Sample background information on COPD treatment and management

For use with medical press

Definition

COPD is a multi-component disease involving airway obstruction, airway inflammation, airway structural changes, muco-ciliary dysfunction and a systemic component.[1,2] These components contribute to a complex of changes in lung function, symptoms and exacerbations, which affect health status and ultimately survival.

Guidelines set by the Global Initiative for Chronic Obstructive Lung Disease (GOLD), a collaboration between the US National Heart, Lung and Blood Institute (NHLBI) and the World Health Organisation (WHO), have defined COPD as 'a disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with abnormal inflammatory response of the lung tissue to damaging particles such as cigarette smoke'.[1]

Risk factors

Risk factors for COPD include both host susceptibility and environmental exposures, and the disease usually arises from an interaction between the two types of factors[1]. There is no doubt that the biggest single risk factor for COPD is smoking[3]. Other major environmental factors are heavy exposure to occupational dusts and chemicals (vapours, irritants, fumes) and indoor/ outdoor pollution[1]. The host risk factors include bronchial hyper-responsiveness and a relatively rare hereditary disorder involving a deficiency in the enzyme alpha-1 antitrypsin[1].

Diagnosis

The diagnosis of COPD is usually based on a history of exposure to risk factors and the presence of airflow limitation that is not fully reversible. Patients who have a chronic cough and sputum production with a history of exposure to risk factors should be tested for airflow limitation even if they do not have dyspnoea. Spirometry is the most reproducible, standardised and objective means of measuring airflow limitation. A ratio of $FEV_1/FVC < 70\%$ and a post-bronchodilator $FEV_1 < 80\%$ predicted confirms the presence of airflow limitation that is not fully reversible. The diagnosis of COPD often does not occur until the disease has progressed significantly. This is usually due to a lack of serious symptoms in the early phase of the disease and because spirometry is not routinely performed by primary care physicians.

Exacerbations

An exacerbation occurs when a COPD patient's symptoms suddenly worsen. There is no clear agreement on the definition of an exacerbation. It may be defined as a worsening of a specified group of COPD symptoms, or if a patient experiences an acute change in clinical state that requires prescription of antibiotics and/or oral corticosteroids, or that requires hospitalisation.

People with moderate and severe COPD may have prolonged symptomatic effects following an exacerbation[4]. Exacerbations are a common cause of hospital admission and for patients with persistent symptoms hospitalised after their first exacerbation, are a major cause of frequent re-hospitalisation [4].

Treatment

Stopping smoking is the single most effective intervention to reduce the risk of developing COPD and stop its progression[1]. Therapeutic interventions include the following:

a) Bronchodilators

Inhaled bronchodilators are used to treat airways obstruction, working to reduce airways resistance and pulmonary hyperinflation by reducing bronchomotor tone. Bronchodilators include inhaled short and long-

acting $beta_2$-agonists, which cause bronchodilation by stimulating the $beta_2$-receptors, and anticholinergics (e.g. ipratropium) that block the action of acetylcholine at cholinergic receptors on smooth muscle cells. Short-acting $beta_2$-agonists (e.g. salbutamol) relieve acute symptoms, but have little or no effect on exacerbations or health status. In contrast, long-acting $beta_2$-agonists (e.g. salmeterol) have shown clear benefits in reducing exacerbations and improving health status[5].

b) Corticosteroids
Inhaled corticosteroids (ICS) are well established as a prophylactic therapy in asthma, acting on the underlying inflammation and bronchial hyperactivity. The mode of action of ICS in COPD is still not fully understood, but it is thought they act to reduce the presence of a number of inflammatory cells (e.g. neutrophils, CD8 cytotoxic lymphocytes) and inflammatory mediators (e.g. IL-8, TNFα, LTB_4). The use of ICS has been shown to reduce the rate of exacerbations and slow decline in health status[6]. Patients with acute exacerbations are usually treated with oral corticosteroids and/or antibiotics.

c) Combination therapies
Combining an inhaled corticosteroid and a long acting $beta_2$-agonist offers superior benefits than the individual components administered alone[7, 8]. Combination therapies target many of the pathophysiological components[9] and have been shown to reduce exacerbations[8], achieve symptomatic control[7,10,11] and improve lung function[7,11].

Clinical Management of COPD
The WHO and the US National Institutes of Health have produced the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines[1], offering a strategic plan to manage COPD. Goals are to be achieved via a four-component programme: assessment and monitoring of disease, reducing risk factors, managing stable COPD and managing exacerbations. Inhaled bronchodilators are central to long term COPD management, with inhaled corticosteroids added when indicated. Influenza vaccination is recommended, as is a rehabilitation programme for all patients, and oxygen therapy in patients with severe disease.

Ultimately, increasing awareness of the burden of COPD needs to be addressed to ensure optimal disease management. Goals of successful COPD management are prevention, early diagnosis, symptom control, prevention of deterioration of lung function, prevention of developing complications and improvement of health status for patients. Patients are also key to the future management of their disease as they may underestimate the severity of their symptoms and therefore, unknowingly, do not seek appropriate help.

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Enquiries:
Media enquiries: *[Insert your own contact details here]*

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