Introduction

Chronic obstructive pulmonary disease (COPD) poses a challenge to current and future health care systems. As a result of increased tobacco consumption and demographic development, COPD is expected to become the third leading cause of death worldwide by the year 2020 ***(Murray & Lopez, 1997).***

Air trapping is a critical clinical feature of COPD. Early diagnosis and intervention is necessary to prevent a further decline of lung function in these patients. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommends spirometery as the gold standard for the diagnosis of COPD, since it is the most reproducible, standardized and objective way of measuring airflow. However, to perform spirometery, experienced and regularly trained medical assistants are needed as well as physicians for interpreting the results ***(Hill et al., 2010).***

A novel approach to non-invasive monitoring of the lungs is low frequency chest ultrasonography ***(Dos Santos et al., 2008).***

Ultrasound has received increasing interest from chest physicians in recent years. Modern ultrasound devices are used easily, inexpensive, lightweight and portable, which

makes them suitable for outpatient settings as well as bedside investigation of the severely ill patient ***(Middleton et al., 2004).***

Conventional ultrasound with frequencies ranging from 2 to 10 MHz is increasingly used for the diagnosis of pulmonary diseases including pneumothorax, pleural effusion, alveolar-interstitial syndrome and lung consolidation ***(Colmenero et al., 2010).***

Aim of the Work

The aim of this study was to evaluate the role of chest ultrasonography in detection of air trapping in COPD patients.

Chronic Obstructive Pulmonary Disease (COPD)

Chronic Obstructive Pulmonary Disease (COPD), a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients. The chronic airflow limitation characteristic of COPD is caused by a mixture of small airways disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes and narrowing of the small airways. Destruction of the lung parenchyma, also by inflammatory processes, leads to the loss of alveolar attachments to the small airways and decreases lung elastic recoil; in turn, these changes diminish the ability of the airways to remain open during expiration ***(Lopez et al., 2006).***

COPD is one of the most important causes of death in most countries. The Global Burden of Disease Study projected that COPD, which ranked sixth as a cause of

death in 1990, will become the third leading cause of death worldwide by 2020; a newer projection estimated COPD will be the fourth leading cause of death in 2030. This increased mortality is mainly driven by the expanding epidemic of smoking, reduced mortality from other common causes of death (e.g. ischemic heart disease, infectious diseases), and aging of the world population ***(Mathers & Loncar, 2006).***

COPD is associated with significant economic burden. In the European Union, the total direct costs of respiratory disease are estimated to be about 6% of the total health care budget, with COPD accounting for 56% (38.6 billion Euros) of this cost of respiratory disease ***(Halbert et al., 2006).***

Pathophysiology

Airflow Limitation and Air Trapping. The extent of inflammation, fibrosis, and luminal exudates in small airways is correlated with the reduction in FEV1 and FEV1/FVC ratio, and probably with the accelerated decline in FEV1 characteristic of COPD. This peripheral airway obstruction progressively traps air during expiration, resulting in hyperinflation ***(Hogg et al., 2004)***.

Gas Exchange Abnormalities. Gas exchange abnormalities result in hypoxemia and hypercapnia, and have several mechanisms in COPD. In general, gas transfer for oxygen and carbon dioxide worsens as the disease progresses. Reduced ventilation may also be due to reduced ventilatory drive. This may lead to carbon dioxide retention when it is combined with reduced ventilation due to a high work of breathing because of severe obstruction and hyperinflation coupled with ventilatory muscle impairment. The abnormalities in alveolar ventilation and a reduced pulmonary vascular bed further worsen the V/Q abnormalities ***(Rodriguez et al., 2009).***

Mucus hypersecretion, resulting in a chronic productive cough, is a feature of chronic bronchitis and is not necessarily associated with airflow limitation. Conversely, not all patients with COPD have symptomatic mucus hypersecretion. When present, it is due to an increased number of goblet cells and enlarged submucosal glands in response to chronic airway irritation by cigarette smoke and other noxious agents. Several mediators and proteases stimulate mucus hypersecretion and many of them exert their effects through the activation of epidermal growth factor receptor (EGFR) ***(Burgel & Nadel, 2004).***

Pulmonary Hypertension. Pulmonary hypertension may develop late in the course of COPD and is due mainly to hypoxic vasoconstriction of small pulmonary arteries, eventually resulting in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/ hyperplasia ***(Peinado et al., 2008).***

Exacerbations

Exacerbations of respiratory symptoms often occur in patients with COPD, triggered by infection with bacteria or viruses (which may coexist), environmental pollutants, or unknown factors. Patients with bacterial and viral episodes have a characteristic response with increased inflammation. During respiratory exacerbations there is increased hyperinflation and gas trapping, with reduced expiratory flow, thus accounting for the increased dyspnea. There is also worsening of V/Q abnormalities, which can result in hypoxemia. Other conditions (pneumonia, thromboembolism, and acute cardiac failure) may mimic or aggravate an exacerbation of COPD ***(Parker et al., 2005).***

Systemic Features. It is increasingly recognized that many patients with COPD have comorbidities that have a major impact on quality of life and survival. Airflow limitation and particularly hyperinflation affect cardiac

function and gas exchange. Inflammatory mediators in the circulation may contribute to skeletal muscle wasting and cachexia, and may initiate or worsen comorbidities such as ischemic heart disease, heart failure, osteoporosis, normocytic anemia, diabetes, metabolic syndrome, and depression ***(Barnes & Celli, 2009).***

Diagnosis

A clinical diagnosis of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and a history of exposure to risk factors for the disease. Spirometry is required to make the diagnosis .the presence of a post bronchodilator FEV1/FVC < 0.70 confirms the presence of persistent airflow limitation and thus of COPD ***(Zwar et al., 2011).***

Symptoms

The characteristic symptoms of COPD are chronic and progressive dyspnea, cough, and sputum production that can be variable from day-to-day. Chronic cough and sputum production may precede the development of airflow limitation by many years ***(Kessler et al., 2011).***

Dyspnea

A cardinal symptom of COPD, is a major cause of disability and anxiety associated with the disease. Typical COPD patients describe their dyspnea as a sense of increased effort to breathe, heaviness, air hunger, or gasping ***(Simon et al., 1990).***

However, the terms used to describe dyspnea vary both by individual and by culture ***(Elliott et al., 1991).***

Cough

Chronic cough, often the first symptom of COPD to develop, is frequently discounted by the patient as an expected consequence of smoking and/or environmental exposures. Initially, the cough may be intermittent, but later is present every day, often throughout the day. The chronic cough in COPD may be unproductive. In some cases, significant airflow limitation may develop without the presence of a cough ***(Georgopoulas & Anthonisen, 1991).***

Sputum production. COPD patients commonly raise small quantities of tenacious sputum after coughing bouts. Regular production of sputum for 3 or more months in 2 consecutive years (in the absence of any other conditions that may explain it) is the epidemiological definition of chronic bronchitis, but this is a somewhat arbitrary

definition that does not reflect the range of sputum production in COPD patients ***(Stockley et al., 2000).***

Wheezing and Chest Tightness. Wheezing and chest tightness are nonspecific symptoms that may vary between days, and over the course of a single day. Additional Features in Severe Disease. Fatigue, weight loss and anorexia are common problems in patients with severe and very severe COPD ***(Schols et al., 1993).***

Medical History

Exposure to risk factors, past medical history. Family history of COPD or other chronic respiratory disease. Pattern of symptom development. History of exacerbations or previous hospitalizations for respiratory disorder Presence of comorbidities ***(Holguin et al., 2005).***

Physical Examination

Physical signs of airflow limitation are usually not present until significant impairment of lung function has occurred, and their detection has a relatively low sensitivity and specificity. A number of physical signs may be present in COPD, but their absence does not exclude the diagnosis ***(Kesten & Chapman, 1993).***

Spirometry

The presence of a post bronchodilator FEV1/FVC < 0.70 confirms the presence of airflow limitation. Classification of Severity of Airflow Limitation in COPD (Based on Post-Bronchodilator FEV1). In patients with FEV1/FVC < 0.70:

- Mild FEV1 ≥ 80% predicted
- Moderate 50% ≤ FEV1 < 80% predicted
- Severe 30% ≤ FEV1 < 50% predicted
- Very Severe FEV1 <30% predicted

(Jackson & Hubbard, 2003)

Lung Volumes and Diffusing Capacity:

COPD patients exhibit gas trapping (a rise in residual volume) from early in the disease, and as airflow limitation worsens static hyperinflation (an increase in total lung `capacity) occurs. These changes can be documented by body plethysmography. These measurements help characterize the severity of COPD but are not essential to patient management. Measurement of diffusing capacity (DLCO) provide information on the functional impact of emphysema in COPD and is often helpful in patients with breathlessness that may seem out of proportion with the degree of airflow limitation ***(Pellegrino et al., 2005).***

Imaging

A chest X-ray is not useful to establish a diagnosis in COPD, but it is valuable in excluding alternative diagnoses and establishing the presence of significant comorbidities such as concomitant respiratory (pulmonary fibrosis, bronchiectasis, pleural diseases), skeletal (e.g., kyphoscoliosis), and cardiac diseases (e.g., cardiomegaly). Radiological changes associated with COPD include signs of lung hyperinflation (flattened diaphragm on the lateral chest film, and an increase in the volume of the retrosternal air space), hyperlucency of the lungs, and rapid tapering of the vascular markings. Computed tomography (CT) of the chest is not routinely recommended ***(Fishman et al., 2003).***

Oximetry and Arterial Blood Gas Measurement

Pulse oximetry can be used to evaluate a patient's oxygen saturation and need for supplemental oxygen therapy. Pulse oximetry should be used to assess all stable patients with FEV1 < 35% predicted or with clinical signs suggestive of respiratory failure or right heart failure. If peripheral saturation is < 92% arterial blood gases should be assessed ***(Kelly et al., 2001).***

Treatment

Smoking Cessation.

Medications

Bronchodilators:

Medications that increase the FEV1 or change other spirometric variables, usually by altering airway smooth muscle tone. Such medications improve empting of the lungs, tend to reduce dynamic hyperinflation at rest and during exercise. Bronchodilator medications are given on either an as needed basis or regular basis to prevent or reduce symptoms ***(O'Donnell et al., 2006).***

Beta2-agonists:

The principal action of beta 2 - agonists is to relax airway smooth muscle by stimulating beta2- adrenergic receptors, the bronchodilator effect of short acting B2 agonist usually wear off with in 4 to 6 hours .Long acting B2 agonists show duration of action of 12 or more hours ***(Calverley et al., 2007).***

Anticholinergics:

The most important effect in COPD patients of anticholinergic medications, such as ipratropium, oxitropium and tiotropium bromide, appears to be blockage of acetylcholine's effect on muscarinic receptors. The bronchodilator effect of short acting anticholinergics last longer than of short acting B2agonist ***(Vincken et al., 2002).***

Methylxanthines:

They may act as nonselective phosphodiesterase inhibitors, but have also been reported to have a range of non-bronchodilator actions, the significance of which is disputed ***(Singh et al., 2011).***

Combination Bronchodilator Therapy:

Combining bronchodilators with different mechanisms and durations of action may increase the degree of bronchodilation for equivalent or lesser side effects. For example, a combination of a short-acting beta2-agonist and an anticholinergic produces greater and more sustained improvements in FEV1 ***(Vogelmeier et al., 2008).***

Corticosteroids:

Inhaled Corticosteroids

The dose-response relationships and long-term safety of inhaled corticosteroids in COPD are not known. Only moderate to high doses have been used in long-term clinical trials. The efficacy and side effects of inhaled corticosteroids in asthma are dependent on the dose and type of corticosteroid, but whether this is also the case in COPD is unclear. The effects of corticosteroids on pulmonary and systemic inflammation in patients with COPD are controversial, and their role in the management

of stable COPD is limited to specific indications. Regular treatment with inhaled corticosteroids improves symptoms, lung function, and quality of life, and reduces the frequency of exacerbations in COPD patients ***(Spencer et al., 2004).***

Combination Inhaled Corticosteroid/Bronchodilator therapy. An inhaled corticosteroid combined with a longacting beta2-agonist is more effective than the individual components in improving lung function and health status and reducing exacerbations in patients with moderate to very severe COPD ***(Boyd et al., 1997).***

Oral Corticosteroids

Oral corticosteroids have numerous side effects. An important side effect of long-term treatment of COPD with systemic corticosteroids is steroid myopathy, which contributes to muscle weakness, decreased functionality, and respiratory failure in subjects with very severe COPD ***(Decramer et al., 1996).***

Phosphodiesterase-4 Inhibitors:

The principal action of phosphodiesterase-4 inhibitors is to reduce inflammation by inhibiting of the breakdown of intracellular cyclic AMP, It is a once daily oral medication with no direct bronchodilator activity, although it has been shown to improve FEV1 in patients treated with salmeterol or tiotropium ***(Fabbri et al., 2009).***