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RESEARCH ARTICLE

Non-invasive assessment of endothelial function of systemic arteries during obstructive chronic bronchopneumopathy in African subjects

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is characterized by high mortality. The latter is strongly attributed to cardiovascular events whose onset is endothelial dysfunction and vascular remodeling. **Aims and Objectives:** The aims were to evaluate the endothelial function during COPD and the factors involved with possible endothelial dysfunction in Senegalese Black men aged at least 18 years. **Materials and Methods:** This was a prospective and cross-sectional study. After a complete clinical examination and evaluation of the body composition by a bioimpedancemeter, the endothelial function was evaluated by EndoPAT2000®. **Results:** the mean age was 61.50 years ± 6.62. Of the subjects, 70% were smokers and 50% had limited chest enlargement. According to the body mass index and the percentage of body fat, underweight was 30% and 40%, respectively. The 75.5% of the meager could not be evaluated by bio-impedancemeter due to extreme thinness. Endothelial dysfunction involved 70% of subjects. In addition, 86% of subjects with endothelial dysfunction were smokers. The reactive hyperemia index was positively correlated with body mass index ($P = 0.017$ and $r = 0.73$), body fat percentage ($P = 0.016$ and $r = 0.73$) and visceral fat level ($P = 0.008$ and $r = 0.78$). **Conclusion:** The evolution of COPD would be laced with serious cardiovascular complications often fatal. The assessment of nutritional status and endothelial function is fundamental for better management of the disease.

KEY WORDS: Chronic Obstructive Pulmonary Disease; EndoPAT2000®; Endothelial Dysfunction; Reactive Hyperemia Index; Senegalese Black Men

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic respiratory disease with progressive evolution. It is a chronic noncommunicable disease such as obesity, hypertension, and diabetes. According to Jeeva and Bhattacharya, the prevalence of chronic noncommunicable diseases is on the rise and thus becomes a public health problem.[1] COPD is characterized by a partially or completely irreversible obstructive ventilatory syndrome.[2] Its symptomatology is dominated by the pulmonary signs, but in reality, these would be only the visible part of the iceberg. While for a long time considered a disease local inflammatory that is to say bronchopulmonary, COPD is currently conceived as a systemic disease.[3,4] Observational studies have shown that during COPD there is a systemic inflammatory process, generalized hypoxia,

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oxidative stress, and activation of the sympathetic nervous system.[4] This could attest to its systemic nature. In addition, COPD is characterized by high mortality[5] which would be largely attributed to cardiovascular events. An increased risk of cardiovascular morbidity and mortality has been found in patients with COPD.[6-8] Compared to same-age and same-sex subjects without COPD, patients with COPD who die from cardiovascular events are significantly higher.[9] Large epidemiological studies such as the Framingham heart study and the Copenhagen city heart study have shown that a decrease in peak forced expiratory volume in one second (FEV1) during COPD is associated with an increased incidence of cardiovascular events.[4] In the lung health study, it was noted in patients with mild to moderate COPD that a 10% reduction in the predicted FEV1 value is associated with a 28% increase in the frequency of coronary events fatal.[10,11] Authors say interactions between cardiovascular and respiratory pathologies are not fully understood, but COPD and asthma are associated with increased risk of ischemic heart disease, stroke, and cardiovascular death.[12-14] Thus, the cardiovascular component is considerable during COPD. Recent evidence has shown that COPD is associated with a range of comorbidities that may worsen its course.[4] Shared risk factors and physiopathological mechanisms, including systemic inflammation,[15] cardiovascular, and respiratory diseases coexist frequently. Thus, cardiovascular complications during COPD may be related to the comorbidity noted and that would be predominated by conventional cardiovascular risk factors. However, authors have shown that COPD is at the same time an independent cardiovascular risk factor.[16] Moreover, bronchial obstruction and hypoventilation of pulmonary capillaries in COPD would be largely caused by vascular remodeling. The mechanisms involved in the pathogenesis of this vascular remodeling have been examined to date by the little study. Some studies have shown stiffening of the arteries and endothelial dysfunction in patients with COPD.[17] Most studies, which measured endothelial function during COPD, were limited to the pulmonary vessels and were rarely concerned with systemic vessels[18] and they used methods that could vary depending on the operators. In addition, no study of endothelial function during COPD in African Black subjects, especially with EndoPAT2000® as a measurement tool, has not been done to our knowledge. Thus, the objective of our study was to evaluate the endothelial function in the systemic vessels and to determine the factors involved in possible endothelial dysfunction in Black Senegalese patients with COPD.

MATERIALS AND METHODS

Our study protocol has been validated in the Department of Physiology and functional investigations of the Faculty of Medicine, Pharmacy, and Odontology of Cheikh Anta Diop University (UCAD) in Dakar, Senegal. It has been designed in accordance with the guidelines of the Helsinki declaration and has been approved by the UCAD ethics committee. All subjects included in this study were informed of the purpose and interest of the work and signed a written consent form.

This was a prospective, cross-sectional and analytical study that was conducted in the physiology laboratory over a period of 7 months (April–October 2017). The study included 10 men with COPD recruited from the physiology department, from the respiratory functional exploration registry. In this laboratory, which includes various exploration rooms such as a respiratory and cardiovascular exploration room. We used the EndoPAT2000® measuring device and the computer on which the recording and analysis software is installed.

We included subjects aged at least 18 years, who had previously been diagnosed with COPD and confirmed by spirometry with beta-2-mimetic reversibility test.

An anamnesis was conducted in each subject followed by a complete physical examination including anthropometric parameters, clinical constants, and body composition parameters.

The size was measured using a tape measure to the nearest centimeter. The measurement of the thoracic perimeter at rest, then in deep inspiration and finally in deep expiration were made thanks to a tape measure. The thoracic amplification, equal to the difference of the value of the deep inspiration to that of the deep expiration could thus be calculated. The difference was considered abnormal when it was <7 cm in men.[19]

The weight of the subjects was measured by leading scales Secca® in kg near. Then, the body mass index (BMI) was calculated according to the Adolphe Quetelet formula of 1869 (BMI = Weight/Height^2) and then classified according to the standards of the World Health Organization of 1995.

The body composition parameters: Body fat percentage (BFP), lean mass (LM), and visceral fat level (VFL) were measured using an Omron® brand bio-impedancemeter. The reference values of the body composition parameters were taken according to the recommendations of Moreno MV and Oliveira EP.[20,21]

The Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) were measured using an Omron® electronic sphygmomanometer with cuff adapted to the subjects' arms. This decision was made respecting the required conditions (for example, after at least 10 min of rest). According to the recommendations of the ESH/ESC in 2007 for the management of hypertension, high blood pressure was considered effective when the SBP was ≥140 mmHg and/or the DBP was ≥90 mmHg.

The mean arterial pressure (MAP) was calculated according to the formula of Messai E. Ed. Arnette Blackwell (Paris) 1995: Mean blood pressure = (SBP + 2 ×DBP)/3.

The blood oxygen saturation (SaO_2) was assessed using a quirumed brand digital oximeter, model CMS50D, and reference OXYM4000. The heart rate was also measured in beats per minute (bpm) with the same Omron® electronic sphygmomanometer. A heart rate was considered normal when it was between 60 and 100 bpm, such as bradycardia when it was below 60 bpm, and as a tachycardia when it was >100 bpm.[22].

The parameter of the endothelial function we had chosen to analyze was peripheral arterial tone. It is a parameter that reflects endothelial responsiveness. It was evaluated using a device called EndoPAT2000®. It is a device that is used in pre-eminent clinical facilities, research centers, and clinical phase pharmaceutical studies. The results are not the dependent operator. It is an easy-to-use device, ensuring automatic and immediate availability of test results. It provides a reproducible index of endothelial function after a 15 min test. Its operating principle is based on the measurement of peripheral arterial tone using disposable biosensors placed on the index fingers of both hands.

In a patient lying on an examination table, the device measures endothelium-mediated changes in arterial tone in peripheral vascular pathways. These changes in arterial tone are caused by creating a hyperemic response induced by a standard 5 min occlusion of the brachial artery performed with an inflatable cuff. Contralateral arm measurements are used to control competing for non-endothelial changes dependent on the vasculature. The automatically calculated result is an index of endothelial function: Reactive hyperemia index (RHI) [Figure 1]. It reflects the endothelium-dependent vasodilatation of the subject's systemic blood vessels and thus allows for detect subclinical endothelial dysfunction. The normal value of RHI is at least 1.67. An RHI value below 1.67 can therefore be considered endothelial dysfunction.[23]

Statistical Analysis

All variables were saved in an excel table. Quantitative variables were described using mean ± standard deviation, and qualitative variables were described using absolute values and percentages. The student's *t*-test was used to compare the mean of the quantitative variables. Spearman correlation tests were performed to investigate associations between endothelial function and the other parameters studied. The results are considered significant for a value of $P < 5\%$. The exploitation of the data was carried out thanks to SPSS software version 16.0.

RESULTS

Descriptive Results

Characteristics of patients and disease

The mean age of the study population was 61.50 ± 6.62 years (with extremes of 50 and 71 years). Seven subjects (70% of the study population) were smokers, and five subjects (50% of the population) had a physical activity that was limited to regular walking. Only one subject (10%) was hypertensive known and followed. There was no diabetes or dyslipidemia in the subjects.

The thoracic amplification we measured was low in half of the population (50% of subjects) with values of 5 and 6 cm. We found variable symptomatology from one patient to another which is summarized in Table 1. More than half of our patients were in the category of moderate COPD according to the GOLD 2017 classification. We noted only one case of very severe COPD.

Evaluation of BMI and body composition parameters

The mean BMI in the study population was 19.76 kg/m^2. For the body composition, three subjects (30% of the population) could not have an assessment of their body composition by bio-impedancemeter due to their extreme thinness, only the 7 remaining subjects were evaluated, and the LM and BFP were below the normal range of the adult in most of the cases. The means of these parameters are reported in Table 2. According to BMI and BFP, underweight was found in 30% of subjects in the study population. We found that 6 subjects (60% of the cases) were in low LM.

Study of cardiovascular constants

We found that MAPs and mean heart rate were normal in the study population. Mean SaO2 was 95 ± 4.4% at rest; all subjects had a saturation below 99%, Table 3.

Assessment of endothelial function

The mean of RHI was 1.63 ± 0.42 in the population studies. In addition, 70% of patients with COPD had RHI below normal value. The 86% of subjects with endothelial dysfunction were smokers. All lean BMI subjects had endothelial dysfunction while overweight subjects had preserved endothelial function. Depending on the level of visceral fat, the only subject with abdominal obesity had normal endothelial function.

Analytical Results

Bivariate analyzes

After Spearman correlation tests, RHI was positively correlated with BMI ($P = 0.017$, $r = 0.73$), total body fat ($P = 0.016$, $r = 0.73$), and level of visceral fat ($P = 0.008$, $r = 0.78$).

Table 1: Pulmonary signs noted in our patients

Pulmonary signs	*n* (%)
Cough/sputum	4 (40)
Dyspnea	9 (90)
Sibilant rattles	5 (50)

Table 2: Study of mean BMI and body composition parameters

Variables	Mean±SD
BMI (kg/m²)	19.76±5.06
LM (%)	23.78±16.50
BFP	14.30±10.66
LVF	6.57±4.19

BMI: Body mass index, LM: Lean mass, LVF: Level visceral fat, BFP: Body fat pourcentage, SD: Standard deviation

Table 3: Mean cardiovascular constants of the study population

Variables	Mean±SD
HR (bpm)	86.20±17.96
SBP (mmHg)	127.00±17.67
DBP (mmHg)	78.00±14.76
MAP (mmHg)	94.33±12.18

Bpm: Beats per minute, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, SD: Standard deviation, HR: Heart rate

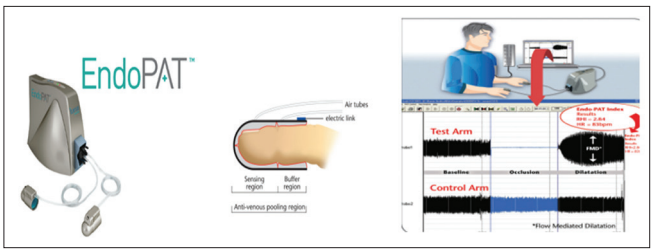

Figure 1: Plot obtained during a vascular evaluation with an EndoPAT2000® device

Multivariate analyzes

After linear regression tests, we found that among the parameters that were associated with the RHI, none was independently influential on the RHI.

DISCUSSION

We had worked on a male population with COPD. The subjects were relatively old with a mean age of 61.50 ± 6.62 years. Half of the subjects were strictly sedentary and the others were only regular. The majority of the study population (70%) was smokers. The mean of BMI was 19.76 kg/m² ± 5.06 and the LM and BFP were below normal adult values. Extreme thinness was noted in 30% of subjects. All subjects had arterial oxygen saturation below the normal lower limit, but their arterial pressures were in normal ranges. The mean value of RHI in the study population was 1.63 ± 0.42. In addition, 70% of patients had impaired endothelial function with a predominance of subjects who were smokers 86% of endothelial dysfunctions was positively correlated with BMI ($P = 0.017$, $r = 0.73$), BFP ($P = 0.016$, $r = 0.73$), and VFL ($P = 0.008$, $r = 0.78$). However, none of these parameters was independently associated with endothelial function.

These results have shown that COPD is responsible for endothelial dysfunction. This corroborates the data of the literature. Indeed, alteration of endothelial function during COPD has been demonstrated by many authors.[24] Some studies have even reported that COPD patients have much greater endothelial function impairment compared to control smokers or non-smokers.[25,26] Similarly, impaired lung function has been associated with the presence and development of atherosclerotic lesions.[27-29] However, a direct association between the severity of COPD and the occurrence of endothelial dysfunction has not been objectified.

The alteration of endothelial function in COPD is thought to be related to intimal thickening, the proliferation of smooth muscle cells in the media, collagen and elastic fiber deposits. This remodeling of the vessels is partly responsible for bronchial obstruction and alveolar hypoventilation.[30] In our study population, smokers and former smokers were the most affected by endothelial dysfunction. These results go in the same direction as the data from the literature. Smokers with COPD have an arterial relaxation disorder related to vascular endothelial cell dysfunction[31] which would be a consequence of the absorption of tobacco components.[32] However, Ives *et al*. had shown an alteration of endothelial function in patients with COPD but without a direct association between endothelial dysfunction and the history of smoking.[33] The subjects in the study were relatively old, and they were all male. We did not find a direct association between the endothelial dysfunction of our patients and their age which may be related to the weakness of the workforce. However, vascular function declines progressively with age and aging would result in a decrease in arterial compliance[34] and the occurrence of endothelial dysfunction.[35] In addition, Zureik *et al*., in their study, said that men were more prone to intima-media thickness because they found a positive association between bronchial hyperresponsiveness and high intima-media thickness values in men of the common carotid artery showing underlying atherosclerosis[36] whose bedrock is endothelial dysfunction.[37] In addition, Herinirina NF. reported in his study that age and gender were the determining factors of intima-media.[38] A positive association was found between endothelial function and BMI, BFP, and VFL in our patients. In addition, endothelial dysfunction was more noticeable in the lean subjects of our study. A change in body composition would be associated with the decline in vascular function. Japanese studies have established a positive association between thickness-intima-media (TIM) and excess fat, especially of visceral localization.[39] According to Juo, obesity and TIM share the same genetic factors.[40] In our study, however, we found a negative association between thinness and endothelial function. The existence of undernutrition during COPD is widely described in the literature,[41] and it appears that exacerbation and death

are more common in malnourished COPD patients.[42] Then, endothelial dysfunction could be one of the adverse effects of undernutrition during COPD with the consequent increase in cardiovascular events.

The results we obtained allow us to say that the endothelial dysfunction described during COPD is well established. In addition, this is a generalized endothelial dysfunction which would partly explain the increase in the incidence of cardiovascular events observed during COPD. The risk of endothelial dysfunction occurring during COPD would be increased by concomitant with classical cardiovascular risk factors (age, smoking, undernutrition, etc.). However, studies on larger cohorts are needed to better establish these results.

The originality of our work lies in the fact that, to the best of our knowledge, this study is the first that describes the endothelial function during COPD in Sub-Saharan African Black subjects. It is also the first study that uses EndoPAT2000® as a tool for measuring endothelial function in patients with COPD. Previous studies were based on the manual measurement of flow-mediated dilatation (FMD), which is based on measuring the size of the humeral artery before and after occlusion of arterial flow with a cuff inflated to 200 mmHg for 5 min.[25,43] EndoPAT2000® is a new tool that allows ambulatory measurement of endothelial function. It allows objective measurement, simple, fast, non-invasive, reproducible, and above all not dependent on the operator. Its use as a tool for evaluation of endothelial dysfunction and the predictive interest of it has already been validated by other authors.[44,45]

Indeed, RHI measured by EndoPAT2000® evaluates endothelium-dependent vasodilatation and accurately reflects endothelial responsiveness. The lower the value of the index of reactivity and <1.67, the more the endothelial function is impaired.

It is, therefore, a method that can be used in exploration laboratories and clinical services and could reduce diagnostic delays.

The limitations of our work were related to the small size of our study sample, which was mainly due to the fact that we did not include subjects with very severe COPD in the study.

It is, therefore, necessary to pay more attention to the increased cardiovascular risk of patients with COPD for better management of the disease.

CONCLUSION

These results allowed us to understand that alongside pulmonary signs, vascular status, and changes in body composition play a role in the evolution of COPD. These are cardiovascular risk factors that explain the high morbidity and mortality noted during COPD. It is, therefore, necessary to pay more attention to cardiovascular risk in the management of patients with COPD.

Subclinical screening for the impaired endothelial function may be a means of predicting the occurrence of cardiovascular complications during this disease. It could allow the implementation of preventive strategies. To do this technique such as Doppler ultrasound coupled with the measurement of FMD are possible. However, a fast, non-invasive technique is easy to use and reproducible,regardless of the operator has recently seen the day, and could also be used.

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