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Original Research Article

STUDY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AMONG SMOKERS AT OUR TERTIARY HOSPITAL

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Abstract

Background: Tobacco smoking has been considered as the leading cause underlying rising prevalence of COPD across the globe. Smoking whether active or passive is a well-known risk factor for general health. Present study was aimed to study of chronic obstructive pulmonary disease among smokers at our tertiary hospital. **Materials and Methods:** Present study was prospective, observational study, conducted among 100 male subjects between 19-58 years of age. They were further divided into-25 complete non-smokers (Group I), 25 mild smokers (<5 pack year), 25 moderate smokers (<5-10 pack years) & 25 chronic smokers (>10 pack years) (Group II). Pulmonary function test values FVC, FEV1, FEF 25-75%, PEFR were noted. **Result:** There was no statistically significant difference between the mean age height, weight and body surface area of smokers & non-smokers. Value of FEV1 in mild smokers was on the lower side in comparison to the control group and p-value was not significant. The value of FEVI of moderate and chronic smokers and the p-value was statistically significant ($p<0.001$). The value of FEV1/FVC in moderate and chronic smokers were much lower as compared to the control group and p-value was statistically significant ($p< 0.05$). Value of PEFR in moderate and chronic smokers was lower than the control group and the P-value was statistically significant ($p<0.001$). The results of the present study were in deceasing trends in the values as we proceed from non-smoker to heavy smokers. Value of MVV in moderate and chronic smokers was less than that of non-smokers and the P-value is statistically significant ($p<0.001$). **Conclusion:** We obtained significantly lower values of FEV, FEV1, FEV% expiratory flow rates and MVV in middle aged smoker than their non-smoking counterpart FEV, FEF decrease more extensively than other flow rates.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is defined as a preventable and treatable disease, characterised by persistence of respiratory symptoms and limitation of airflow due to abnormalities of the respiratory tract and/or alveolar duct, generally caused by exposure to toxic gases or mediated by occupational exposure or of some other kind.[1]

COPD is a multifactorial complex disease, considerably influenced by interaction of genetic and environmental risk factors.[2] Tobacco smoking has been considered as the leading cause underlying rising prevalence of COPD across the globe. Almost all forms of smoking products such as cigarettes and bidis used were found to be significantly associated with COPD.[3]

Smoking whether active or passive is a well-known risk factor for general health. There are more than 4000 individual substances isolated from cigarette smoke which include nicotine carbon monoxide, volatile, Aldehydes, Hydrogen Cyanides etc. Smoking is also the major cause of chronic bronchitis and emphysema and interferes with oxygen uptake transport and delivery. Long term smoking causes airway inflammation characterized by neutrophil, macrophage and activated T lymphocyte infiltration and by increased CRP and cytokine concentration.[4] Present study was aimed to study of chronic obstructive pulmonary disease among smokers at our tertiary hospital.

MATERIALS AND METHODS

Present study was prospective, observational study, conducted in department of Physiology, at Sri Guru Ram Das Institute of Medical Sciences & Research, Mehta Road, Post Office Vallah, Amritsar, India.

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Study duration was of 1 year (January 2021 to December 2021). The protocol of the study was approved by the ethical committee of our institute.

Study was explained to patients in local language & written consent was taken for participation & study. This study included 100 male subjects between 19-58 years of age. They were further divided into-25 complete non-smokers (Group I), 25 mild smokers (<5 pack year), 25 moderate smokers (<5-10 pack years) & 25 chronic smokers (>10 pack years) (Group II). 1 pack year = 20 Cigarettes/Day for one year was considered. Persons having asthma or chronic infection of lungs having persistent cough treated recently for any respiratory illness were excluded.

The subjects were drawn from amongst the staff and students of the institute and residents of the city. Pulmonary function test values FVC, FEV1, FEF 25-75%, pefr, were noted. A detailed history of smoking was taken (type of smoke inhaled, numbers of bidis/cigarettes, smoked per day).

The data collected was analyzed and compared with the available literature the ventilator tests were carried out with a computerized Spiro meter. Meds Spiro it was designed to be used with electromechanical pneumonia tech volume differential method. Its overall accuracy is within ± -1% its range for volume is 0 to 10 Liters and for flow is 0-20 liters per sc. and its range body surface area was calculated using Dubois formulae.

Data was collected and compiled using Microsoft Excel, analysed using SPSS 23.0 version. Frequency, percentage, means and standard deviations (SD) was calculated for the continuous variables, while ratios and proportions were calculated for the categorical variables. Difference of proportions between qualitative variables were tested using chi- square test or Fisher exact test as applicable. P value less than 0.5 was considered as statistically significant.

RESULTS

Study population included was 75 mild, moderate and chronic smokers {25 mild smokers (<5 pack year), 25 moderate smokers (<5-10 pack years). 25 chronic smokers (>10 pack years)} & 25 complete non-smokers. There was no statistically significant difference between the mean age height, weight and body surface area of smokers & non-smokers.

Table 1: Anthropometric Measurements between control group (non-smokers) & smokers (Mild, Moderate & chronic).

	Non-Smokers (Group I) (n=25)	Smokers (Group II)		P-Value
Age (years)	34.56± 10.64	Mild smokers (n=25)	31.36 ± 8.31	0.74
		Moderate smokers (n=25)	34.55 ± 9.01	0.35
		Chronic smokers (n=25)	41.36 ± 11.21	0.043
Height (cms)	168.68 ± 9.96	Mild smokers (n=25)	163.84 ± 13.38	0.71
		Moderate smokers (n=25)	163.84 ± 9.44	0.64
		Chronic smokers (n=25)	163.52 ± 11.58	0.62
Weight (kgs)	65.04 ± 11.80	Mild smokers (n=25)	63.08 ± 13.02	0.72
		Moderate smokers (n=25)	59.28 ± 10.29	0.66
		Chronic smokers (n=25)	59.08± 13.12	0.42
Body Surface Area	1.74 ± 0.17	Mild smokers (n=25)	1.68 ± 0.22	0.45
		Moderate smokers (n=25)	1.64 ± 0.16	0.043
		Chronic smokers (n=25)	1.66 ± 0.19	0.52

In present study, value of FEV1 in mild smokers was on the lower side in comparison to the control group and p-value was not significant. The value of FEVI of moderate and chronic smokers and the p-value was statistically significant (p<0.001).

In present study, the values of FEV1/FVC in mild smokers were lower as compared to the control group and p-value is statistically not significant. The value of FEV1/FVC in moderate and chronic smokers were much lower as compared to the control group and p-value was statistically significant (p< 0.05). FEV1/FVC showed significantly greater airway obstruction in smokers as compared to non-smoker. The negative impact of smoking was apparent in most measures but was most progressive in FEV1/FVC ratio.

Value of PEFR in moderate and chronic smokers was lower than the control group and the P-value was statistically significant (p<0.001). The results of the present study were in deceasing trends in the values as we proceed from non-smoker to heavy smokers.

In present study, value of FEF25-75% in mild and moderate smokers was less as compared to non-smokers and the P-value is statistically highly significant (p<0.001). Value of MVV in moderate and chronic smokers was less than that of non-smokers and the P-value is statistically significant (p<0.001).

Table 2: Comparison of respiratory parameters

Parameter	Non-Smokers (Group I)		Smokers (Group II)	P-Value
FVC (Litres)	3.22± 0.64	Mild smokers (n=25)	2.93± 0.70	0.56
		Moderate smokers (n=25)	2.76± 0.51	<0.01
		Chronic smokers (n=25)	2.46± 069	<0.001
FEVI (Litres)	2.98± 0.64	Mild smokers (n=25)	2.78± 0.68	0.53
		Moderate smokers (n=25)	2.27± 0.05	<0.01
		Chronic smokers (n=25)	1.90± 0.48	<0.001

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PEFR (L/sec)	4.18± 1.67	Mild smokers (n=25)	7.08± 1.63	N.S.
		Moderate smokers (n=25)	5.66± 2.23	<0.01
		Chronic smokers (n=25)	4.38± 1.68	<0.001
FEF 25-75%	4.18± 142	Mild smokers (n=25)	3.95 ± 1.45	0.62
		Moderate smokers (n=25)	3.59 ± 1.34	<0.01
		Chronic smokers (n=25)	2.25 ± 1.37	<0.001
FEVI1/FVC	60± 6.31	Mild smokers (n=25)	94.56± 6.91	0.61
		Moderate smokers (n=25)	87.00± 12.21	<0.01
		Chronic smokers (n=25)	78.98± 15.64	<0.001
MVV (L/min)	110.24 ± 46.61	Mild smokers (n=25)	109.68 ± 36.39	0.74
		Moderate smokers (n=25)	77.60± 27.67	<0.01
		Chronic smokers (n=25)	67.60± 29.33	<0.001

DISCUSSION

A chronic progressive disease like COPD reduces the lifespan and quality of life among the affected population. In addition, the lost productivity and high treatment cost can affect the nation in a broader scale. Pulmonary function data in smokers indicate narrowing of smaller airways chiefly bronchioles which lead to slowly progressive COPD. Globally the increase in the burden of COPD has been attributed to cigarette smoking among men and women, longer survival of populations, and high levels of air pollution, particularly in developing countries.[5]

Studies reported deaths due to COPD as a proportion of deaths attributable to smoking: numbers ranged from 12.8% across several industrialized countries46 to 20.9% in the USA.[6] Older age, lower socio-economic status, level of education, poor knowledge about smoking consequences, and rural areas were found to be associated with smoking.[7]

Tobacco smoking, which is the primary risk factor for COPD, begins in adolescence, and it would take 20–25 years of exposure to tobacco smoke to induce characteristic pathophysiologic changes of COPD in human lungs.[8] Tobacco smoke contains in excess of 4000 chemicals in each puff and more than 70 cancer-causing chemicals or carcinogens. In previous studies, it has been seen that subject, who were current and past smokers were at an increased risk of having COPD in comparison with those who were never smokers.[9,10]

Both first-hand/active smoking and passive/shand smoke at office and home in the form of cigarette/bidis, cigars were prevalent and were associated with COPD among study participants. These studies also referred to the age of starting consumption of tobacco, years of smoking, frequency, current smoking status, pack-years and smoking index as risk factors for COPD.[11] Higher age, smoking, low values of FEV1 and FEV/FVC ratio is directly associated with high risk of COPD. The finding of the study shows evidence of smoking history in terms of pack of years as a major risk factor for COPD prevalence.[12]

Rapidly declining lung function in smokers is predictive of COPD. Air flow limitation is progressive and associated with abnormal inflammatory pulmonary function.

Data in smokers indicate narrowing of smaller airways chiefly bronchioles which lead to showily progressive COPD. Severity of COPD depends upon smoking dose, and certain other factors like duration of each puff, ventilation of place of smoking, cigarette or bidi used, force of inhalation, size of butt etc. are likely to affect the actual dose. Since tobacco smoking is the most known and established risk factor for COPD, the male predominance is partly explained on the basis of the male: female differences in smoking habits, particularly in India.[13] Prevalence of COPD was documented to be around 4.1% (5% males to 3.2% females). Smokers had 3 times more risk to develop COPD as compared to non-smokers and Bidi smokers were at higher risk of developing COPD (8.2%) than their Cigarette smoking counterparts (5.9%).[14]

It is inflammatory response of lungs to noxious gases or particles oxidative stress induced by smoking also induces COPD. Correlation between smoking habits and dysnoca morning cough, sputum production was confirmed it was also established that lung function decrease with increasing number of pack years rapidly declining lung function in smokers is predictive of COPD.15 Airflow limitation is progressive and associated with abnormal inflammatory respiratory response of lungs to noxious gases or Particles. COPD leads to affixed narrowing of Airways and destruction of alveoli mainly, in the peripheral parts of lungs.[15,16]

In present study, value of FEV1 in mild smokers was on the lower side in comparison to the control group and p-value was not significant. The value of FEVI of moderate and chronic smokers and the p-value was statistically significant, p<0.001. The above finding is in agreement with findings of Siatkoska H et al,[17] Sherrill D1 et al,[18] & Islam SS et al,[19] These studies also reiterate that chronic smoking related charges in pulmonary function are reflected an accelerated decrease in FEVI. The lung functions also showed a decline with increasing number of pack year. In present study, value of FEF25-75% in mild and moderate smokers was less as compared to non-smokers and the P-value is statistically highly significant with the findings of Walter S et al,[20] and Marq Minette et al,[21] which showed a decreasing trend as we proceed from non-smokers to chronic smoker.

Parasuramalu BG et al,[22] noted that overall prevalence of COPD was 4.36%. The prevalence among males and females were 5.32% and 3.41% respectively. The prevalence was found to be

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increasing with an increase in age. The tobacco smoke and exposure to environmental tobacco smoking (ETS) was significantly associated with higher odds of COPD with adjusted odds ratio 2.97 and 2.67 respectively. Thus, there was a significant association between tobacco smoking and ETS exposure with COPD. The longitudinal studies have shown rapid fall in the FEV1 in a smoking dose-response relationship i.e., larger the frequency and duration of smoking, the more the chances that one develops COPD.[23]

COPD is a major and growing cause of morbidity and mortality with smoking being recognized as its most important causative factor. Several meta-analyses have shown that all pharmacotherapies for smoking cessation are twice as likely more efficacious than placebo with an abstinence rate in the 25-30% range at one year when pharmacological treatment and behavioral support are combined.[24] The chief determinates of flows at low lung volumes are elastics recoil of type lung and resistance of small airways. COPD is widely under diagnosed.

CONCLUSION

We obtained significantly lower values of FEV, FEV1, FEV% expiratory flow rates and MVV in middle aged smoker than their non-smoking counterpart FEV, FEF decrease more extensively than other flow rates. Many smokers are unaware that they have early-stage disease, and simple spirometry is am extremely valuable and simple test that can help people feel better and live longer.

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