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Original Research Paper

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C - REACTIVE PROTEIN AND ITS ASSOCIATION WITH SERUM URIC ACID AND BICARBONATE LEVEL IN COPD

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ABSTRACT

AIM: The study was planned to evaluate the association of C - reactive protein with Serum Uric Acid and Bicarbonate Level in COPD.

MATERIAL AND METHODS: In the present study total 100 (n=100) patients diagnosed for COPD, were enrolled for the study. Patients with neoplastic pathologies, pneumonia and Liver or renal diseases, pregnant and lactating females were excluded from the study.

RESULT: The mean level of Serum Bicarbonate, C-reactive protein and uric acid were significantly higher in COPD patients. A significant association was observed ($p \leq 0.0001$).

CONCLUSION: In the present study higher bicarbonate levels that could be the individual biomarker which can assess the respiratory acidosis and CRP and Uric Acid levels judges the severity the disease.

KEYWORDS : COPD, CRP, Bicarbonate, Uric acid

INTRODUCTION

Chronic obstructive pulmonary disease is characterized by chronic bronchitis, chronic airway obstruction, airway remodeling, and emphysema, leading and irreversible decline in lung function.[1]

COPD is characterised by chronic airflow restriction caused by a combination of small airway illness and parenchymal damage (emphysema), the proportional contributions of which differ from person to person. COPD is a common, preventable disease characterized by persistent respiratory symptoms and airflow limitation caused by airway and/or alveolar abnormalities, which are usually caused by significant exposure to noxious particles or gases and influenced by host factors such as abnormal lung development. [2] Chronic respiratory symptoms may occur prior to the onset of airflow restriction and are linked to the onset of acute respiratory episodes.[3] Individuals with normal spirometry might also experience chronic respiratory problems. The presence of emphysema, airway wall thickening, and gas trapping in a significant percentage of smokers without airflow limitation indicates structural evidence of lung disease. [3-4]

Chronic obstructive pulmonary disease (COPD) is presently the world's fourth biggest cause of mortality, but it is expected to rise to the third position by 2020.[5]In 2012, more than 3 million individuals died, accounting for 6% of all fatalities worldwide. COPD is a serious public health problem that may be avoided and managed.

COPD is a leading source of chronic morbidity and death across the world; many individuals suffer from it for years before succumbing to it or its consequences. Because of ongoing exposure to COPD risk factors and population ageing, the global burden of COPD is expected to raise in the future decades.[6]

However, a shift in respiratory exchanges, such as that seen in severe COPD, might result in CO2 retention. CO2 is then hydrated with the formation of carbonic acid that subsequently dissociates with release of hydrogen ions (H+) in the body fluids according to the following equation

$$CO2+H2O \Longrightarrow H2CO3 \Longrightarrow -HCO3+H+.$$ [1]

As a result of the altered gas exchange, hypercapnia in COPD patients leads to a rise in H+ concentration and the development of respiratory acidosis, also known as hypercapnic acidosis.[7] According to the Henderson-Hasselbach equation, it expresses the relationship between pH (logarithm of inverse concentration of H+), bicarbonate ion concentration (−HCO3), and partial pressure of CO2 (pCO2).

MATERIALS AND METHODS

The study was conducted in Department of Biochemistry in association with Department of Respiratory Medicine of Mahatma Gandhi Medical College & Hospital, Jaipur. Total 100 (n=100) patients diagnosed for COPD, visiting the Outpatient Department (OPD) and Inpatient Department (IPD) of Respiratory Medicine fulfilling the inclusion criteria were enrolled for the study.

Patients with neoplastic pathologies, pneumonia and Liver or renal diseases, pregnant and lactating females were excluded from the study.

Blood samples for all subjects (acute exacerbation and stable COPD patients) were collected using standard aseptic technique and analysed C - reactive protein by immunorate, Serum uric acid by uricase and Serum Bicarbbonate by enzymetic method. Using Vitros 5600- Dry Chemistry analyser.

The results obtained shall be presented as mean + SD and subjected to statistical evaluation. A p-value of < 0.05 shall be considered as statistically significant.

Table1: Distribution of variables between COPD patients and Healthy subjects.

Parameters	COPD patients	Healthy subjects	t-value	p-value
Age (years)	56.14 ± 10.10	49.44 ± 12.71	2.91	0.004

Serum Bicarbonate (mmol/L)	28.65±8.50	22.75 ± 3.06	4.61	<0.0001
C-reactive protein (mg/L)	12.72±9.66	7.33 ± 4.81	3.52	<0.0001
Uric Acid (mg/dL)	6.75 ± 2.42	5.70 ±1.76	2.44	0.008

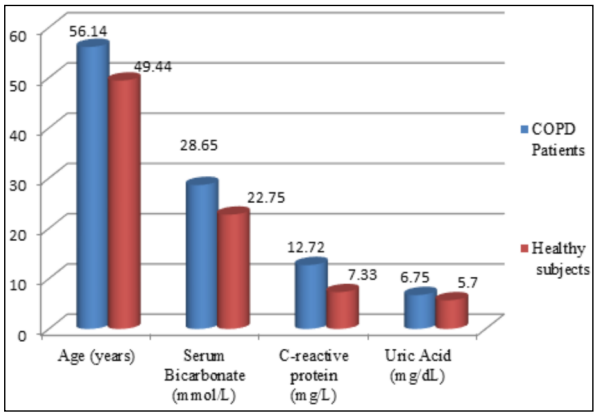

Figure 1: Distribution of variables between COPD patients and Healthy subjects.

RESULT:

Parameters evaluated in the COPD patients and healthy subjects are present in (Table 1). The mean level of age was significantly increased in COPD patients (56.14 ± 10.10) as compared to healthy subjects 49.44 ± 12.71 (p = 0.004).

The mean level of Serum Bicarbonate were significantly higher in COPD patients (28.65±8.50) as compared to healthy subjects (22.75 ± 3.06) (p = <0.0001). C-reactive protein were significantly higher in COPD patients (12.72±9.66) as compared to healthy subjects (7.33 ± 4.81) (p = <0.0001).

The mean level of Serum Uric Acid level was significantly higher in COPD patients (6.75 ± 2.42) as compared to healthy subjects (5.70 ±1.76) (p = 0.008).

DISCUSSION

Chronic obstructive pulmonary disease (COPD) is a lung disorder characterized by persistent airflow limitation that interferes with normal breathing and is not fully reversible Pauwel et al., 2001. It enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases.[8]

Carbon dioxide (CO2), derived from oxidation of nutrients containing carbon, are produced. Under normal conditions, the production of CO2 is removed by pulmonary ventilation healthy subject. However, an alteration in respiratory exchanges, as occurs in advanced phase of COPD, results in retention of CO2. Carbon dioxide is then hydrated with the formation of carbonic acid that subsequently dissociates with release of hydrogen ions (H+) in the body fluids according to the following equation:

$$CO2+H2O \Longrightarrow H2CO3 \Longrightarrow -HCO3+H+.(1)$$

The metabolic disorders and respiratory disorders lead to alteration in bicarbonate and pCO2 respectively. The body tries to maintain and minimize changes in the pH by kicking in the compensatory mechanisms to keep the bicarbonate/ pCO2 ratio constant. The compensation can be predicted to some extent and is based on primary metabolic or respiratory disorder.

Leuzzi G et. al, 2017 concluded their study that Systemic inflammation plays an important role in COPD pathogenesis and disease progression. CRP levels could be used, in combination with other biochemical markers, to target preventive and therapeutic strategies in COPD disease.[9]

Patients with stable COPD have increased CRP levels when compared with healthy controls, and patients with severe COPD had higher serum CRP levels then those with moderate COPD shown by Pinto-plata VM et. al, 2006.[10]

Serum uric acid is a widely and rapidly available, easy to interpret, low–cost biomarker, suggest a possible role for serum uric acid in the identification of COPD patients at an increased risk of adverse outcomes who may need early intensive management reported by Embara S et. al, 2019.[11]

Some other studies found a strong correlation between hyperuricaemia and COPD. An imbalance between oxidant/antioxidant statuses favors oxidative stress induced injury of the airways. Pro-inflammatory effect of UA with increased serum concentration has also been proposed as a cofactor in pathogenesis of COPD. Pro-oxidant effects of uric acid with raised level in free-radical generation, inflammation predominates over its antioxidant effects. Significantly increased serum UA levels among COPD cases compared to controls (p<0.05) Aida Y et al., 2008.[12]

Conclusion: The study concluded that in COPD patient's high levels of Bicarbonate, uric acid and CRP was observed when compared with healthy patients. Bicarbonate levels could be the individual biomarker which can assessed the respiratory acidosis, which is also called hypercapnic. Other than that CRP and Uric Acid levels judges the severity the disease. However, for selection of appropriate treatment protocol and identification of newer marker can be used for convenient evaluation. By the regular monitoring of these biomarkers the mortality and morbidity rate can be controlled.
The study further suggests that correlation of these markers in acute exacerbation and stable COPD patients.

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