Differentiating Asthma from COPD : How and Why?

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ABSTRACT

Asthma and COPD are both airway diseases but by definition are distinguished by virtue of the fact that whilst asthma is reversible. Apart from some overlap asthma and COPD are two distinct diseases and must be labeled correctly when the patient is initially assessed. Studies from around the world show that asthmatics are often mislabeled as COPD and vice-versa. This can have a significant impact on the patient's future management and care. Too often asthma and COPD are managed as one and the same disease when in reality they are distinct. Too often patients with COPD are managed as patients with poorly responsive asthma when correct initial labeling and diagnosis might have improved the patient's course. The drug treatment of choice in asthma i.e. inhaled steroid has a marginal effect if any on patients with COPD. Pursuing such drugs in large doses in patients with COPD might expose these elderly patients to all the side effects and costs of inhaled steroids with none of the dramatic benefit this treatment has in asthma.

A good history can help distinguish these two conditions. More detailed tests of lung function can also be resorted to. Accurate labeling will ensure these two distinct groups of patients get appropriate and optimal treatment.

DIFFERENTIATING ASTHMA FROM COPD: HOW AND WHY?

Asthma and COPD are both airway diseases but by definition are distinguished by virtue of the fact that whilst asthma is reversible, COPD is not. Reversibility is defined as at least 15% improvement in FEV1 after administration of an inhaled beta agonist. Having made this important distinction it is important to realize that there are important exceptions to this rule. For example, a subset of patients with long-standing asthma will start off with reversible airways obstruction, but over the years their airways obstruction may become "fixed" so that these patients are now indistinguishable from COPD. Patients with years of poorly controlled asthma are more likely to develop fixed airways obstruction and airway re-modeling may be responsible. Asthmatics who smoke are also more likely to end up with irreversible airways obstruction. Overall, 1:10 asthmatics will develop fixed airways obstruction. If late-onset asthmatics are considered 1:4 will develop fixed airways obstruction. Epidemiological research has indicated that asthma, which historically had been thought of as an entirely reversible disorder, has a major irreversible and progressive component in a significant number of patients.[1,2] Thus there is considerable overlap between these groups of patients.

AIRWAY REMODELLING n ASTHMA

Despite apparently optimal therapy, some adults develop irreversible airflow obstruction solely from asthma, with an accelerated decline in FEV1, even when treated with systemic corticosteroid therapy. Reed surveyed a Mayo Clinic population over 65 years of age treated for asthma in 1993.[3] Many had severe, poorly reversible obstruction, but there was no correlation between FEV1 and duration of disease. Persistent allergen exposure, noncompliance, and steroid resistance may be factors in some cases of rapid decline. Thickening of the bronchiolar walls and remodeling of the airway were found at autopsy even more consistently than inflammatory infiltrates throughout the conducting airways, even in asthmatic subjects in whom death was not primarily attributable to asthma.[4] Asthmatic airway inflammation is believed to cause tissue injury and subsequent structural changes. These changes are referred to collectively as airway remodelling. Thus it is now clear that asthma, which historically had been thought of as an entirely reversible disorder, has a major irreversible and progressive component in a significant number of patients. The loss of lung function seen in most epidemiological studies of asthmatics followed up over years is now believed to be causally related to airway remodeling seen pathologically. Most studies have shown that most elements of the airway wall (smooth muscle, non-smooth muscle connective

tissue and mucous glands are increased, including submucosal and adventitial tissues. These changes are found in the airways of all sizes in fatal asthma, including bronchioles less than 2 mm in diameter. The changes are the same regardless of the underlying cause of the asthma; atopic, occupational or intrinsic. HRCT scanning has begun to be applied to the analysis of airway wall thickness to allow more widespread analysis of airway remodelling and serial interventional and observational studies.

Apart from some overlap asthma and COPD are two distinct diseases and must be labeled correctly when the patient is initially assessed. Studies from around the world show that asthmatics are often mislabeled as COPD and vice-versa. This can have a significant impact on the patient's future management and care. Too often asthma and COPD are managed as one and the same disease when in reality they are distinct. Too often patients with COPD are managed as patients with poorly responsive asthma when correct initial labeling and diagnosis might have improved the patient's course. The drug treatment of choice in asthma i.e. inhaled steroid has a marginal effect if any on patients with COPD. Pursuing such drugs in large doses in patients with COPD might expose these elderly patients to all the side effects and costs of inhaled steroids with none of the dramatic benefit this treatment has in asthma.

Some generalizations about the role of inhaled steroids in COPD can be made here. Generally speaking, patients with COPD should not receive inhaled steroids as COPD is a steroid-resistant disease. Unfortunately many patients with COPD continue to receive them in high doses and develop all the side effects with none of the benefit. The four reasons for not using inhaled steroids in COPD are;

1. No significant effect on inflammation.
2. No effect on progression of disease.
3. High risk of adverse systemic effects in these vulnerable elderly patients.
4. Expense.

The only justification for their use is that some studies (Euroscop study, Isolde study[5] and the Copenhagen study) show there may be a reduction in the frequency of exacerbations in COPD patients who receive inhaled steroids in COPD. Whether this reduction in exacerbations is clinically relevant or not is debatable and at present inhaled steroids cannot be routinely recommend in COPD. Similarly first-line treatments for COPD like tiotropium would have marginal benefit when administered to patients with asthma.

DIFFERENTIATING ASTHMA FROM COPD: IS USUALLY STRAIGHTFORWARD.

1. History: Most asthmatics are younger and give a history of atopy and often a family history of asthma or atopy. In contrast, patients with COPD are usually middle-aged to elderly and have a long standing history of smoking. The only type of COPD that runs in families is that associated with alpha-1-antitrypsin deficiency. Nocturnal worsening of asthma and the presence of clear triggering factors are also useful pointers in the history. Having said this, it must be stressed that at times, it is difficult to distinguish late-onset asthma from COPD especially in a smoker who may well have both diseases co-existing.
2. Spirometry: both diseases are characterized by airways obstruction. The main difference is an asthmatic, by definition will demonstrate 20% reversibility after inhaled salbutamol has been administered, whilst a patient with COPD will not. However, even this rule is not binding and some asthmatics may not demonstrate reversibility when it is first tested whilst some patients with COPD may have significant reversibility.
3. Diffusion capacity: Patients with emphysema have a decrease in their transfer factor whilst those with asthma have a normal or increased transfer factor.
4. Methacholine challenge: Bronchial hyper-reactivity can be demonstrated in patients with asthma with far lower concentrations (PC20) of histamine or methacholine than in patients with COPD.

Too often patients with asthma and COPD are mislabeled and end up being denied medicines which can impact on the quality of their lives. A thorough clinical and PFT assessment can give these patients the correct diagnostic label and allow their health care providers to give them the most appropriate therapy.

As this lecture will try and emphasise COPD and asthma are two distinct diseases with:

1. Different causes.
2. Different inflammatory cells.
3. Different mediators.
4. Different inflammatory consequences
5. Different degrees of airway hyper-responsiveness.
6. Different response to treatment.

Despite these obvious differences, according to Peter Barnes, most patients with COPD are still managed as though they have poorly controlled asthma. This lecture will try to emphasise the similarities and differences between these two common airway diseases.

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