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Asthma and COPD: Similarities and Differences in the Pathophysiology, Diagnosis and Therapy

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Abstract

Asthma and chronic obstructive pulmonary disease (COPD) are two of the most common chronic lung diseases worldwide. Distinguishing between these different pulmonary diseases can be difficult in practice because of symptomatic similarities. A definitive diagnosis is essential for correct treatment. This review article presents the different symptoms of these two chronic inflammatory lung diseases following a selective search of the PubMed database for relevant literature published between 1996 and 2012. While cough occurs in both diseases, asthmatics often have a dry cough mainly at night, which is often associated with allergies. In contrast, COPD is usually caused by years of smoking. Paroxysmal dyspnea, which occurs in asthma, is characterized by shortness of breath, while in COPD it occurs during physical exertion in early stages and at rest in later stages of the disease. Asthma often begins in childhood or adolescence, whereas COPD occurs mainly in smokers in later life. It is possible to live with asthma into old age, whereas the life expectancy of patients with COPD is significantly limited. Currently, there is no general curative treatment for either disorder.

Keywords

Asthma • COPD • Detection • Symptoms • Treatment

1 Introduction

Bronchial asthma and chronic obstructive pulmonary disease (COPD) are among the most common chronic diseases of the lungs. They have similar symptoms that include shortness of breath, cough, and sputum, and these similarities can often cause difficulties in making the correct

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diagnosis for physicians in everyday practice (Carolan and Sutherland 2013). These lung diseases differ from each other mainly in terms of pathogenesis, disease progression, prognosis, and treatment options (Chang and Mosenifar 2007). It is important to distinguish between asthma and COPD for proper treatment, but early diagnosis and initiation of treatment remain a major challenge for even the experienced clinician and some pulmonologists (Rothe 2012). The correct guideline-based diagnosis and appropriate pharmacological treatment of these two common diseases is extremely important.

Against this background, the present study investigates the various clinical symptoms of asthma and COPD to improve their rapid early detection. A search of previous relevant publications from 1996 to 2012 in the PubMed database was conducted, and their results were analyzed. Using this information, improvements can be made to the guidelines for drug treatment options for these diseases. Another major objective of this study is to discover new distinctive features in clinical symptoms, diagnosis, and treatment of both asthma and COPD.

2 Allergic Asthma

Allergic asthma is characterized as a form of chronic inflammation of the lungs and in particular by immunoglobulin-E (IgE)-mediated hypersensitivity of bronchi to aero-allergens, with increased mucus secretion. While the airway inflammation induced by allergen exposure is almost always eosinophilic, most non-allergic asthma is also associated with eosinophils. The result of this progressive chronic bronchial inflammatory process is called allergic asthma (Postma and Kerstjens 1998). The airway is obstructed by increased mucus secretion, with irreversible structural remodeling of the airways (Aoshiba and Nagai 2004). In addition, the bronchial walls can thicken over the course of the disease due to an increase in mucous hypersecretion. The structural remodeling of bronchi is largely responsible for the long-term narrowing of airways and thus it underlies the major disease symptoms, such as shortness of breath, cough, and sputum in both severe asthma and also in COPD (Górska et al. 2009). Bronchi of the lower airways are particularly hypersensitive to cold air, perfumes, cigarette smoke, and other non-specific stimuli in the air, which is referred to as airway hyperresponsiveness (Miglino et al. 2012; Bleecker 2004). Asthma can often be treated by individually tailored drug therapy to manage symptoms, but it currently is still an incurable, chronic disease (Cukic et al. 2012).

Often, when given the diagnosis of asthma, patients ask ‘Why me?’ The fact is that according to the World Health Organization more and more people suffer from asthma, with over 300 million people with asthma in the world and approximately 250,000 associated deaths (Cukic et al. 2012). There are certain risk factors that contribute to the severity of asthma that are considered the casual factors for the dramatic increase in the incidence of this disease. In addition, genetic predisposition and certain environmental factors probably are at play in the appearance of asthma (Kaneko et al. 2013; Bleecker 2004).

3 Chronic Obstructive Pulmonary Disease (COPD)

According to the WHO, COPD is among the most common causes of death worldwide, with approximately six million people affected in Germany and 16 million affected in the United States. These figures may represent low estimates, because there is evidence of missing or delayed diagnoses of COPD (Bleecker 2004). The main risk factor for COPD is tobacco smoke (Sutherland and Martin 2003). Most patients suffering from COPD are either active smokers, passive smokers, or former smokers (Kardos et al. 2006). Accordingly, COPD is also referred to, in common parlance, as smoker’s cough. In some cases, people have a hereditary genetic predisposition to develop COPD, which is related to alpha-1-protease inhibitor deficiency (Kaneko et al. 2013). The lack of the enzyme results in unopposed proteolytic damage to the alveolar capillary membrane and does not rely on

amplified inflammation of the lung caused by a mutation in the protein of alpha-1-protease inhibitor (Kardos et al. 2006; Sutherland and Martin 2003). COPD leads to recurrent infections of the lungs, which accelerates the destruction of lung tissue. In later stages of the disease, comorbidities such as hypertension, coronary artery disease, diabetes, lung cancer, or depression worsen symptoms.

4 Similarities and Differences Between Asthma and COPD

The classical symptom of COPD is exertional dyspnea. Cough and sputum are inconsistent symptoms of COPD especially in ex-smokers (Bleecker 2004). Chronic cough occurs almost universally in smokers without airflow obstruction, which underlines the definition of chronic bronchitis. It usually goes away in COPD patients who stop smoking, which constitutes a majority of patients. Bronchial asthma causes similar symptoms. In both diseases, there is chronic inflammation with cellular and structural changes, known as airway remodeling with reconstruction, and these structural changes lead to the thickening of airway walls, which facilitates airway constriction and, consequently, airflow restriction (Nakawah et al. 2013; Miglino et al. 2012; Górska et al. 2009). Nonetheless, there are key differentiators between the two diseases. The pattern of infiltrated cells and structural changes are different. In asthma, CD4, T-lymphocytes, eosinophils, and mast cells are the predominant cells involved, while in COPD, CD8, T-lymphocytes, and macrophages are predominantly involved. In severe cases of both asthma and COPD, there also can be infiltration of neutrophils into the airway walls. The thickening of smooth muscles dominates in large airways in severe asthma and in small airways in COPD (Sköld 2010; Welte and Groneberg 2006; Aoshiba and Nagai 2004). Structural similarities of the reconstruction process in asthma and COPD consist of the thickening of bronchial wall and airway mucosa, the proliferation of mucus-producing cells, and of the luminal narrowing of airways by increased secretion of inflammatory cells and mucus (Górska et al. 2009). Typical structural alterations in COPD include epithelial changes due to cell metaplasia, mucous membrane changes, and fibrosis of airway walls. The destruction and fibrosis of the alveolar wall is indicative of COPD, but not of asthma (Aoshiba and Nagai 2004). Another difference is the patient age at onset, which is often in childhood and usually before 40 years of age in asthmatics, while COPD usually occurs after 50 years of age. There is often a causal relationship to allergic diseases among asthmatics (Yawn 2009). Occasionally, no allergens are identified in patients with asthma, a situation which constitutes a non-allergic or intrinsic asthma. That could also mean that allergens remain unrecognized, rather than absent, in such patients. It is more difficult to distinguish between asthma and COPD when the patient has a long history of smoking, as the patient may suffer from both diseases (Kardos et al. 2006). Previously, post beta-agonist reversibility of bronchial obstruction was considered a distinguishing feature of asthma from COPD, but this is no longer valid based on recent clinical experience (Nakawah et al. 2013; Bleecker 2004). COPD patients can show a degree of bronchial reversibility, and conversely asthma patients can show poor post beta-agonist reversibility, having a fixed obstruction similar to that appearing in COPD. Thus, in differentiating the two diseases, the patient history and clinical evaluation ought to be taken into account (Kardos et al. 2006; Welte and Groneberg 2006; Bleecker 2004). The most common reason for the lack of bronchial reversibility in asthma is that baseline spirometry is normal. To fully examine the test post beta-agonist reversibility, a trial of corticosteroid is needed. A history of tobacco smoking exacerbates the symptoms not only in asthma and COPD, but also in a range of other lung ailments. Tobacco smoking causes atherosclerosis, resulting in afflictions such as myocardial infarction, depression, osteoporosis, and diabetes (Bleecker 2004).

5 Differences in Diagnosis Between COPD and Asthma

The task of the lung is to exchange gas. Oxygen is breathed from the ambient air *via* airways into the air sacs where it is exchanged with carbon dioxide coming from cells, which is then exhaled from the body. There are various investigative methods to measure lung volume, such as spirometry and body plethysmography (Abramson et al. 2012; Mosenifar 2009). Table 1 shows the most important differentiating factors in the major characteristics of asthma and COPD.

In both diseases, respiratory lung function is reduced. In COPD, airway resistance persistently increases, and while this can also be the case in asthma, airway resistance typically increases during an acute exacerbation of asthma. Inhaling beta2-sympathomimetics may reduce airway resistance and improve lung function in asthmatics. Such a drug effect occurs only to a small extent in patients with COPD. While spirometry gives often normal readings in an attack-free period in asthmatics, such findings are seldom in COPD patients, with a forced expiratory volume in one second (FEV1) less than 70 % of forced vital capacity (FVC) (Tandon et al. 2013; Miravitlles et al. 2012; Oga et al. 2010; Mosenifar 2009; Silvestri et al. 2008; Guerra 2005). The flow rate of exhaled air during the forced exhalation depends on airway resistance and is referred to as peak flow. It can be measured by spirometry or with a peak flow meter. The measurement is useful for asthmatics, who can be administered drugs during large peak flow fluctuations, which do not frequently occur in COPD (Mishima 2009; Pauwels 2004).

Radiographic examination shows no typical signs for asthma. In COPD, on the other side, signs of lung emphysema are present. Emphysema is inferred from vascular paucity or bullae as well as from increased radiolucency of lungs, flattening of the diaphragm, extended intercostal spaces, and rarefaction of blood vessels in the lung periphery seen in radiological examination (Chang and Mosenifar 2007; Martinez et al. 2005); the signs signifying the presence of lung hyperinflation, although hyperinflation can occur without emphysema in pulmonary diseases like asthma. Table 2 lists basic differences in the diagnosis and treatment of asthma and COPD.

In asthma patients, eosinophils are predominantly detected in sputum, whereas in COPD patients neutrophils are found (Pauwels 2004; Sutherland and Martin 2003). A noteworthy difference in drug therapy is that glucocorticoids are highly effective in asthmatics, but are of a meager effect in COPD (Bumbacea and Bogdan 2011; Morice et al. 2008; Kardos et al. 2006; Decramer and Selroos 2005; Löfdahl et al. 2005).

Table 1 Clinical differences between bronchial asthma and COPD

	Asthma	COPD
Symptoms		
Shortness of breath	During acute exacerbation	First during exercise; later at rest
Cough	Dry cough, often at night	Smoker's cough; especially in the morning with mucus
Expectoration	Crystal clear, very tenacious	Clear, yellowish
Age of onset	Any age, more common in children and adolescents, occasionally at age 70 and over	Over 40 years
Disease onset	Often suddenly	slowly, then chronic progressing bronchitis
Trigger	Infection, cold, stress, irritants, exercise, neurodermitis	Tobacco smoke
Cause	Pollen, house dust mites, animal hair, inheritance	Smoking, air pollution
Life expectation	Life possible into old age	Significantly shortened, typically very slow decline

COPD chronic obstructive pulmonary disease

Table 2 Diagnostic and treatment differences between asthma and COPD

	Asthma	COPD
Spirometry		
Respiratory function	Reduced	Reduced
Breathing resistance	Increased during acute exacerbation; reduced by beta-2 sympathomimetics	Increased; hardly reduced by beta-2 sympathomimetics
Spirometry findings	Often normal in the absence of acute exacerbation	FEV1/FVC ratio less than the lower limit of normal
Peak flow	Large fluctuations; reduced in the morning	Very small fluctuations
Radiographic examination	No typical change	Signs of pulmonary emphysema: increased lung radiolucency, diaphragm flattening, extended intercostal spaces, rarefaction of blood vessels in lung periphery
Laboratory	Eosinophils, immunoglobulin-E	Neutrophils
Sputum	Eosinophils	Neutrophils
Glucocorticoids	Great effect	Little effect

COPD chronic obstructive pulmonary disease, *FEV1* forced expiratory volume in one second, *FVC* forced vital capacity, *VC* vital capacity

6 Discussion

The chronic inflammatory respiratory diseases asthma and COPD are found worldwide, but their prevalence is underestimated (Athanazio 2012). The COPD-related mortality is on the rise. It is the fourth leading cause of death around the world (Cukic et al. 2012). Accordingly, COPD and asthma are major causes of chronic morbidity and constitute a global health challenge. It is expected that the prevalence of COPD will continue to grow in the years to come as the life expectancy globally increases; the diseases appears along the aging process (Bleecker 2004). Additionally, there is a long time lag between the initiation of smoking in early life and the late onset of disease. A misdiagnosis of COPD or asthma leads to inadequate treatment and raises health care costs (Spencer and Krieger 2013; Kuebler et al. 2008).

Differentiation between asthma and COPD in their early phases is essential for the introduction of therapeutic measures. Apart from a higher economic burden, COPD has a less favorable prognosis than asthma and is associated with greater morbidity and mortality (Decramer and Selroos 2005). However, due to a high prevalence of both diseases and their common pathophysiological processes, some patients may manifest similar symptoms making the diagnosis ambiguous or difficult (Athanazio 2012). While the most common symptoms, such as shortness of breath, cough, and sputum can singly or simultaneously occur in asthma or COPD, they differ in the way they appear. Other disorders may also be behind these cardinal symptoms, such as cardiovascular or ear, nose, and throat diseases. A simultaneous presence of infectious comorbidities, manifesting a similar clinical picture, can further obscure the diagnosis of asthma or COPD. A cardinal symptom of COPD, which may be accompanied by cough, phlegm, and wheezing, is shortness of breath during exertion. Older people often experience such shortness of breath due to deconditioning or obesity. Cough and sputum is so widespread in smokers that it is often viewed inattentively. Wheezing occurs frequently in asthmatics, but the casual factor could also be viral infections of airways (Martinez et al. 2005; Bleecker 2004). Further, asthma and COPD may coexist. While asthma alone typically occurs in younger patients, the onset of both overlapping diseases may appear at a similar later age. The hospitalization rate in patients with the overlapping diseases is almost twice as high as that for the isolated occurrence of asthma and COPD (Andersén et al. 2013).

Studies suggest that asthma and COPD are pathophysiologically and clinically different. The extent to which allergies and heredity can contribute to the development of asthma is not fully understood. COPD, however, is mainly caused by tobacco smoking, although a certain predisposition may also play a causative role (Bleecker 2004). According to some hypotheses, asthma and COPD either have a common origin with different phenotypic presentations or are caused by the interaction between endogenous and exogenous factors. There are apparent differences in the two diseases concerning the majority of inflammatory cells and mediators, albeit a number of commonalities in the inflammation process have been found. Generally speaking, COPD is diagnosed at a rather advanced age. A diagnosis of asthma from pulmonary function tests implies a completely reversible post beta-agonist narrowing of airways. Hyperinflation at rest makes a diagnosis of COPD likely. COPD should also be considered in cases of limited diffusion capacity, since these measurements are usually normal or even elevated in asthmatics. A reduced elasticity of lungs is a hallmark of COPD, and a pathophysiological enlargement of airspaces with destruction of the airway wall is especially notable with emphysema. In younger patients, atopy is indicative of asthma diagnosis (Chang and Mosenifar 2007; Martinez et al. 2005; Sciurba 2004). Other triggers, such as dust, influenza infection, climbing stairs, smoking, chemicals, or pollen, may contribute to the development of asthma and COPD, with similar triggering frequency in both diseases (Aydin et al. 2013). Preventive actions against these triggers are undertaken more frequently in patients with asthma. In contrast, vaccination rates for influenza and pneumococcal pneumonia are significantly higher in patients with COPD. It seems that the education of these two patient groups should focus on triggers and strategies on how to manage the disease. Both diseases have common features, such as obstruction, inflammation, and hypersensitivity of airways. Nonetheless, since the inherent underlying mechanisms are different, they are considered two different pathologies, which require different assessment, monitoring, and treatment (Hoshino et al. 2009).

Recently, indications have emerged that viral respiratory infections are a major cause of exacerbations of asthma and COPD. Much of the increase in morbidity, mortality, and health care costs are caused by acute exacerbations. There is no effective drug therapy for the prevention or treatment of virus-induced exacerbations. Developing new therapies requires a better understanding of how the molecular and cellular mechanisms shape the role of viral infections in exacerbations of asthma and COPD (Mallia et al. 2007). The frequency of exacerbations is similar in asthma and COPD and it is related to the severity of disease. Common causes are viral or bacterial infections, pollution, and increased allergen exposure. Eosinophilia and neutrophilia have also been associated with exacerbations and avoidance of causative factors reduces the exacerbation rate in both diseases. Pharmacological prevention of exacerbations has been demonstrated in asthma using monoclonal anti-IgE with long-acting inhaled beta2 agonists in addition to inhaled corticosteroid therapy. Exacerbations in COPD have been reduced with inhaled corticosteroids, long-acting inhaled beta2 agonists, or a combination of both with the possible addition of long-acting inhaled anticholinergics (Pauwels 2004).

The gold standard for the diagnosis of asthma and COPD is spirometry, but this is seldom used in practice. For this reason, COPD is often underdiagnosed. While the spirometry findings for differentiating between asthma and COPD are ignored in everyday practice, they are helpful in difficult or questionable cases (Abramson et al. 2012; Miravitlles et al. 2012). Although diagnostic guidelines exist for both diseases, differentiation between asthma and COPD is not full clear. Therefore, a better awareness of differences in spirometry between the two diseases would help promote the optimal treatment (Tinkelman et al. 2006). One study has attempted to develop a questionnaire for the distinction between asthma and COPD (Beeh et al. 2004). By collecting data of adult patients with a diagnosis of asthma or COPD from a

pulmonary specialist practice, a simple quantitative questionnaire has been created to support a diagnosis of COPD with a high score and asthma with a low score. The questionnaire results were compared against the diagnoses made at a doctor's office according to the guidelines of the Global Initiative for Asthma (GINA) and the Global Initiative for Chronic Obstructive Lung Diseases (GOLD), including skin tests, spirometry, and reversibility. The questionnaire appears to facilitate the differentiation between COPD and asthma in daily clinical practice when applied to the former patients (Miravitlles et al. 2012; Beeh et al. 2004). Further studies are needed to confirm these initial observations.

Recent studies have unraveled additional facts for a close epidemiological and clinical relationship between asthma and COPD. Adult patients with asthma are 12 times more likely to develop COPD over the course of their lives than subjects without asthma. Consequently, early identification of patients with signs of asthma could be considered as an indication of a predisposition for the later development of COPD. These findings may have an effect on the prevention of COPD (Guerra 2005). In any case, the exact relationship between the chronic lung diseases asthma and COPD remains to be explored (Jenkins et al. 2005). An enhanced understanding of the pathophysiology of multiple obstructive pulmonary diseases is required to promote efficient, early, and optimal treatment of asthma and to prevent its progression to COPD.

7 Conclusions

Although asthma and COPD have many symptomatic similarities, the causes and pathophysiology of the diseases remain unclear. While COPD is associated with an abnormal radiographic examination, asthma has normal airway resistance between exacerbations. COPD is commonly known as smoker's cough. In asthma, eosinophils are predominantly detected in sputum. Asthma is often caused by animal hair and triggered by neurodermatitis. Differences in the inflammation process must be clarified in both diseases with more extensive research. A disturbed gas exchange that occurs in COPD, but not in asthma, also needs to be investigated, alongside a rather common occurrence of other systemic comorbidities being significantly increased in COPD patients. New diagnostic tools and blood markers help identify and differentiate the two lung diseases, which ensures early treatment. The development of new strategies for better patient-oriented treatment of respiratory diseases is sought.

Competing Interests The authors report no conflicts of interest in relation to this work.

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