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Perspective

The Role of Microbiota in COPD Exacerbations

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Abstract

Microbiota is the community of microbes living in and on an individual. Host and flora live in a symbiotic relationship. Lower respiratory tract infections remain a major cause of morbidity and mortality. COPD patients, namely in Gold stages B and D, are extremely prone to these infections but several of them are viral in etiology, in which antibiotics are useless. To reduce unnecessary use of antibiotics testing of C-reactive protein should be performed. There is also evidence that testing for blood procalcitonin can differentiate bacterial from viral disease, what would help the use of antibiotics to be more rational.

Keywords: *Microbiota; COPD Exacerbations; Antibiotics*

According to Murray P [1], microbiota or normal flora is the community of microbes that live in and on an individual. It can vary substantially in health and disease between environmental sites and host niches.

The term microbiome refers to the aggregate collection of microbial genomes in the microbiota. For example, gut bacteria have 150 times more genes than a human being.

In the microbiome the core and secondary microbiome can be considered. The first compromise the largest part of the microbial population, in spite of being represented by a limited number of species. The secondary microbiome is the microbial species that contribute to the unique diversity of individuals at specific body sites and are present in small numbers.

The host and the microbial flora live in symbiotic relationship. The host provides the Terrain, defined as a place to colonize, the nutrients and some protection from unwanted species. Bacteria provide needed metabolic functions, stimulate immunity and prevent colonization with unwanted pathogens [2].

The composition of our microbiota is influenced by several factors like personal hygiene, diet, water source, exposure to environmental toxins and medicines (especially antibiotics).

The environment is populated with bacteria, the most part of them are relatively avirulent, some others are capable of producing life-threatening diseases. Namely, when they invade normally sterile tissues or fluids, but also when they produce substances called toxins, damaging tissues or causing organ failure. Currently we know five thousand types of bacteria, but just around two hundred (4%) cause disease [3].

Zumla A stated in his paper [4], lower respiratory tract infections remain a major cause of morbidity and mortality worldwide, despite advances in the identification of etiologic microorganisms and availability of effective antibiotic therapy.

If we wipe out bacteria using antibiotics we also wipe out the microbiota balance and since this balance prevent colonization with unwanted pathogens, we turn more susceptible to new infections. The disruption of the normal flora is called dysbiosis and can lead to disease by elimination of needed bacteria or allowing the growth of pathogenic bacteria.

The irrational use of antibiotics caused the bacterial resistance problem, a world health urgency that is compromising the progress of modern medicine, as the general director of WHO stated in September 2017. Bacteria that are resistant to antibiotics will be selected and will endure.

Lung defenses are dependent on a complex array of mechanical, innate and acquired immune defense mechanisms and any disturbance of this internal milieu (the Terrain) results in serious consequences [4].

A large proportion of pulmonary infections seen in medical practice today predominantly occur due to disturbance in the Terrain.

Chronic obstructive pulmonary disease (COPD) is a chronic progressive obstructive lung disease characterized by long term breathing problems, poor air flow, physical activity limitations (with or without muscle wasting) and acute exacerbations (sudden worsening of symptoms). In 2015 it affected around 175.000.000 people in the world (2.5% of global population).

An acute exacerbation of COPD is defined as increased shortness of breath, signs of increased work of breathing, increased cough or sputum production, or change in color of the sputum [5].

The primary risk factor for COPD globally is tobacco smoking [6]. Other factors like air pollution, occupational exposure (workplace dusts, chemicals and fumes) and genetics are also important.

Infections appear to be cause of 50 to 75% of causes (bacteria in 30%, viruses in 23%, and both in 25%) [7-9]. Those with many exacerbations have a faster role of deterioration of their lung function [10]. Those with more severe underlying disease have more frequent exacerbations [11].

Furthermore, as Monsó pointed out [12], although the respiratory flora as a whole only changes in about half of COPD exacerbations, the relative abundance of genera that include specific respiratory pathogens increases significantly compared to previous samples obtained from the same patients when they are stable. The sensitivity of conventional bacterial culture for identifying the causative agent of an exacerbation in COPD is limited.

The Gold guidelines suggest dividing COPD patients into four categories (A, B, C, D) based on symptoms assessment and airflow limitation [13]. Stages B and D are more inflammatory and so more prone to acute exacerbations.

Butler CC., *et al*. published in the NEJM an interesting article [14] stating that testing of C-reactive protein (C-RP) may be a way to reduce unnecessary use of antibiotics without harming patients who have acute exacerbations of COPD. They concluded that C-RP guided prescribing of antibiotics, for exacerbations of COPD in primary care clinics, resulted in a lower percentage of patients who reported antibiotic use and who received antibiotic prescription from clinicians, with no evidence of harm.

In critical care patients Pereira MA., *et al*. [15] demonstrated in their study that early C-RP is not associated with response and prognostic assessment in infected ones. Nevertheless, a fast response pattern tends to exclude initial inappropriate antibiotic therapy.

Theoretically, we could do even better. Since C-RP may be raised in viral as well as bacterial exacerbations of COPD (more than 23% are viral). We could test blood samples of these patients for procalcitonin since there is evidence that this test can differentiate bacterial

from viral disease [16]. According to this assumption the prescription of antibiotics would be even more rational and the issue of bacterial resistance would be minor. Naturally, further studies have to be conducted to testify it.

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