EDITORIAL

Chronic Obstructive Pulmonary Disease: an Ambiguous Name and an Obstacle in the Campaign Against Smoking

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The term chronic obstructive pulmonary disease (COPD) is probably one of the most unfortunate names in modern medicine and, although it is widely used in public health, not all specialists accept it. The authors of one of the most prestigious medical textbooks even prefer to avoid the term.[1] What is clear—and quite regrettable—is that the term COPD is not clearly understood outside the world of medicine and most COPD patients cannot accurately specify the origin or the course of their disease. Because the term is poorly defined, health care professionals find it difficult to explain a diagnosis with such a long, ambiguous name, and which is based on spirometric findings. It seems unreasonable that such a widespread disease with such a high mortality rate should have a name so difficult to communicate to society, to the media and, what is worse, to health authorities themselves. This scenario is what led Claude Lenfant, in his prolog to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) document, to lament the lack of broad social recognition of the disease.[2] In Spain attention has also been drawn to the lack of knowledge of COPD among medical practitioners.[3]

The poor definition of COPD has also led to misuse of the term in nonspecialized medicine, in fact turned it into an umbrella expression that can encompass any case of chronic respiratory disease in an adult. This situation has led to overuse of the diagnosis of COPD in general medicine, especially in emergency departments. For example, a third of one group of British patients who received diagnoses of COPD from primary care physicians did not, when evaluated in specialized centers, fulfill the criteria of airflow obstruction, and many had cylindrical bronchiectasis.[4] Moreover, the lack of consistency in classification gives rise to considerable difficulties when comparing research on COPD and carrying out bibliographic searches.[5]

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The Origin of the Term COPD

At first, the term COPD comprised a group of diseases with the common criterion of airflow obstruction but without pathological and etiological unity. Definitions of diseases based on function or pathogenesis facilitate treatment (for example, the prescription of bronchodilators), but they hinder prevention. This is precisely one of the problems with the term COPD. Initially 4 distinct disease entities were grouped under the category of COPD—chronic bronchitis, pulmonary emphysema, asthma, and bronchiectasis—and were represented by Venn diagrams for didactic purposes. These diseases do not always present with obstruction, which was limited to the superimposed area of the Venn diagram reflecting true COPD. This was indeed a complex situation: at least 4 distinct diseases with diagnostic overlap and with obstruction occurring in only a certain percentage of each. Cases where obstruction presented were true COPD. After the Ciba Symposium, bronchiectasis was excluded and COPD was basically defined as an entity characterized by progressive bronchial obstruction—especially the type that occurs in smokers—the natural history of which was outlined by Fletcher et al.[6,7] Although a small minority of nonsmoking COPD patients with progressive obstruction were identified early on, in light of present knowledge such patients could have been differentiated from COPD patients and categorized in a miscellaneous group that included bronchiolitis obliterans.

The Impact of Present Knowledge and New Technologies on the Concept of COPD

Conceptual, therapeutic, and technological advances have clarified the picture by individualizing the various diseases that can produce progressive airflow obstruction. However, it was the development of high resolution computed tomography (HRCT) that has allowed many of the limitations and intrinsic inconsistencies of the term COPD to be corrected.

The decrease in the prevalence of residual tuberculosis and the correct treatment of pneumonia have led to a spectacular decrease in bronchiectasis, such that it has become restricted only to cystic fibrosis and the infrequent syndromes of immunodeficiency and ciliary dyskinesia, which are well defined entities subject to precise diagnosis. However, this epidemiologic situation is far from being attained in many countries with poorly developed public health care systems, where bronchiectasis should remain in the group of diseases considered in the differential diagnosis of COPD. HRCT has become the gold standard diagnostic technique and has enabled clear-cut distinction between bronchiectasis and the COPD diseases. Nevertheless, many patients who are clinically diagnosed with COPD turn out to have bronchiectasis.[4]

The improved prognosis of asthma brought about by inhaled corticosteroids and the combined effects of international guidelines, standardization of bronchodilation testing, and quantification of bronchial hyperresponsiveness means that asthma (as with bronchiectasis) has been restricted so that there is almost no overlap with COPD diseases. Moreover, the few asthma patients who might pose problems of diagnostic ambiguity with the COPD group—patients with severe asthma, slight spirometric reversibility, and poor response to conventional treatment—also present characteristic HRCT findings: thickening of subsegmental bronchial walls in proportion to the severity of the disease and preservation of the pulmonary parenchyma.[8,9] Such findings are interpreted as radiographic manifestations of tissue alteration known as bronchial remodeling. Asthmatic smokers pose a much more complex diagnostic problem, and bronchial hyperreactivity has been identified as an additive risk factor for accelerated loss of lung function and as a cause of bronchial damage. Within this disease group techniques such as HRCT and the single-breath carbon monoxide diffusing capacity test can probably give an accurate account of the role the 2 risk factors play in airway obstruction.

So the definition of COPD has come to comprise only chronic bronchitis, pulmonary emphysema, and so-called bronchiolitis obliterans, all of which are the result of prolonged, intense exposure to tobacco smoke. As a result, this is implicitly assumed by international consensus and the most recent reviews of the literature.[2,10-12] The term COPD is no longer used to refer to a group of diseases with superimposed limits; it denotes a more specific entity. However, pulmonary emphysema is not a uniform disease in terms of clinical manifestations, histopathology, or cause. Its histological differences are related to etiology. Nowadays it is understood that panacinar emphysema affects intrinsic mechanisms of pulmonary homeostasis and is not necessarily linked to inhaled toxins, while centriacinar emphysema is commonly associated with inhalation of tobacco smoke.[13] Once again HRCT, from first generation technology on, has proven highly reliable in distinguishing the 2 types of emphysema.[14,15] Moreover, radiographic findings of centriacinar emphysema in smokers seem to precede bronchial obstruction.[16]

Practically all the authors who analyze the etiopathogenic implications of COPD assume that only a small percentage of COPD patients have the disease owing to the aggressive effect of substances other than tobacco smoke—which is to say environmental or workplace contamination. Nevertheless, a careful review of occupational causes of COPD has revealed bias in the assessment of workplace-related COPD and little indisputable evidence.[17] The literature describes a causal relation between centriacinar emphysema and occupational disease resulting from accidental inhalation of high concentrations of cadmium salt vapors—a situation that has been reproduced experimentally. Curiously, the only source of inhaled cadmium that could affect a general population is tobacco smoke.[18] Nor is there evidence that centriacinar emphysema could appear spontaneously or idiopathically, as occurs with panacinar emphysema. A recent statement by the American Thoracic Society estimates that occupational exposures contribute 15% of the population burden of COPD.[19] This is not to imply that occupation is the causal factor of the disease but rather an aggravating cofactor of diverse exposures already known to have a toxic effect on the respiratory system.

The clinical and functional picture of subjects who have been exposed to wood smoke for years is quite consistent with the present concept of COPD. This is not so remarkable given the similarities between the smoke from wood, vegetation, and other biomaterial and the smoke from tobacco—a plant. Toxicity of directly inhaled smoke varies: little dilution occurs when an individual is smoking tobacco compared to some dilution when the individual is breathing in a smoke-filled kitchen. This difference in exposure and perhaps certain more subtle differences related to the composition of the biomaterial could explain epidemiological differences and clinical descriptions. What is clear is that some women who cook over wood-burning fires eventually suffer emphysema, severe obstruction, and cor pulmonale, and die in the same way smokers do.[20]

Therefore, the irreversible and progressive airflow obstruction of centriacinar emphysema is linked in great measure to inhalation of tobacco smoke and in countries with practically no exposure to the smoke of other biomaterials, such symptoms are almost exclusively caused by tobacco smoke, with some additional aggravating factors.

The Concept of COPD in the Campaign Against Smoking

If we reach the conclusion that COPD is a disease secondary to smoking tobacco, it would be beneficial to associate both the cause and the damage in the name of

the disease—along the same line as the diagnosis of alcoholic hepatitis. For example, smoker's emphysema.

In the naming of occupational diseases, taxonomy has gone even farther, with generally satisfactory results, by using the causal agent as the basis of the name of the disease; thus we speak of asbestosis, suberosis, and berylliosis. What would we call COPD if it were a strictly occupational disease? Tobaccosis?

One of the tobacco industry's favorite strategies is the propagation of uncertainty and ambiguity regarding the damage they cause. The term "risk factor" assigned to tobacco addiction in COPD fits perfectly into their orchestration of confusion, especially if tobacco addiction is confused with other environmental aggressions.

There is evidence that including the concept of tobacco dependence in the name of the disease has a favorable impact on the rate of smoking cessation. COPD patients who have been diagnosed with "smoker's lung" have a smoking cessation rate similar to that achieved by intensive rehabilitation programs designed to help people give up smoking.[21] Moreover, a change in the name of the disease could contribute to its diagnosis, its social recognition, and prevention of tobacco dependency.

This reflexion leads us to propose the name COPD be changed to one that includes the term tobacco in order to define a specific disease that is characterized by addiction to nicotine, a specific histological substrate (centriacinar emphysema), and a functional disorder (progressive airway obstruction) that is nearly exclusively found in this context. If patients could be told that they have (pulmonary) tobaccosis, the cause of their disease as well as the way to prevent the epidemic from increasing would be immediately apparent. Tobaccosis would have etiological unity, although its clinical, physiological, and pathological presentation might not be entirely specific. However, this does not have a bearing on the problem of inconsistency in the term COPD. After all, the term tuberculosis nowadays denotes a disease whose unity is the causal agent, not the presentation of tubers or granuloma, fever or exhaustion. Another example is berylliosis, which is named for the causal agent, not for clinical, functional, radiographic, or histological factors, which are indistinguishable from those of sarcoidosis. From the broader perspective, tobaccosis would be considered a multisystemic disease that primarily affects the respiratory system, is frequently manifested by progressive airflow obstruction, and is histologically characterized by centriacinar emphysema and damaged airways.

If we continue as at present, respiratory physicians will be shirking their responsibility to society by hiding a disease and epidemic of tremendous magnitude behind a confusing abbreviated name.

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