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A study on microalbuminuria and serum c-reactive protein and its correlation with disease severity in patients with chronic obstructive pulmonary disease

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Abstract

Background: We will be hypothesized that Microalbuminuria and CRP is elevated in patients with COPD independently of other cardiovascular risk factors. To test this hypothesis, we will be determine the prevalence of Microalbuminuria and high CRP in a group of patients with COPD.

Methods: This study was undertaken with the approval of the Institutional Ethics Committee of S.P. Medical College and PBM Hospital, Bikaner, and written informed consent was obtained from each participant prior to participation in the study and the sample collection process. All subjects were evaluated by taking a detailed clinical history.

Results: Maximum patients (68.00%) were from grade 4 followed by 32.00% patients were from grade 3. Maximum patients (52.00%) were from GOLD stage 4followed 28.00% patients were from GOLD stage 3, 14.00% patients were from GOLD stage A and 6.00% patients were from GOLD stage B. Among the study population, more than three fourths of the subjects, 82.00% of the subjects had Micro Albuminuria. Mean CRP level was 16.32±2.97 mg/dl.

Conclusion: We conclude that systemic inflammation is present in COPD patients and CRP is an important biomarker in COPD in means of reflecting disease severity of patients.

Keywords: COPD, CRP, Micro Albuminuria

Introduction

Chronic obstructive pulmonary disease (COPD) a preventable and treatable disease which consists of constant expiratory limitation of the flow of air which is not fully reversible. [1]

In COPD patients increased CRP levels are associated with poor lung function, reduced exercise capacity and worse quality of life as well as being a significant predictor of all-cause mortality. As well as COPD itself, smoking, which is the most commonly encountered risk factor for the disease is also responsible for rise in serum CRP levels. Though to our knowledge the effect of biomass exposure, potentially initiating inflammatory process in the lungs of COPD patients, on serum CRP levels has not been studied previously.[2]

Microalbuminuria (MAB) is the condition in which excretion of albumin in the urine is elevated at amounts not detectable by conventional semiquantitative tests. MAB is now measured by albumin (μg) and creatinine (mg) ratio in a random urine sample.[3]Currently, the National Kidney Foundation recommends to use spot urine albumin /creatinine ratio in the first voided, morn ing, and midstream specimen to detect album inuria.[4]

We will be hypothesized that Microalbuminuria and CRP is elevated in patients with COPD independently of other cardiovascular risk factors. To test this hypothesis, we will be determine the prevalence of Microalbuminuria and high CRP in a group of patients with COPD.

Materials and methods

Study design

Hospital based cross-sectional observational study.

Study duration

6 Months.

Study place

Department of Respiratory medicine, S.P. Medical College and P.B.M Hospital, Bikaner

Sample size

A sample size calculation show that 30 patients were required in each group according to high urine ACR (urine ACR >30 mg/gm) was 9.1% in Group A, 13.3% of Group B, 16.7% of Group C, and 26.9% of Group D as per reference article Gupta K et al at.

Sampling Method

Systematic random sampling

Inclusion criteria

1. Those who will give informed consent
2. Diagnosed cases of COPD

Exclusion criteria

1. Patients with any other acute or chronic infections.
2. Patients with Diabetes Mellitus, Hypertension, CAD
3. Patients not giving informed consent.

Data Collection

This study will be undertaken with the approval of the Institutional Ethics Committee of S.P. Medical College and PBM Hospital, Bikaner, and written informed consent will be obtained from each participant prior to participation in the study and the sample collection process.

All subjects were evaluated by taking a detailed clinical history. The duration of COPD, number of exacerbations, symptoms and treatment history was enquired from each patient enrolled in the study with detailed systemic examination of each patient. Routine blood investigations were done with specific investigations such as urine routine microscopy, MAB, UACR, spirometry, CRP, skiagram of chest in posterior anterior view. For the diagnosis of MAB, care was taken when collecting samples for the urine UACR. An early morning sample was preferred. The patient was instructed to avoid heavy exercises 24 h before the test. In our study we labelled a patient to have MAB when the urine ACR was more than 30 mg/gm. Patients with COPD of different groups were compared with urine ACR values.

Data Analysis

To collect required information from eligible patients a pre-structured pre-tested proforma will be used. Data will be collected and will be analysed by required statistical test.

Results

Table 1: General characteristics

Mean age in yrs	63.21±10.12 yrs
Male: Female	87:13
BMI	21.36±1.36 Kg/mt^2
Smoker	58.00%
Mean pack/yr	24.44±6.23
mMRC grade (3:4)	32:68

Gold stage (A: B:C:D)	14:6:28:52
Micro Albuminuria present	82.00%
CRP	16.32±2.97 mg/dl

Maximum patients (68.00%) were from grade 4 followed by 32.00% patients were from grade 3. Maximum patients (52.00%) were from GOLD stage 4followed 28. 00% patients were from GOLD stage 3, 14.00% patients were from GOLD stage A and 6.00% patients were from GOLD stage B. Among the study population, more than three fourths of the subjects, 82.00% of the subjects had Micro Albuminuria. Mean CRP level was 16.32±2.97 mg/dl.

Discussion

Our study revealed that MAB was present in 82.00% of patients with COPD which is contrast to the result of previous study done in 2015 (24%).[5]

Urinary ACR was increased in the subjects who were more symptomatic and had high risk (26.9%) as com pared to the patients who had less symptoms with low risk (9.1%) which was similar to the study done in 2017. [6]There was a significant difference in the urine ACR between the groups when they were categorized on the basis of FEV1, history of exacerbations and CAT.

The aforementioned results of our study are supported by previous studies who found that smoking induces albuminuria.[7] Smoking decreases renal blood flow, causing decline in the glomerular filtration rate (GFR). A decrease in the GFR will enhance the renovascular resistance causing renal arteries to be thickened and hence further decreasing the renal blood flow. Therefore, in persons exposed to cigarette smoke with a normal GFR, the filtration rate decreases. These transient multiple renal hypoperfusion episodes damage some glomeruli leading to structural changes such as hyperfiltration leading to MAB along with hypertrophy of remaining glomerulus.

Thus, we observed that patients with COPD who were more hypoxic and more hypercapnic had more MAB than compared to COPD patients without MAB which was statistically significant because hypoxia results in endothelial dysfunction due to loss of physiological equilibrium of vasodilation and vasoconstriction which results in loss of peritubular capillaries in tubule inter stitium.[8] As MAB is now used as a screening tool for patients of cardiovascular risk, patients with COPD should be regularly advised for urine routine microscopy to detect MAB which will alert them for development of further cardiovascular events and hence take necessary precautions and treatment for the same. There is a possibility to assess a therapeutic role of blockers of RAAS system in reducing cardiovascular events in COPD.[9]

The main finding of the present study is that CRP levels are raised in stable COPD patients independent of smoking behavior and history of biomass exposure. Augsti et al also demonstrate higher CRP levels were related to low FEV1% predicted, SpO2 and 6MWD and to high MMRC levels among the prognostic predictors of the disease, in concordance with the previous reports [10] and moreover indicate that serum CRP levels are most strongly related to BODE index and concomitant systemic hypertension.

With the growing awareness of COPD being a complex disease involving several organs with a clearly established low-grade systemic inflammation, biomarkers have been more focus of interest in clarifying the pathogenesis and progression of COPD as well as in designing new therapeutic targets for the disease. Elevated serum CRP levels indicating a low grade persistent systemic inflammation in COPD patients was first described in early 2000's. [11-12] Then direct relation ship between CRP levels and important prognostic

clinical variables in stable COPD patients was reported in 2006 by de Torres *et al.*.[13]They published that CRP levels in stable COPD patients are associated with arterial oxygen tension, 6MWD, FEV1, FVC, inspiratory capacity/total lung capacity, GOLD stage of the disease and BODE index

Conclusion

Microalbuminuria is significantly associated with GOLD severity staging and acute exacerbations. The deter mination of microalbuminuria is simple and inexpensive. These markers can serve to diagnose in COPD patients in resource limited and emergency setting. This study suggests that endothelial and microvascular mechanisms are promising targets for early detection of COPD and management. Systemic inflammation is inherent to COPD independent of ever-smoking status and correlates with disease severity, concomitant systemic hypertension and pulm onary hypertension. We conclude that systemic inflammation is present in COPD patients and CRP/MAB are the important biomarker in COPD in means of reflecting disease severity of patients.

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