Asthma and chronic obstructive pulmonary disease (COPD) are highly prevalent chronic airway diseases. Chronic bronchitis was defined in 1965 as productive cough for at least three months a year. (1) In the 70’s we were taught that asthma was a respiratory disease based on bronchospasm and we first heard about inflammation. The term chronic obstructive pulmonary disease (COPD) was later created to include chronic bronchitis and emphysema but more recently it was defined as the physiologic finding of non-reversible pulmonary function impairment. Asthma treatments progressed with the evolution of the concept of the disease. (2)

In the middle 20th century asthma was called a disease with paroxysmal dyspnoea and reversible bronchoconstriction. Spasm of bronchial smooth muscle was thought to be the major mechanism of airway narrowing. (3) In the 60’s asthma was defined as disease of airway hyper responsiveness. (4)

Around the 1980s, inflammation was found to be related with airway hyper responsiveness and asthma symptoms and was characterised by the presence of eosinophils and type 2 helper T lymphocytes who contributes to histological and functional airway’s changes including desquamation of bronchial epithelium and airway hyper responsiveness. (5)

COPD is an inflammatory disorder that typically develops in cigarette smokers in middle age. Indoor biomass smoke exposure can be another important cause of COPD, predominantly in low and middle income countries. Airways obstruction is the hallmark of COPD disorder, and the response to corticosteroids is modest or absent. The Key COPD inflammatory cells are predominantly neutrophils and involve CD8 lymphocytes.

The Global Initiative for Asthma (GINA) started in 1993 and the first report was published in 1995. GINA developed the concept of airway inflammation. (6) The National Asthma Education and Prevention Program Expert Panel Report states that asthma is "a complex disorder characterised by variable and recurring symptoms, airflow obstruction, bronchial hyper responsiveness, and underlying inflammation."

The executive summary or the Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease (GOLD) defines COPD as "a common preventable and treatable disease, characterised by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response". (7) Asthma and COPD represent a challenge to all physicians in their diagnosis and management. Both are characterised by heterogeneous chronic
airways inflammation and obstruction. In both conditions, chronic inflammation affects the whole respiratory tract, from central to peripheral airways, with different inflammatory cells recruited, different mediators produced, but different responses to treatment. Asthma and COPD can easily be misdiagnosed for one another and that is why testing is so important to determine the exact diagnosis. But there are clear differences between asthma and COPD.

Asthma is an allergic disease with recurrent episodes of symptoms such as wheezing, breathlessness, chest tightness and coughing, which commonly occur at night or in the early morning. These flare up periods are associated with triggers that cause obstruction of the airways, which is usually reversible.

COPD is a disease characterised by the progressive obstruction of the airways that leads to a reduction in airflow and respiratory function which is not completely reversible. Inhaled noxious particles or gases, such as cigarette smoke, are associated as risk factors and damage the lungs.

Diagnosis of COPD usually occurs in older adults with a history of exposure to inhaled irritants. Patients with asthma have typically intermittent and reversible airway obstruction on spirometry and variability on peak flow measurements, whereas in COPD obstruction is progressive and largely irreversible. Symptoms and spirometry values vary over time with in asthma, whereas in COPD they are associated with a slow, steady decline. Another difference is that in asthma airflow can be restored, but in COPD airflow is only temporarily restored or not restored at all.

In general, COPD treatment has a much more limited effect compared with that of asthma. Inhaled corticosteroids (ICSs) are the cornerstone of the pharmacological management of patients with persistent asthma, while inhaled bronchodilators (β2-agonists and anticholinergics) are the elective treatment for COPD patients. There are no disease-modifying medications currently available that can alter the progression of airflow obstruction in either asthma or COPD. Smoking cessation, however, is an essential component of the successful management of any obstructive airway disease.

Lung function declines over decades in both asthma and COPD. It is believed that this is largely owing to airway remodelling secondary to inflammation. Moderate to severe asthma that begins at an early age tends, in mid-life, to resemble COPD clinically, particularly if it has not been well controlled by ICS. The presence of eosinophils in sputum, blood, and airway histology has long been recognised as a feature of asthma, yet they are commonly found in patients with COPD, particularly during acute exacerbations. Similarly, exhaled nitric oxide (FENO), once described as a marker for asthma, is often absent in asthma and can be present in the exhaled breath of smokers with COPD.

The diagnosis of asthma COPD overlap (ACO) may be useful and suggests appropriate treatment. ACO is increasingly recognised as a distinct clinical entity. Some 10%-20% of asthma patients will have features of COPD, and 10%-20% of COPD patients will have features of asthma. However, adult-onset asthma is a well-recognised phenomenon. Patients with COPD also have variability in their symptoms and often experience repeated exacerbations.

In short, age of onset, disease course, and bronchodilator response (BDR) are helpful in distinguishing one disease from the other, but they are not definitive. Eventually there may by cellular, histologic, or genetic profiles that help with disease identification, but we are not there yet. Genetic studies have not identified profiles that reliably allow differentiation.

Although cellular, genetic, spirometry, and histologic testing cannot reliably identify a patient as having asthma, COPD, or ACO, clinical features are sufficient to alert the physician to the presence of overlap. ICSs are an important component of management, even if the primary diagnosis is COPD; and, for now, LABA monotherapy should probably be avoided.
Take Home Message

- **COPD**
  - Smoker or ex-smoker
  - Symptoms rare under age 35
  - Persistent and progressive breathlessness worse with exercise
  - Chronic productive cough with sputum production
  - Diagnosis - spirometry, exposure, symptoms
  - Baseline treatment - smoking cessation + inhaled bronchodilators (LAMA and/or LABA)

- **Asthma**
  - Symptoms are common under age 35
  - Breathlessness is variable
  - Night-time waking with breathlessness and/or wheeze is common
  - Diagnosis - history, symptoms, evidence or reversible airflow obstruction
  - Baseline treatment – inhaled ICS (+ SABA)

Original Abstract

http://www.woncaeurope.org/content/3754-can-general-practitioners-differentiate-between-asthma-and-copd-didasco-study

References

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