Microalbuminuria in Chronic Obstructive Pulmonary Disease with and Without Co-morbidities

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Dear Sir,

A study published in the first issue of volume 33 (2018) of your journal has drawn our attention which reports of microalbuminuria as an early nephropathy marker in diabetes, hypertension and pregnancy induced hypertension. We have conducted a similar study at our centre as a part of ICMR Short term studentship in Chronic Obstructive Pulmonary Disease patients without exacerbations and got encouraging results in a small sample size (feasible for the time duration of the project of 2 months). COPD patients often have other chronic coexisting diseases which may include hypertension (HTN), hypercholesterolemia, Coronary artery disease (CAD), diabetes mellitus (DM), osteoporosis, depression and cancer. Recently the link between COPD and co-morbid conditions has caused a great deal of interest [1]. Cardiovascular disease (CVD) is a leading cause of death in patients with COPD [2]. Airflow limitation as measured by FEV1 or FEV1/FVC ratio is a predictor of cardiovascular risk [3]. Patients' with COPD have systemic manifestations that are not reflected by the FEV1 [4]. Therefore a non-invasive yet good marker of early endothelial dysfunction like MAB can prove highly beneficial. MAB is a predictor of cardiovascular events and all-cause mortality in the general population. It has a stronger association with cardiovascular events and death than C-reactive protein and reflects a state of generalized endothelial dysfunction [5]. Earlier studies have reported

an association of MAB with COPD in patients with acute exacerbation and with co-morbidities. We hypothesized to assess the prevalence of MAB in COPD patients without acute exacerbations, in presence as well as in absence of co-morbidities and assess the association of MAB. Our findings, possibly for the first time report of MAB frequency in COPD patients without acute exacerbations in presence as well as in absence of associated co-morbidities. We found an increased frequency of MAB in these COPD patients without acute exacerbations and a positive correlation of MAB with blood pressure. The frequency of MAB in COPD patients was 81% (n = 25). Most COPD patients reported at GOLD'S CLASSIFICATION stage II or stage III, which is a matter of concern. We observed in COPD patients without any co-morbidity and acute exacerbations (group I) to have a significantly higher prevalence of MAB i.e. 87%, as compared to COPD patients with co-morbidity (group II) (75%), suggesting MAB maybe a potential independent marker that can be used in COPD patients CVD risk assessment even in absence of any associated co-morbid condition. A Pearson's correlation analysis showed the significant positive correlation of DBP with MAB and non-significant positive correlation of FEV1%, BODE, BMI, SBP, WHR. Most of the COPD patients were at high severity and had positive association of MAB with FEV1% and BODE index, although a non-significant. The low significance of MAB with FEV1% and BODE index could be owing to the low sample size of the study. Further larger prospective studies in these patients can help in better understanding of the association of MAB with these parameters. Thus, in theory, MAB could be a promising biomarker to identify patients with COPD at increased cardiovascular risk, even in absence of co-morbidities. More carefully obtained longitudinal data of MAB and its relationship with others biomarkers and

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cardiovascular risk factors can support a potential role of MAB as a target in the management of patients with COPD should be considered.

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