Comments on "Value of past clinical history in differentiating bronchial asthma from chronic obstructive pulmonary disorder in male smokers presenting with shortness of breath and fixed airway obstruction"

Sir,

We read with great interest your article "Value of past clinical history in differentiating bronchial asthma from COPD in male smokers presenting with shortness of breath (SOB) and fixed airway obstruction".[1] Authors have highlighted the importance of past history in differentiating asthma from chronic obstructive pulmonary disorder (COPD). But, recent insight into current evidence emphasizes the need for correct classification of asthma and COPD patients by recognizing the subgroup with overlap syndrome.[2]

Traditionally asthma is considered a childhood/early adulthood onset disease with reversible airflow limitation while COPD is considered to manifest after 40 years of age and has irreversible or partially reversible airflow limitation. The pathogenesis of COPD has shown that it has much early origin than previously thought. *In utero*/childhood smoke exposure and decreased lung growth in early adulthood are linked to increased risk of COPD at a later age.[3] At one end COPD patients may have partially reversible airflow limitation and at the other end asthmatics may show partially irreversible obstruction due to airway remodeling by long-term disease process.

Cytokines play a complex role in pathogenesis of asthma and COPD. According to textbook teaching COPD patients show neutrophilic inflammation while asthmatics show eosinophilic inflammation in their airway but a growing body of evidence is reporting existence of overlap between asthma and COPD. GINA guidelines have recently defined Asthma-COPD Overlap Syndrome (ACOS) as a distinct entity.[4]

Neutrophilic inflammation does not respond to corticosteroids while eosinophilic inflammation is corticosteroid responsive. Sputum eosinophilia has been reported in 20-40% of sputum samples from stable COPD patients also.[5] Airway inflammation in asthmatics is mediated by Th2 cells which express signature interleukins (IL) for allergic (eosinophilic) inflammation i.e. IL-4, 5 and 13.[6] Bafadhel *et al*. reported that increased sputum IL-5 levels were associated with sputum eosinophilia in COPD patients which was attenuated by oral corticosteroid therapy.[7]

Michael *et al*. reported three biological clusters on the basis of sputum cytokine profile of 86 asthmatics and 75 COPD patients.[8] Cluster 1 consisted of mainly asthmatics and had elevated sputum Th2 cytokines and eosinophils. Cluster 2 comprised both asthma and COPD patients with sputum neutrophil predominance. Cluster 3 had mainly

COPD patients. This group had mixed neutrophilic and eosinophilic inflammation.

In the light of current evidence, we therefore, emphasize that classification of the patients should be based on pathobiology rather than clinical phenotypes. Such classification is important not only for optimization of currently available therapies but also for enrolment of patients in future trials for upcoming therapies like anti-interleukin 5 monoclonal antibodies.

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DOI: 10.4103/0970-2113.159637