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Defining Chronic Obstructive Pulmonary PR Gupta **Disease-GOLD** is still Gold

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Global initiative for chronic obstructive pulmonary disease (GOLD) [1] has defined chronic obstructive pulmonary disease (COPD) as a common preventable and treatable disease, characterized by persistent airflow 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. Essential components of above definition of COPD are the chronic inflammatory response and the exposure to noxious particles and/or gases. Accordingly, the diagnosis of COPD is made when someone, who is exposed to the known risk factors, presents with symptoms and his spirometry shows fixed airway obstruction (post bronchodilator FEV_1 to FVC ratio of <0.7).

An ATS/ERS position paper on COPD [2] has, however, defined the disease on the basis of the post bronchodilator FEV₁ to FVC ratio of <0.7 alone. As a result, several researchers [3-7] have used this parameter alone to diagnose COPD in their patients, even though they did not have risk factors for COPD and had asthma (or bronchial hyper-responsiveness) during child hood, thus ignoring the different etiological factors and the resultant airway inflammation therein, of the 2 diseases. This has led to erroneous conclusions like the one made by Tai et al. [7], namely "The association between childhood asthma and adult COPD". These authors have considered the history of childhood asthma as equivalent to failure of lung growth, a minor risk factor in the aetio-pathogenesis of COPD.

In the eye of this author and may be, several others, the above definition of COPD is crippled one as it undermines the importance of the risk factors and the resultant chronic inflammatory response, typical of the disease. Zeki et al. [8] has reviewed the literature on overlap syndrome and questioned that the natural history of asthma may get altered with airway remodeling or in the presence of certain confounders, i.e. smoking, air pollution and these patients might ultimately present with fixed airway obstruction but should they be called as suffering from COPD? Logically also, a patient who have asthma during his childhood should have asthma during adulthood, or at best, an "overlap syndrome", rather than COPD. Even GOLD has considered the issue, as controversial. Further, Fabbri et al. [9] have found **Corresponding author: PR Gupta**

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important differences between the airway inflammations in the patients with COPD with that in asthma.

Traditionally, steroid trials have been used to differentiate asthma and COPD in such difficult clinical situations, where a fixed airflow limitation persisting after a short course of systemic steroid therapy was considered as diagnostic of COPD but a positive response was considered as diagnostic of asthma [10].

This author along with others [11] reasoned that COPD and bronchial asthma pursued a different natural history, at least in the initial phases i.e. patients with COPD pursue a progressive downhill course, often worsened by exacerbations but asthma is characterized by an intermittent course, marked with frequent exacerbations and remissions. Further, history of atopy in self or family, in the form of skin, eye or nasal symptoms and/or wheezing is the hallmarks of asthma rather than COPD. Although, this natural history of asthma may get altered in due course yet the past history may continue to guide the diagnosis of asthma in these patients. We, accordingly, exploited the past history of allergic symptoms in male smoker patients presenting with shortness of breath along with post bronchodilator FEV, to FVC ratio of <0.7 (fixed airflow obstruction) to differentiate asthma from COPD. It was found out that past history of respiratory or non-respiratory allergic symptoms favored the diagnosis of bronchial asthma rather than COPD and such an approach was at least as effective as the steroid trial. On the basis of the above study, it could be inferred that the presence of asthma during childhood should lead to asthma during adulthood, rather than COPD and the presence of fixed airway obstruction, alone, should not be taken as diagnostic of COPD.

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Defining all the patients presenting with fixed airway obstruction (post bronchodilator FEV_1 to FVC ratio of <0.7) as COPD is thus unjustified. Many of these patients are truly the cases of severe

persistent asthma or "overlap syndrome". The confusion is best avoided by strict adherence to the GOLD definition of COPD. GOLD is still gold.

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