The new GINA and GOLD guideline: do they lead to more confusion ?

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It is interesting to note that the GOLD definition of COPD is changed radically shaking off the long borne and painfully developed concept and understanding of the disease. In one blow the experts have discarded the whole reversibility issue and choose to embrace a definition to include post bronchodilator FEV1/FVC ratio less than 70 % as the sole criteria of diagnosis of COPD(1,2). This has led to serious questions about a) the scientific basis of such a change and b) the purpose of such radical simplification.

Firstly, FEV1/FVC ratio itself is age dependent (3,4). Since it falls with age, there is a clear risk of overdiagnosis of COPDin elderly people in whom the ratio is less than 70 % (4). Even for the sake of agreement, if one takes the ration as a sure reflection of airflow limitation, the gamut encompasses a whole lot of obstructive airway diseases as asthma, bronchiectasis, and of course, ACOS and COPD. On top of that, the very omission of reversibility to differentiate asthma from COPD keeps one baffled. It is a shock to those who has been trying to learn the obstructive airway disease for years with the help of GINA and GOLD initiatives. A simple question irks the mind that do the experts have discovered a definite and easily usable and available biomarker to differentiate the two conditions across the globe or do they have unshakable evidence that the age old Dutch hypothesis (that COPD and asthma are same disease) is the final word in diseases of airflow limitations or do they have evolved any special hypothesis like COPD may often beget asthma or FEV1 reduces in asthma without changing the ratio of FEV1/FVC (a difficult mathematics, but possibly applicable in reversibility over 200 ml and 12% with FEV1/FVC ratio above 70 %) or something beyond the appreciation of our poor common sense?

With this great development, we fail to understand how to fit together an eosinophilic desquamative inflammation with type-1hypersensitivity with a neutrophilic airway inflammation from inhalation of noxious gases. Here, we descend to sip at a concoction of two distinct and different identities as their pathologies and pathogenesisare concerned (4). Frankly, one finds it difficult to equate the protease upregulation with an allergic eosinophilic inflammation. We get further confused when the traditional teaching and understanding of asthma as an obstructive airway disease with features as reversibility, variability, and unpredictability is merged with characteristics of COPD as a progressive and poorly reversible airflow limitation; both of them in course of their natural history may have just an FEV1/FVC as <70 %.

The more confusing part is possibly the flip side of the development. It is really intriguing to speculate or appreciate the reason of such a remarkable change of definition without changing the area of treatment of the two diseases as asthma or COPD. If the reversibility does not matter, the very question of diagnosing them separately is superfluous. Hence, we would like to treat patients with FEV1/ FVC as <70 % but over 200 ml of reversibility with LAMA and LAMA + LABA as we do in COPD and not inhaled corticosteroid the role of which is questioned in the WISDOM and OPTIMA trials in COPD (5, 6). Surely this will cause more non adherence to guideline and will make the scenario further difficult and confusing in future. On top of it, one may get further perplexed to understand the diagnostic criteria of COPD or asthma for research or for clinical trials? The GINA and GOLD pundits could have initiated a global survey on the proposed definition for its validation or acceptability and that would have been possibly the right approach than just giving to swallow the confusion and ponder over its rationality.

Reference:

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