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Editorial

Deflators and Not Bronchodilators in COPD

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Editorial

Chronic Obstructive Pulmonary Disease (COPD) is defined 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". Cigarette smoking is the major risk factor [1,2].

Diagnosis of COPD requires confirmation of airflow limitation that is not fully reversible via spirometry by a fixed low post bronchodilation FEV_1/FVC ratio (<0.70) [2]. Furthermore, in the everyday clinical practice and in order to distinguish COPD from Asthma, a bronchodilation test is performed in order to verify that the changes in FEV_1 are minimal in COPD in comparison to that in Asthma [1,3].

Under the heading of Bronchodilators in COPD are the short and long-acting Beta2-agonist (SABA, LABA) the short and long –acting Anticholinergics (SAMA, LAMA) and the methylxathines. Although, we expect small effect on dilatation of the airway caliper in COPD by using those drugs, we call them Bronchodilators. Indeed, recent guidelines define these medications as Bronchodilators and consider them as the cornerstones of treatment at all stages of the disease [1-3].

All major mega trials investigating the effects of the above medications had shown minimal effect on FEV_1, but significant improvement in Quality of Life, exercise capacity, morbidity, and mortality, as well as, in the frequency of exacerbations [4].

It is obvious that those medications had another significant action (s) rather than their weak bronchodilatory one. Recent studies have shown that their main effect is the reduction of lung volumes [5-8].

It is well known that the major pathophysiological even in the natural cause of the disease is Hyperinflation of the Respiratory System. Hyperinflation is defined as an increase in the End-Expiratory Lung Volume (EELV) and/or an increase in Total Lung Capacity (TLC) above 120% predicted and considered to be static and dynamic [9]. Hyperinflation puts the ventilator pump in a very disadvantage position to produce pressure, especially the respiratory muscles, and increases significantly the work and the energy cost of breathing [9,10]. In addition, hyperinflation has deleterious effects on the cardiovascular system [11].

Although, hyperinflation is the major mechanical defect of breathing in COPD is the only way the patient can increase ventilation when needed, for example during exercise (climbing up stairs) or during Acute Exacerbation. However, hyperinflation has significant consequences, such as, respiratory muscle fatigue and respiratory failure [11,12]. Hyperinflation of the Lungs has been a significant biomarker of survival in COPD and therefore has become an important therapeutic target [11,13].

Fortunately, all inhaled medications called so far, "bronchodilators", have been shown to have a greater effect on reducing hyperinflation than improving FEV_1. This is true during rest but more pronounced during exercise! [11,6,9]

It is obvious, that the main pathophysiological action of this class of drugs is the deflation of the lungs by which they affect dyspnea, quality of life, sleep, exercise capacity and consequently exacerbations, morbidity and mortality. Therefore, it is more relevant to rename them Deflators and not "Bronchodilators". By doing so, we emphasize that hyperinflation is the major mechanical defect in COPD that is targeted by the treatment, and that it is not the bronchodilatory effect but deflation the crucial action of SABA, LABA SAMA, LAMA in COPD.

Thus, it is better to call them Deflators and no Bronchodilators!!

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