cor pulmonale during exacerbation and after remission.
Subjects and methods: This study was created on 50 subjects, 30 COPD patients according to inclusion and exclusion criteria (BNP level will be measured during exacerbation and after remission) plus 20 age matched apparently healthy control subjects, ten of them are non smokers and ten are asymptomatic smokers. For all subjects, history taking, full clinical exam done. PFT (spirometry), BNP level measurement on human serum by ELIZA, routine labs (CBC, liver and renal function). ECG. echocardiography.
Results: Levels of BNP were significantly higher in COPD patients with mean (60.52 ± 30.98 pg/mL) than control with mean (21.13 ± 4.61 pg/mL) and higher during exacerbation (60.52 ± 30.9 pg/mL) than during remission (35.65 ± 16.54 pg/mL), BNP was significantly higher in grade (III, IV) with mean (86.94 ± 40.19, pg/mL) than grade (II) (56.76 ± 6.2 pg/mL) and grade (II) was significantly higher than grade (I) with mean (37.86 ± 8.81 pg/mL) and it was a significantly inversely related to post FEV1% and post FEF 25–75% and significantly direct correlated to paco2 and non significant negative correlation to pao2.
Conclusion: Plasma BNP can be used as a useful prognostic biomarker of COPD and a good predictor of exacerbation, As BNP level was significantly higher in COPD patients than in control groups, ($p < 0.005$) and also significantly higher in grade (IV, III) than grade (II) and was significantly higher in grade (II) than grade (I) COPD patients, BNP level significantly higher ($p < 0.005$) during exacerbation than during remission of COPD patients.

Introduction

COPD is a common preventable and treatable disease is characterized by persistent air flow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients [1].

Acute exacerbation of chronic obstructive pulmonary disease is defined as acute event characterized by a worsening of the patients respiratory symptoms that is beyond normal day to day variations and leads to a change in medications [1].

Natriuretic peptides are peptides that are released from the heart in situations of pressure and volume overload of the ventricles. There are 3 types of natriuretic peptides: atrial natriuretic peptide, B-type natriuretic peptide (BNP), and C-type natriuretic peptide. Atrial natriuretic peptide is released predominantly from the atria, BNP from the ventricles, and C-type natriuretic peptide from the endothelium [2].

Release of NPs results from cardiac wall stretch, ventricular dilation, or increased pressures from circulatory volume overload. The effects of NPs result in lowering blood volume and pressure [3].

Plasma brain natriuretic peptide (BNP) is a useful biomarker for the detection and follow up of heart diseases [4].

Abbreviations: BNP, brain natriuretic peptide; NT proBNP, N-terminal pro-B-type natriuretic peptide; NPs, natriuretic peptides.

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Plasma BNP level is elevated in patients with pulmonary hypertension (PH) and chronic lung disease with right ventricular overload [5]. Plasma BNP levels in Stable COPD patients without PH or cor pulmonale have not been studied well [6].

Aim of the work

This study was carried out to evaluate the changes in BNP level in COPD patients without pulmonary hypertension or cor pulmonale during exacerbation and after remission.

Subjects and methods

Study design

Prospective case control study.

Subjects

This study was performed in Benha University Hospital Chest Department on 50 subjects. They were divided into 2 groups:

1. Group A: included (30) patients with acute exacerbation of chronic obstructive pulmonary disease and will be classified according to grading intoGroup [A] 1: (10) patients grade I (FEV1 $\geqslant$ 80% of predicted).
   Group [A] 2: (10) patients grade II (50% $\leqslant$ FEV1< 79%).
   Group [A] 3: (10) patients grade III–IV (30% $\leqslant$ FEV1 < 49% of predicted. FEV1<30% of predicted) respectively.
2. Group B: included (20) healthy subjects, (10) of them are non smokers and (10) are asymptomatic smokers.

Inclusion criteria

Patients with COPD diagnosed according to GOLD (2016) criteria.

Exclusion criteria

1. Pulmonary hypertension, cor pulmonale or other chronic respiratory disease.
2. Exacerbations due to pneumothorax or cardiac failure without acute exacerbation chronic obstructive pulmonary diseases.
3. Patients with a history of symptoms and medication for cardiac, renal, neurological and psychological disease.
4. Malignancy.

All subjects were submitted to the following:

1. History taking: History of smoking, chest symptoms and any other co-morbidities.
2. Clinical examination: both general and local examination.
3. Radiological examination: Plain chest X ray postero-anterior and lateral views.
4. Pulmonary function tests (spirometry) before and after bronchodilatation.
5. Routine investigations as: Electrocardiography, complete blood count, liver function tests, kidney function tests and fasting blood sugar.
6. Measuring the oxygen saturation in the blood by pulse oximetry.
7. Electrocardiography (ECG) A 12 lead ECG including 3 bipolar limb leads, 3 unipolar limb leads and 6 unipolar precordial leads was performed.
8. Echocardiography was done to exclude pulmonary hypertension cases.
9. Plasma Brain natriuretic peptide B type measurement [7].

Using commercially available enzyme-linked immunosorbent assay (ELISA) were uesd according to the manufacture's instruction (Human NPPB (BNP) ELISA Kit, Thermo Scientific).

Statistically analysis

The collected data were analyzed using SPSS version 16 for windows. Categorical data were presents as number and percentage while continuous variables were presented as mean and SD if parametric, and as median and range if non parametric, chi square, paired t-test and spearman's correlation coefficients were used as tests of significance. Two sided $p < 0.05$ value was considered significant.

Results

Table 1 show that (100%) were males COPD patients.

The mean age for patients was (47.9 ± 6.48) years old and for control was (49.1 ± 5.01) years.

FEV1% and FVC% were significantly higher in control group than case group.

Table 2 and Fig. 1 show that BNP level significantly higher in COPD patients than control group.

Table 3 and Fig. 2 Show that BNP level significantly higher in COPD patients during exacerbation than after stability.

Table 4 and Fig. 3 show that BNP level was significantly higher in Group A3 than Group A1 and Group A2 and in Group A2 than Group A1 and that it was higher during exacerbation than during remission of COPD patients.

Table 5 and Fig. 4 show that BNP level significantly higher in Group (A) COPD patients than group (B) control smokers and non smokers. And significantly higher in control healthy smoker than control non smoker.

Table 6 and Fig. 5 show a significant direct correlation between BNP level and smoking index in COPD patients.

Table 7 and Fig. 6a and b show a Significant direct correlation between BNP level and paco2 and a non significant negative correlation between BNP and pao2.

Table 1
Demographic data of studied group.

	Case group	Control group
Sex n & %		
Male	30 (100%)	20 (100%)
Age in years		
Range	41–69	42–63
Mean ± SD	47.9± 6.48	49.1± 5.01
BMI		
Range	20–34.2	23.2–31.4
Mean ± SD	25.05 ± 3.9	26.68 ± 5.07
FVC%		
Range	39–98	80–99
Mean ± SD	77.5 ± 17.3	88.9 ± 7.72
FEV1%		
Range	25–81	72–100
Mean ± SD	59.5 ± 19.8	86.8 ± 7.7
FEV1/FVC%		
Range	38–69	72–87
Mean ± SD	60.03 ± 10	79.35 ± 4.97

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