

Are the Biomarkers Ready to Enter into Guidelines for Exacerbation in COPD?

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Exacerbations in chronic obstructive pulmonary disease (ECOPD) play a major role in the quality of life, prognosis and mortality of these patients. Recently, a large retrospective study Serra-Picamal., *et al.* [1], showed that near 20% of the patients hospitalized for ECOPD die within the following year from discharge and, in the same study, early mortality (< 30 days) was linked to re-admissions. Likewise, it is well established that early recognition of symptoms and prompt therapy in ECOPD patients lead to a faster recovery, reduce risk of hospitalization, and it is associated with a better quality of life [2].

On the other hand, despite several years have passed since the article of Bafadhel., *et al.* [3], conclusive proved a logical truth: the exacerbations in COPD are heterogeneous and have different etiological triggers, we continued using a variation the symptom-based definition by Anthonisen., *et al.* [4], combined with a healthcare-based definition [5] for most of our guidelines and in our daily practice [6-8]. The major problem is that these definitions are pragmatic approaches, which often oversimplify the pathogenic pathways implicated in an ECOPD.

The identification of different phenotypes in ECOPD is a step forward to the findings of prognosis and therapeutic strategies directed to improve care and avoid the risks of unnecessary therapies. In studies of cluster analysis of ECOPD [3,9], patients are sorted either by predominant type of cells in sputum (eosinophilic, neutrophilic, mixed granulocytic and paucigranulocytic) [9], or by type of etiology [3] (viral, bacterial, mixed and pauciinflammatory) and all of them showed different response to treatment and prognosis.

Furthermore, there is evidence that the patients with undergoing exacerbations have dynamic changes in the sputum bacterial microbiome [10] and this disbalance is correlated with inflammatory makers and clinical parameters [11] (FEV1). Interestingly, the authors suggested evaluation of the bacterial ratio as guidance for antibiotic use, because the lack of change in the microbiome during the exacerbation could indicate a non-bacterial exacerbation etiology [11].

Antibiotic treatment is a critical aspect of ECOPD. Several studies demonstrated the inadequate over-use of antibiotics in exacerbations. Currently, this is guided by the purulence of sputum [12] or self-reported sputum color [13] with mixed results. However a recent meta-analysis [14] found that protocol procalcitonin-based for the use of antibiotics (with a cutoff above $0,25\alpha g/L$) could be helpful, limiting the antibiotic prescription without worsening the clinical outcomes. This is a promising and objective tool to discriminate which patients need antimicrobial therapy.

So, the answer to the question: Are the biomarkers ready to enter into guidelines for exacerbation in COPD? The response is.... not yet!. Currently, there are not biomarkers ready for the clinical practice, because we do not fully understand the pathophysiology underlying exacerbations on COPD. Although, newer studies [15] are focusing in networking medicine that hopefully could connect the dots in this picture and make a clearer vision into the heterogeneity of exacerbations. We must demand a more precise definition of exacerbations in COPD. The entire new proposal for a definition has to be done with a multilevel system that includes biomarkers for the definitions, microbiology, comorbidities and for guiding the therapeutic approach; all these framed by a patient-centered clinical context.

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Lastly, I think that it is time embrace the precise medicine in ECOPD, because the upcoming evidence is directed to eliminate the "onesize fits all" approach and we need to walk into the future with our eyes wide open.

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