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THE INFLUENCE OF CARDIOVASCULAR RISK FACTORS ON PATIENS WITH BRONCHOPNEUMOPATHY

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Abstract

Chronic Obstructive Bronchopenopathy (COPD) is today a the fourth cause of death in the United States and Europe and the most important cause of morbidity and respiratory mortality. Chronic inflammation what resulting from exposure to cigarette smoking as well as other types of exposure, leads to the lesions that characterize the disease.

These consist of obstruction of small airways and destruction of parenchyma lung changes that ultimately lead to airflow limitation. Dyspnoea, chronic cough and / or sputum production, presence of some factors risk, especially cigarette smoking, must attract attention clinician on this disease, whose early diagnosis is not difficult, requiring only a simple spirometry. Pre-therapeutic staging of disease is based on the analysis of the symptoms, the degree of limit airflow (using spirometry), the risk of exacerbation and comorbidities.

Key words: Smoking, comorbidities, pre-therapeutic staging, chronic inflammation

INTRODUCTION

Chronic bronchitis involves inflammation and swelling of the mucosa of the airway leading to narrowing and obstruction of the airways. (Sin D. et al., 2006) Inflammation also stimulates the production of mucus. Obstruction of the airways, especially mucus, increases the risk of bacterial lung infections. (Mathers C.D. et al.,2006)

In the emphysema, the lung tissue and the alveoli are affected, the air being blocked inside them. The air becomes "captive" in the alveoli and reduces the ability of the lungs to reduce the time of expiration. As a result, less air reaches the alveoli for gas exchange (Broekhuizen R. et al., 2006).

The exchange of carbon dioxide and oxygen between the air and the capillary blood takes place along the thin walls of the alveoli. (Wouters E.F. 2002) Destruction of alveolar walls decreases the number of capillaries available for gas exchange. (Stephens NG et al.,1997).

Chronic obstructive bronchopneumonia is a combination of the two diseases, the more the disease gets worse, the breathing becomes more and more difficult. (Bonetti PO et al., 2003) Although the disease can be kept under control, there is currently no treatment to cure patients of this condition. (Cai H et al., 2000)

Chronic obstructive bronchopneumonia is characterized by a persistent and progressive bronchial obstruction due to structural changes in the airways (bronchitis and bronchiolitis) and pulmonary parenchyma (pulmonary emphysema). (Maadamanchi N. et al., 2005) The severity of lung disease influences the risk for some extrapulmonary determinations. Thus, in mild and moderate forms of COPD, cardiovascular co-morbidities predominate. (Bach P.B. et al., 2001)

The results of multiple clinical trials show that obstructive respiratory dysfunction is an important risk factor for cardiovascular disease both pathogenic and evolutionary. (Lindenauer P.K. et al., 2010)

VEMS, the main spirometric parameter associated with bronchial obstruction, may have, beyond diagnostic value in assessing the severity of bronchial obstruction, respectively, of chronic pulmonary disease, and a prognostic value in assessing the risk associated with the occurrence or evolution of certain cardiovascular diseases. (Gibson et al., 2005)

Epidemiological data suggest that COPD is a risk factor for atherosclerosis, with smoking as a common risk factor for both conditions, is the most important etiopathogonal link that links these two diseases. (Lindenauer P.K. et al., 2010)

It is not entirely known the intimate mechanisms by which COPD leads to cardiovascular disease, but a key role plays endothelial dysfunction.

Increasing the intimate-mean thickness index is the first observable sign of vascular damage in atherosclerosis, its substrate being fibrocellular hypertrophy and hyperplasia of smooth muscle cells in arterial blood. (Makris D. et al., 2009)

Represents the cumulative thickness of the intima and arterial mean measured at the common carotid artery, on the distal wall, approximately 10 mm before the carotid bifurcation. (Cai H et al., 2000)

The measurement is performed on an ultrasonographic image of the carotid spindle in module B in longitudinal section. It is an index that is obigatorally measured during Doppler ultrasound examination of cervico-cerebral arteries. (Anthonisen NR.et al., 2009)

Increased oxidative stress associated with cardiovascular risk factors results in vascular lesions and increased permeability of dysfunctional endothelial cells for LDL-cholesterol particles, followed by their oxidation in the intima. (Balter MS et al., 2003)

Accordingly, a number of cellular and profibrotic growth factors are released, thus stimulating the proliferation of smooth muscle cells and excess collagen production, with initiation of the formation of the atheroma plate (Balter MS et al., 2003).

This phenomenon causes arterial remodeling with increased intima-mean thickness, followed by decreased arterial distention, so-called early arterial stiffening (Makris D. et al., 2009).

If the factors that induce endothelial dysfunction persist, the atheroma plate will evolve to the vulnerable, unstable plate stage (Makris D. et al., 2009, Mraz C. et al., 2012, Pallag A. et al., 2018).

The unstable plaque is complicated by erosion or rupture, exposing prothrombotic factors such as subendothelial collagen, tissue factor or von Willebrand factor to the surface of the vessel, and triggering platelet aggregation and aggregation as well as inflammatory infiltration. This stage plays an essential role in the emergence and evolution of acute cardiovascular events. (Balter MS et al., 2003)

MATERIAL AND METHOD

The study comprised 82 patients with a mean age of 62-75 +/- 1. Patients were clinically and paraclinically examined (anamnesis, complete physical examination, blood pressure, total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol). Endothelial function was assessed by the GIM scanning of the carotid artery.

Clinical evaluation included medical history and clinical examination, including measurement and weighing for the calculation of the body mass index (BMI = weight (kg) / [height (m)], abdominal circumference (measured mid- a and antero-superior iliac spine) and the circumference of the hip (measured at the level of the big trochanter).

The diagnosis of obesity was sustained with an IMC of ≥30 kg / m2. Abdominal obesity was diagnosed according to IDF criteria (abdominal circumference greater than or equal to 80 cm for women and 94 cm for men) and ESC criteria (abdominal circumference greater than or equal to 88 cm in women and 102 cm in men) and the data obtained were analyzed separately. The harvesting and vascular paraclinical examinations were performed in the morning between 7:00 AM and 8:00 AM, after a 12-hour fasting period when the patients did not smoke or drink alcoholic beverages. Smokers have been defined subjects who have regularly smoked for more than 10 years prior to enrollment.

All numerical variables were expressed as mean ± standard deviation (SD). Pearson's correlation analysis was conducted to determine whether IMT, ABI results correlated with risk factors for carotid arterial disease. Possible correlations between IMT, ABI and the Syntax score were

evaluated by linear regression analysis. Potential predictors of the Syntax score were tested with multiple regression analysis.

RESULTS AND DISCUSSION

The risk of carotid arteriopathy increased in the proportion of patients: 90% had intimate-mean thickness (IMT) above 0.9 mm, and 53.4% had atherosclerotic plaques at carotid level. The risk of peripheral arteriopathy was identified in 18.95% of patients, exhibiting values below 0.9 of the ankle-arm index (ABI).

Table 1.

Characteristics of the patients included in the study

Variable	Average	Standard deviation
Age	67,32	9,10
TAS	132,52	14,602
TAD	87,75	12,201
HDL cholesterol	37,82	6,305
LDL cholesterol	115,85	11,605
Total cholesterol	195,55	14,624
Triglycerides	150,11	39,99
Syntax score	18,95	9,93
ABI	1,55	0,25
IMT (mm)	0,957	0,165
obezity	n=49,5	46,8%
HTA	n=55	51,5%
Smoking	n=58	53,4%

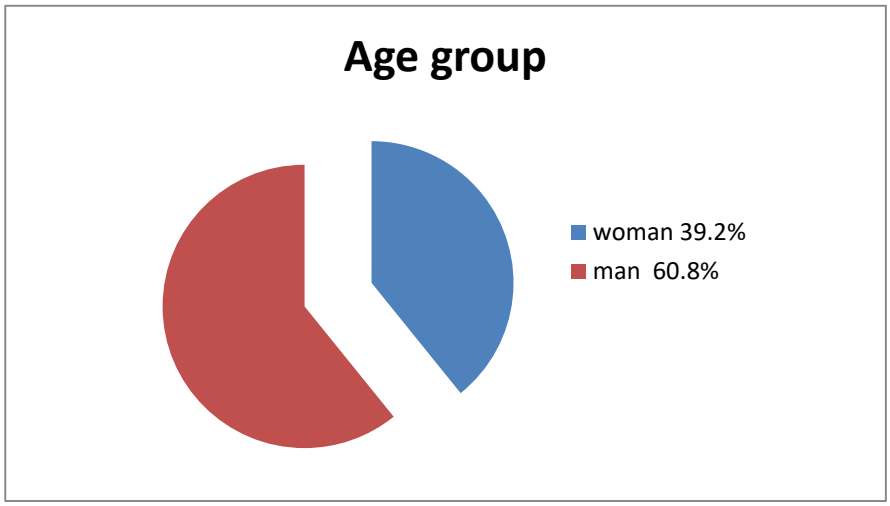

Fig. 1. Distribution of the number of associated cardiovascular risk factors by age group

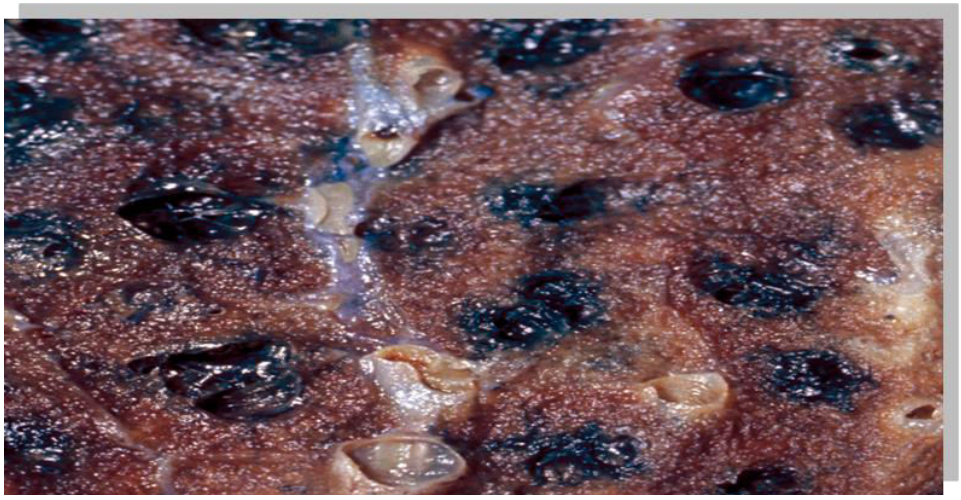

Fig.2. Emphysema characteristic of patients over 10 years of smoking

From the point of view of the ultrasound scan of the mean carotid artery thickness, the values were above the 0.9 mm threshold in most patients (90%), even in those who had no history of vascular disease, indicating vascular disease as comorbid common in patients with COPD.

Significant correlations of the intima-mean thickness at carotid level with systolic pressure in the pulmonary artery and the ankle-arm index with hematocrit were determined, important in the context in which the chronic increase in systolic pressure in the pulmonary artery in patients with COPD and hematocrit , under the conditions of chronic hypoxemia, could increase the risk of carotid or peripheral vasculopathy.

CONCLUSIONS

Old age, smoker status (increased number of packets per year) have been shown to be risk factors for the coexistence of carotid vasculopathy in patients with COPD.

Average for average interstitial thickness was higher for smokers with obstructive dysfunction than for healthy smokers, respectively non-smokers. Increase in mean mean thickness correlated significantly with VEMS decrease. C-Protein is an important indication of systemic inflammation associated with COPD. On the other hand, the importance of inflammation in the atherosclerotic process is known.

From this point of view, the study supports the hypothesis of a pathogenic link between COPD and atherosclerosis in the systemic inflammatory process. Thus, the presence of atherosclerotic plaque

correlated with CRP increase, independent of the role of age, smoking, body mass index, blood pressure, glycemia, cholesterol, heart rate.

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