

Towards Characterizing COPD Exacerbations

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An exacerbation of chronic obstructive pulmonary disease (COPD) is an acute event characterized by a worsening of the patient's respiratory symptoms that is beyond normal day-to-day variations and leads to a change in medication [1]. They are associated with substantial morbidity and mortality [1,2].

Most exacerbations are caused by respiratory viral, bacterial infections and environmental agents [3-6]. However, there is a remarkable group of patients whose exacerbation cause could not be identified [3,4].

The pathophysiology of the exacerbation of COPD is multifactorial. The most influential factors are inflammation and dynamic hyperinflation. However, it is still unclear which of these two components is the most relevant or if both are involved in all occasions.

Several authors suggest that the concept of exacerbation should include the concept of inflammation, as already happened with the disease definition. Nevertheless Hurst et al. [7] Studied 36 plasmatic biomarkers without obtaining conclusive results so far, only the increase of plasmatic CRP, in presence of a major symptom of exacerbation, was useful to confirm the exacerbation diagnostic.

There are exacerbations which cardinal symptom is breathlessness in absence of the rest of criteria of Anthonisen. Some studies have identified a close association between the presence of dyspnea as the main symptom of exacerbation and the development of dynamic hyperinflation [8].

Bafadhel et al. [9] tried to establish clinical phenotypes of exacerbations, for that, they studied different biomarkers in serum and sputum, identifying three biological clusters of exacerbation that relate to inflammatory patterns perfectly identifiable with the causal pathogens (the ones related with bacterial, viral and eosinophilia inflammation etiology). Nevertheless, they obtained a fourth cluster called pauci-inflammatory showing low concentrations in sputum with limited changes in the inflammatory profile and few events associated with known etiology. This would support the theory of the existence of a group of exacerbations which origin is non-inflammatory.

Washko et al. [10] studied the mechanism and the potential benefit of volume reduction surgery in the exacerbation of patients with COPD in the cohort study NETT (National Emphysema Treatment Trial). The surgical cohort achieved to reduce the number of exacerbations and increases the time to first exacerbation compared with the patients cohort treated with

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optimal medical treatment [11,12]. Therefore, this study suggests that the number of exacerbations could be reduced through the improvement in function and lung mechanic, regardless from the anti-inflammatory effects that could be attained in pharmacological way.

Corticosteroids anti-inflammatory profile has shown potentially reduce the risk of exacerbations, but other clinical studies confirm that LAMA and LABA are also effective in reducing and preventing them. This would be explained by the ability of these drugs to reduce airway resistance and therefore improve secondary lung hyperinflation. Improving inspiratory capacity in turn produces a significant decrease in lung volume at the end of expiration. So probably the improvement in lung mechanics produced by long-acting bronchodilators make patients less vulnerable to different exacerbations triggers.

The above mentioned would lead us to believe that, although the inflammation is critical in the pathogenesis of COPD, no all exacerbations are associated with an increase in inflammation.

Thus, we need further studies to better known en understand the different pathophysiologic mechanisms that occur in exacerbations. As a result each cluster or type of exacerbation will be beneficiary most, from a treatment or other and such treatment could be targeted depending on the type thereof.

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