ORIGINAL ARTICLE

Profile of a Brazilian population with severe chronic obstructive pulmonary disease*

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Chronic obstructive pulmonary disease (COPD) is a public health problem. Tobacco smoking is the major cause, but not the only one. Air pollution, exposure to chemicals, environmental smoke exposure, and passive smoking are among other contributing causes; being viral and bacterial infections also risk factors. Gender and weight are associated to the severity of the disease. Co-morbidity is frequent. **Objective:** To characterize a population of COPD outpatients followed at an outsourced medical service. **Methods:** Questionnaires were applied to patients with COPD. The data included gender, age, weight, body mass index (BMI), oxygen delivery users, and FEV_1, exposure to tobacco smoke, exposure to wood smoke, history of tuberculosis and co-morbid diseases. **Results:** Of the 70 patients enrolled in the study, 70% (49) were men with an average age of 64 ± 10 years, average weight of 63 ± 16 kg and average BMI of 22 ± 5 kg/m^2. Mean FEV_1 was 35 ± 14% and 45.7% were oxygen dependent. Nine (12.8%) patients never smoked, while 78.8% had quit tobacco smoking, (38 ± 11 pack/years was the average). Nine (12.8%) smoked corn husk cigarettes. Eighteen (25.7%) were exposed to wood smoke. Eleven (15.7%) patients had tuberculosis, 5.7% complained of asthma symptoms, 2.8% had bronchiectasis, 11.4% diabetes mellitus, 51.4% hypertension, and 20% *Cor pulmonale*. **Conclusion:** Other possible COPD etiologies must be investigated. Determinants of the pulmonary injury could be environmental smoke exposure associated to former infections. Men with low BMI are typically representative of this severe patient population. Hypertension and *Cor Pulmonale* are frequent co-morbidity factors. *(**J Pneumol** 2003;29(2):64-8)*

Key words – Chronic obstructive pulmonary disease. Smoke. Tuberculosis. Body mass index.

Abbreviations used in this article
COPD – Chronic obstructive pulmonary disease
BMI – Body mass index
FEV_1 – Forced expired volume in the first minute
GOLD – Global Initiative for Chronic Obstructive Lung Disease

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a public health problem [1]. All over the world, millions of people suffer from this illness and die prematurely due to its complications. Currently, COPD is the 12th most prevalent disease in the world and the World Health Organization reckons it will be the fifth by the year 2020. From the sixth cause of death, it will be the third in the same time period [2].

COPD is a clinical entity characterized by the presence of obstruction or chronic limitation of the airflow, presenting a slow and usually irreversible evolvement [1,3]. Bronchus obstruction is due to a pulmonary inflammatory response to noxious particles or gases.

Tobacco smoking is the main cause of COPD [4]. However, not all smokers develop COPD: only 15% of smokers present the phenotype of the disease, suggesting that, together with the individual susceptibility, additional factors are involved in the establishment of the disease [5]. The need to improve strategies for the reduction of tobacco smoking is vital, however, smoking is not the only cause of COPD and, probably, not the most important in many places in the world.

Environmental pollution, chemical exposure, inhaled smoke, passive smoking [6], viral [7] and bacterial [8] infections, alpha-1-antitripsine deficiency [5] and other associated illnesses (pulmonary or not) are considered important risk factors for the development of COPD.

COPD diagnosis seems to be more prevalent in men than in women, mainly when the findings are related to mortality and hospital admissions, suggesting higher severity of the disease for males [9]. Weight and muscle mass loss, as well as depletion of organic tissues, are frequent findings in chronic inflammatory diseases such as COPD. These alterations may indicate a worse prognosis as a consequence of impairment of the peripheral muscle function and reduction of exercise capacity [10].

The purpose of this study is to characterize a population of COPD patients assisted in the ambulatory of an outsourced health service.

MATERIAL AND METHODS

Standard questionnaires were applied to COPD patients of the ambulatory of Obstructive Lung Diseases of Hospital das Clínicas from Faculdade de Medicina da Universidade de São Paulo.

Data collected included: gender, age, weight, body mass index (BMI), smoking (starting and quitting age, years of smoking, number of years/pack, use of corn husk cigarette), contact with wood burning smoke, previous history of lung tuberculosis, associated diseases, such as asthma, bronchiectasis, diabetes mellitus, systemic hypertension and *cor pulmonale*, use of home oxygen and pulmonary function (forced expiratory volume in the first second – FEV_1).

RESULTS

Seventy COPD patients were sequentially included. Average forced expiratory volume in the first second (FEV_1) was 0.8 ± 0.42 litters or 35.5 ± 13.63% of the expected. Thirty two patients (45.7%) depended on oxygen at the time of the clinical evaluation.

Forty nine (70%) patients were male and 21 (30%), female (Figure 1). Age ranged from 40 to 83 years, with a mean of 64 ± 9.71 years. The patients´ weight varied from 38 to 110 kg, with a mean of 63 ± 15.95 kg and the body mass index, from 15.67 and 38.06 with an average of 22.46 ± 5.03 kg/m^2 (Figure 2).

Of the 70 patients studied, nine (12.8%) had never smoked; 10 were still smoking (14.3%) and 51 had quit smoking (78.8%). The mean smoking onset age was 16 ± 7.15 years and the mean smoking period was 38 ± 11.10 years/pack, 1.3 ± 0.81packs/day in average. Nine patients (12.8%) reported having smoked corn husk cigarettes.

Eighteen patients (25.7%) reported having had contact with wood burning smoke; two of them smoked a little and one had never smoked (Figure 3).

Eleven patients (15.7%) had diagnosed pulmonary tuberculosis, treated in the past. Among the associated diseases, four patients (5.7%) presented sibilant-related dyspnea and positive response to bronchodilator (12% increase of $FEV_1$and 200 ml), two (2.8%) had bronchiectasia observed at high-resolution computer assisted tomography; eight (11.4%) had diabetes mellitus, 36 (51.4%) were treated for systemic hypertension and 14 (20%) were diagnosed with *cor pulmonale* (Figure 4).

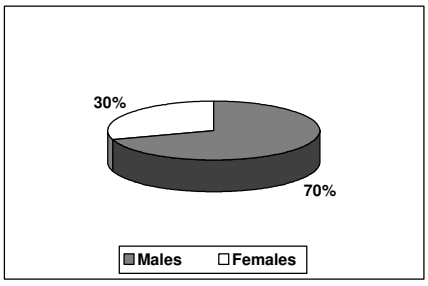

Figure 1 – Profile of a Brazilian population with severe chronic obstructive pulmonary disease. Gender distribution.

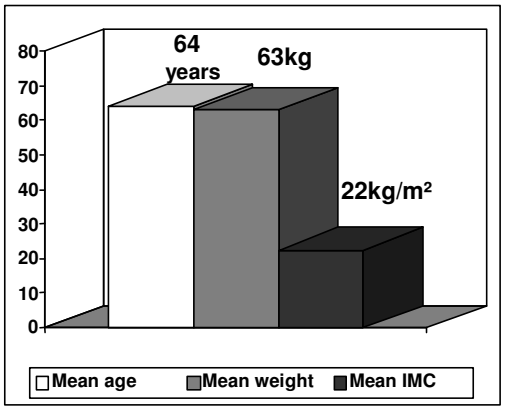

Figure 2 – Profile of a Brazilian population with severe chronic obstructive pulmonary disease. Mean age, weight and BMI.

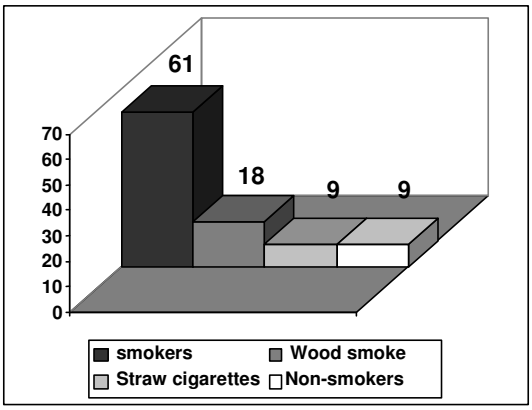

Figure 3 - Profile of a Brazilian population with severe chronic obstructive pulmonary disease. Number of patients exposed to smoke.

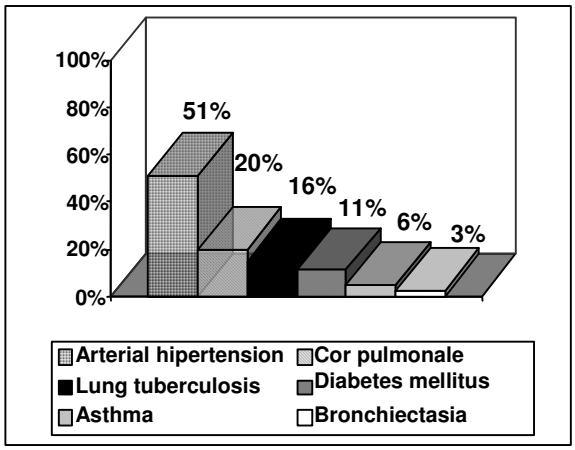

Figure 4 - Profile of a Brazilian population with severe chronic obstructive pulmonary disease. Co-morbidities.

DISCUSSION

Chronic obstructive pulmonary disease is characterized by limitation of the airflow, which is not completely reversible. This limitation is caused by an impairment of the small airway (obstructive bronchiolitis) and pulmonary destruction (emphysema). Predominance of each factor varies from individual to individual [11].

The population of patients evaluated in this study has moderate COPD, according to international patterns – Stage II B of GOLD [1], since mean FEV_1 is between 30 and 50% of expected. According to the Brazilian patterns [3], this group would be characterized as having a severe or very severe disease (stages III and IV) because, in addition to a mean FEV_1 of 35.5% of the expected, almost 50% of them were hypoxemic dependent on continuous home oxygen therapy. COPD severity in this group of patients, as well as other characteristics, is related to the limitations of the study: these are patients of a highly complex reference service, sequentially selected. The sample represented 15% of the total number of patients in the service. The use of FEV_1 alone as a COPD diagnostic criterion and severity classification allows the estimate that approximately 1.5% of North-American COPD patients are considered moderate or severe [12].

COPD incidence is higher in men than in women and increases considerably with age. The gender differences may result from the higher prevalence of tobacco smoking and occupational exposure among men. With the increase of smoking among women, the findings may change in the future [3]. Recent evidence indicate that women may be more susceptible to the side effects of cigarette smoke than men; the active search of early COPD cases by spirometry reduces the difference of COPD prevalence between men and women [9].

In this population of severe patients the BMI was lower than 25 kg/m^2, even though the upper and lower weight limits showed a large variation. Weight loss, reduction of muscle mass and tissue depletion are common findings in COPD patients. Up to 35% of COPD patients under rehabilitation present this depletion. The loss of free fat mass is directly related to the impairment of respiratory and peripheral muscles and to the reduction of the exercise capacity that occurs in COPD patients. These systemic alterations in COPD may be associated to a worse prognosis, indicating that patients with lower BMI survive less [10]. In at least one series of COPD patients, BMI under 25 kg/m^2 was an independent predictor of higher mortality [13]. Loss of free fat mass seems to be directly related to the level of systemic inflammation, as reported in several studies [10].

Three mechanisms participate in the evolvement of limited airflow in COPD: bronchial alterations with inflammation and metaplasia of caliceform cells, hypertrophy of smooth muscle and fibrosis can narrow lumen of the respiratory tract. Destruction of the alveolar walls can reduce the alveolar junctions and the elastic

shrinking of the lung, decreasing the air entry pressure, producing the collapse of small airways. Chronic bronchiolitis, peribronchial inflammation and cicatricial fibrosis of the small airway change its structure [14].

Chronic inflammation occurs as a response of the individual to smoke exposure. The reduction of tobacco use is related to health benefits and consequently, to a drop of the prevalence, morbidity and mortality of COPD patients [15]. One cigarette has more than 4,000 substances producing pulmonary lesions. However, for some years, the wood burning smoke has been recognized as a COPD pathogenic agent [16-19]. In this population of COPD, 25.7% of the individuals reported having been exposed to wood burning smoke, and in three of them, cigarette could not be the main COPD causal factor.

The inflammatory response in COPD would also be related to a chronic colonization of the respiratory airways by pathogenic agents that would amplify the response of the individual to cigarette smoke, a mechanism that has been called the "vicious cycle hypothesis" [20]. Recent studies show that infections of the respiratory tract in children make these patients become more susceptible to develop COPD from exposure to potentially noxious agents [21]. In smokers, decrease of mucociliary clearing and of autoimmune local defenses allow infectious agents (virus, bacteria) to colonize the lower respiratory tract. These pathogenic agents and their degradation products can elicit even more mucociliary damage, due to an increased production of mucous secretion, interruption of normal ciliary activity and damage to the airway epithelium [21-23].

In this sense, pulmonary tuberculosis could contribute to the COPD pathogenesis by mechanisms of chronic inflammation and destruction of pulmonary parenchyma which, added to smoking, would raise the probability of COPD evolvement [24-25]. Even though tuberculosis is poorly mentioned in the literature, it can be a developing factor of COPD in countries with high prevalence, such as Brazil. In this sample of 70 patients, 11 (15.7%) reported pulmonary tuberculosis and two (2.3%) presented bronchiectasias suggestive of previous infections/inflammations. The chronic character of the pulmonary infection/tuberculosis could contribute to the worsening of COPD [26,27].

In this COPD population, four (5.7%) patients had clinical characteristics of asthma. This number is in agreement with the international literature data, which puts the intersection between asthma and COPD in 10% [28]. The inflammatory cellular mechanisms of typical COPD differ from those of asthma [29,30]. There is, however, a group of patients characterized as COPD carriers who present characteristics common to asthma, including increased eosinophils in the exacerbated sputum. The use of steroids (oral or inhalatory) would be beneficial in this group of patients, differing of typical COPD patients, in which corticosteroids do not prevent the progressive loss of pulmonary function. The name "sibilant bronchitis" has been proposed for the pulmonary disease in this subgroup of patients [28].

The most frequently diseases associated to COPD found in this sample of patients, systemic hypertension and diabetes mellitus, could be directly related to the chronic use of systemic corticoids [31,32], even though we cannot discharge the higher incidence of chronic degenerative diseases in a population of elder smokers or ex-smokers. The existence of 20% of patients with *cor pulmonale* may be understood as a severe evolvement of the disease per se [33].

The current, and particularly the future, high morbidity-mortality of COPD, demands a better care in the way the suspected patients are investigated. The early diagnosis is essential with the determination of possible aggravating or triggering factors. COPD must be considered not as a simple disease caused by individual abuses, but rather as an addiction disease. The inhaled smoke adds to the infections in susceptible individuals, determining the pulmonary injury. Other possible etiologies must be meticulously investigated to determine other adequate and individual therapeutic actions, as well as to establish the prognosis.

In a worldwide effort to fight the disease, more national studies must be carried out to determine COPD pathogenesis, recruiting and training staff that may develop new investigation tools and techniques.

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