Article

Assessment of Some Aspects of Cardiovascular Function Status in Male Patients with Stable COPD

Magfura Pervin[1], Noorzahan Begum[2], Taskina Ali[3]

Abstract

Background: COPD is a preventable and treatable disease with some important extra pulmonary effects, which may contribute to the severity of this disease. Among these extra pulmonary ailments cardiovascular disorders are noteworthy. Although its silent involvement is known, but little attention paid to this major comorbidity while treating COPD patients. **Objective:** to assess rate, systolic blood pressure (SBP) and peak systolic velocity (PSV) of blood flow to observe the cardiovascular function status in stable COPD patients **Methods:** This cross-sectional study was carried out in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka during 2010. For this purpose 60 (sixty) male, smoker (5 to 10 pack years) patients of stable COPD (FEV_1/FVC% <0.70; FEV_1 e" 80%; without any exacerbation for last 4 weeks) were randomly selected from the Out Patient Department of the Department of Cardiology, BSMMU and also from a private clinic in Dhaka city. On the basis of spirometric findings, 30 mild (group B_1) and 30 moderate stage (group B_2) of COPD patients with age 35-45 years were included in the study. In addition, 30 age and BMI matched apparently healthy male persons were studied as control (group A). For assessment of cardiovascular function, pulse rate, SBP and PSV were measured. PSV of blood flow in distal part of the right brachial artery in arm was measured by Color Doppler ultrasonography. For statistical analysis, Independent sample t-test and Pearson's Correlation Coefficient test were used. **Results:** Significantly ($p<0.001$) higher mean pulse rate and SBP were observed in moderate stage of COPD patients than those of control and mild stage. PSV was significantly ($p<0.001$) higher in both stages than those of the control as well as in moderate stage to that of the mild COPD. In addition, SBP and PSV were negatively correlated with FEV_1 in moderate stage which was statistically significant ($p<0.01$). **Conclusion:** This study suggests that, cardiovascular status may be altered in stable COPD and this alteration is inversely related to the severity of the disease.

Key words: PSV, blood flow, cardiovascular function status, COPD.

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For Authors Affiliation, see end of text.

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is one of the major causes of chronic morbidity and mortality throughout the world[1]. Once developed, this disease along with its comorbidities can not be cured totally. However, its progression and consequences can be reduced.[1]

Many people suffer from this morbid disease for years and die too early from its complications. It is the fourth leading cause of death in adults of USA & also projected to be the third by 2020.[2-4]

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COPD is also becoming a rising burden for both developed and developing countries day by day. This upsurge of the morbidity is thought to be due to urbanization, industrialization and change of profession of people from 'agriculture and fresh air' based rural communities to 'industry and smoking based urban settings in our country. Total burden of COPD patients in Bangladeshi population is about 6 million now.[5]

The pulmonary component of COPD is characterized by airflow limitation which is usually progressive and not fully reversible. Along with the pulmonary changes it has also various extrapulmonary (systemic) effects, such as raised circulatory inflammatory markers and polycythemia.[6] These systemic effects may lead to different comorbid conditions, such as ischemic heart disease and cardiac failure[6-8]. It has been proposed that about 50% death in COPD patients may result from cardiovascular cause rather than pulmonary cause itself.[9] In addition, the cardiovascular risk has been reported to be independent of the effects of smoking and other factors like physical fitness, aging process, life style etc in this group of patients.[10] However, airflow limitation and persistent low grade systemic inflammation may increase the cardiovascular risk in patients with COPD.[3]

Findings of some recent studies suggested that COPD itself is an important risk factor for different manifestations of cardiovascular diseases.[11-13] Within these, increased pulse rate is the commonest presenting symptom due to chronic hypoxia in this group of patients.[1] Significantly higher values of pulse rate has been observed in COPD patients when compared with the healthy control by different investigators.[2,3]

In addition, increased systolic blood pressure (SBP) or isolated systolic hypertension has been proposed to be a direct risk factor of coronary heart disease.[14] SBP is influenced by stiffness of large arteries and left ventricular ejection pattern[15]. Many investigators of different countries reported higher values of SBP in patients with COPD.[2,11,13,16,17]

Moreover, there has been much recent interest in the relationship between arterial stiffness of large arteries and different cardiovascular diseases.[18,19] Another group of researchers proposed that arterial stiffness is independently associated with the severity of emphysema in COPD patients.[12] Basically arterial stiffness describes the rigidity of the arterial wall and used to express the qualitative property of an elastic vessel wall[18]. It has been reported that this arterial stiffness can be assessed indirectly by measuring peak systolic velocity (PSV) of blood flow in any peripheral artery.[20] PSV has also been used to detect vascular stenosis which might be due to increment of arterial stiffness.[21] PSV of different arteries in patients with COPD were studied and its higher values were reported.[22,23]

In Bangladesh, no data is available to document the changes in pulse rate, SBP and PSV of blood flow in COPD patients.Although it has been known that there is silent cardiac involvement in COPD patients but little attention is paid to this issue while treating this group of patients.

Therefore, the study was undertaken to assess the pulse rate, SBP and also PSV as cardiovascular function status in male patients with stable COPD.

Methods

This cross-sectional study was carried out in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka between January to December, 2010. Protocol of this study was approved by the Institutional review board of this university. For this study, 60 male, age (35-45 years), patients of stable COPD (without any exacerbation for last 4 weeks[12]) were randomly selected from the out patient department of Department of Cardiology, BSMMU and also from a private clinic in Dhaka. On the basis of spirometric staging, patients were divided into mild

(group B_1) (FEV_1 > 80% of predicted; n=30) and moderate (group B_2) (FEV_1 < 80% but > 50% of predicted[5]; n=30) stage of COPD. For comparison, 30 age, BMI (24.7 to 25.6 kg/m^2), serum lipid profile (cholesterol: 150 to 200 mg%; TG: 50 to 150 mg%; HDL: >40 mg%; LDL: <150 mg%[24]) and smoking status (5 to 10 pack years[25]) matched apparently healthy male persons as control(group A) were randomly selected from the community. Any subject with diabetes mellitus (Fasting plasma glucose >7 mmol/dl[26]), systemic hypertension (SBP > 140 and DBP > 90 mm of Hg[27]) before the diagnosis of COPD, with any pulmonary comorbidity (e.g, bronchiectasis, pulmonary fibrosis, pneumonectomy, lobectomy) or any other systemic disease (e.g, rheumatoid arthiritis, connective tissue disorder), treatment with long term steroid or theophylline or any immunosuppressive therapy or with history of any heart disease, were excluded from the study[13].

After selection, all the subjects were thoroughly informed about the aim, benefit and procedure of the study and were encouraged for voluntary participation. An informed written consent was taken from them. The subjects were requested to attend the Department of Physiology at 7.30 am in a fasting state on the day of examination. In addition, all of them were also instructed to abstain from tea, coffee or any type of smoking for at least 12 hours before that very day. At the examination day, their detailed personal, medical and drug history were taken and thorough physical examinations were done. All information was recorded in a pre-fixed questionnaire.

Then with all aseptic precautions 5 ml of venous blood was collected from left ante-cubital vein to measure the serum lipid profiles and glucose to exclude hypercholesterolemia and diabetes mellitus. Cardiovascular status of all subjects was assessed by counting pulse rate, measuring SBP and PSV. PSV of blood flow in the distal part of the right brachial artery in arm was measured by Color Doppler ultrasonography in the Department of Cardiology, BSMMU, Dhaka. PSV measurement was done by using a vascular transducer (L10-5*), with operating frequency of 5-10 MHz in a thermally controlled room, after an accommodation period for at least 5 to 7 minutes rest in supine position. Cut point for pulse rate > 90 beats/min[28], for SBP > 140 mm of Hg[27] and PSV > 100 cm/sec[29] were considered for increased values of these parameters.

All the data were expressed as mean±SD. For statistical analysis, Independent samples t-test and Pearson's Correlation Coefficient test were performed by using SPSS for windows version-12, as applicable and $p<0.05$ was accepted as significant.

Results

Baseline data of all the subjects are given in Table I. Mean pulse rate and SBP were higher in both the study groups compared to control, which were statistically significant only in B_2 ($p<0.001$). In addition, the mean PSV were significantly ($p<0.001$) higher in both the study groups than that of control, as well as in B_2 than that of B_1 (Table II). Moreover, in group B_2, 12(40%), 23(77%) and 18(60%) patients were with increased pulse rate, SBP and PSV, respectively (Figure 1).

Again, pulse rate was negatively correlated with FEV_1 in group B_2 but positively in A and B_1. SBP was negatively correlated with FEV_1 in both the study groups, but positive in control subjects. Moreover, PSV was negatively correlated with FEV_1 in group A and B_2 but positively in B_1. However, these relationships were statistically significant ($p<0.01$) only in B_2 for SBP and PSV (Figure 2).

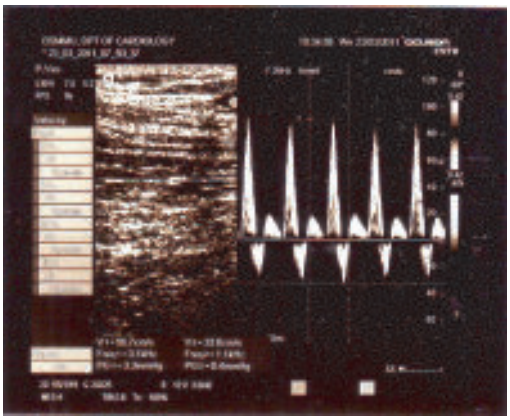

Diagram 1: *Measurement of PSV by color Doppler ultrasonography as V_1*

Table I: Baseline characteristics in different groups (n=90)

Groups	Age (years)	BMI (Kg/m^2)
A	47.1±5.29	25.6±2.24
(n=30)	(37-55)	(21-30)
B_1	47.2±6.04	25.0±2.3^{ns}
(n=30)	(35-55)	(21-30)
B_2	47.7±5.32	24.7±3.13 ^{ns}
(n=30)	(35-55)	(19-32)

Data were expressed as mean±SD. Figures in parentheses indicate ranges.
For statistical analysis, one way ANOVA test was done for comparison among the groups
A = apparently healthy subjects (Control)
B = COPD patients (Case)
B_1 = Mild stage
B_2 = Moderate stage
ns = non significant

Table II: Mean±SD of pulse rate, SBP and PSV in different groups (n=90)

Groups	Pulse (beats/min)	SBP (mm of Hg)	PSV (cm/sec)
A	75.1±5.84	118.2±8.25	61.06±4.01
(n=30)	(68-88)	(100-130)	(52.7-68.4)
B_1	76.7±5.57 ^{ns}	120.7±6.91 ^{ns}	70.12±6.18****
(n=30)	(68-88)	(110-130)	(56.3-79.4)
B_2	86.6±6.97****	128.8±8.78****	108.59±18.76****
(n=30)	(72-98)	(120-145)	(80.3-145.2)

Figures in parentheses indicate ranges.
For statistical analysis, one way ANOVA test was done for comparison among the groups
A = apparently healthy subjects (Control)
B = COPD patients (Case)
B_1 = Mild stage
B_2 = Moderate stage
ns = non significant

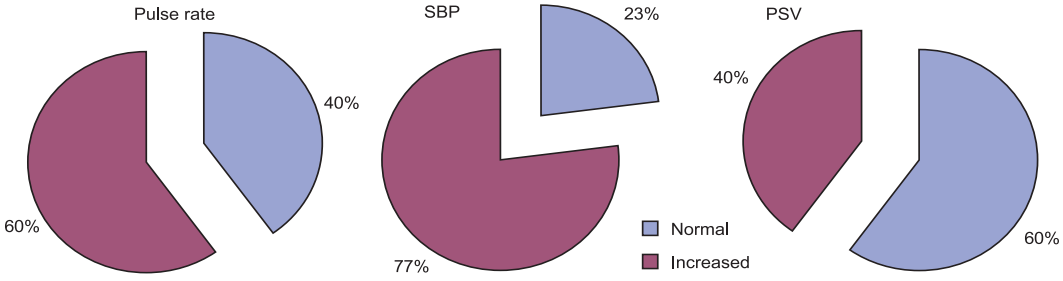

Figure 1: Frequency distribution of subjects by increased Pulse Rate, SBP and PSV in moderate COPD

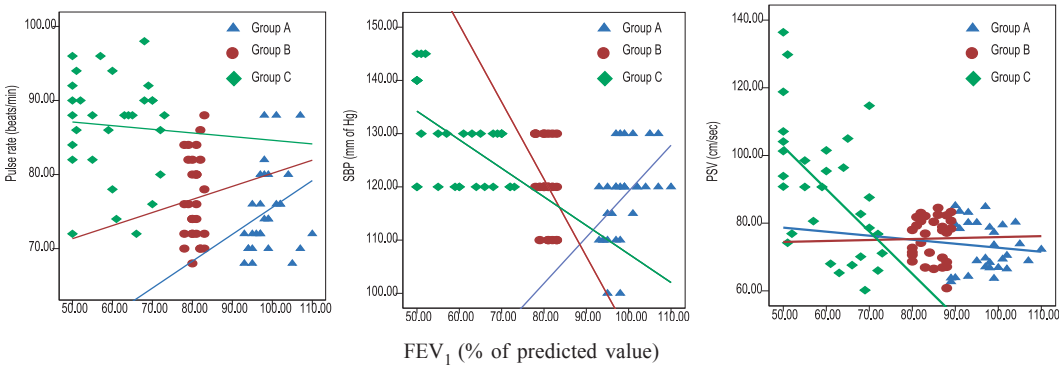

Fig 2: Correlation of Pulse rate, SBP and PSV with FEV_1 in different groups (n=90)
A = apparently healthy subjects (Control), B_1 = COPD patients (in mild stage),
B_2 = COPD patients (in moderate stage)

Discussion

In the present study, findings of all the variables in healthy control group were within the normal range and also similar to those of the observations of various investigators.[2,4,11,13,30]

In this study, mean pulse rate and SBP were significantly higher in moderate COPD than control and mild stage of COPD. These findings agree to the findings of other researchers.[2,3,13,16] In addition, PSV of the brachial artery was significantly higher in COPD patients than control and also in moderate than mild COPD. Similar findings were also reported by several investigators.[22,23]

Literature review suggested several mechanisms for these changes in cardiovascular function of COPD patients.[1,3,31,32,33,34]

It has been suggested that the chronic airflow limitation in the COPD patients might be attributed to the development of chronic hypoxia,[1] resulting an increment of pulse rate in COPD patients. In this study, it was supported by the increased pulse rate among 40% moderate COPD patients and its negative correlation with the severity of airflow limitation.

In addition, persistent low grade systemic inflammation in COPD may cause excessive neutrophil elastase activity resulting increased consumption of elastic fibers of large arteries, leading to the development of arterial stiffness.[34] Other researchers proposed that elastin (core portion of elastic fiber) might be involved in the regulation of vascular smooth muscle cell functions in COPD patients. As the elastin consumed, there might be vascular smooth muscle cell dysfunction followed by the development of arterial stiffness[31]. So, the increased PSV of blood flow in COPD patients of the present study might be due to increased arterial stiffness.

Along with the PSV, arterial stiffness has also been proposed as a causative factor for the decrement of vascular compliance.[33] Moreover, it has also been proposed that there might be early structural and functional changes in the vessel walls of the large arteries in COPD patients.[2] However, reduced vascular compliance[28] in addition to early structural changes[2] in the vessel wall of stable COPD patients might also be the cause of increased SBP in these study population.

Conclusion

From this study, it may be concluded that the cardiovascular status may alter in stable COPD patients and which is inversely related to the severity of airflow limitation.

Authors Affiliations

1. Magfura Pervin, Assistant Professor, Department of Physiology, Tairunnessa Memorial Medical College and Hospital, Gazipur, Dhaka. Email: dr.magfura@gmail.com Tele: 01720380416
2. Noorzahan Begum, Professor, Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka. Email: noorzahan52@gmail.com
3. Taskina Ali, Associate Professor, Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka. Email: taskinadr@gmail.com

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