Original Article

Smoking related airflow limitation in asymptomatic healthy adults

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Objective: To compare peak expiratory flow rates amongst smokers and nonsmokers.

Methodology: This cross sectional study was carried out at 1 Mountain Medical Battalion and PAF Hospital Rafiqui from January to March 2014. Healthy male adults accompanying patients were selected by non-probability convenience sampling after verbal consent. Exclusion criteria included females, asthmatics, respiratory tract infections or patients unable to comprehend the technique of using peak flow meter. Hand held Mini-Wright peak flow meter was used to measure peak expiratory flow rate (PEFR) in a standing position. Best of three attempts was recorded. Height, weight and chest circumference were measured. History of smoking was obtained and quantified in terms of pack years.

Results: 376 individuals were stratified into three groups: group 1 (18-26 years) having 116 individuals including 29 (25.00%) smokers; group 2 (27-34 years), having 147 individuals including 56 (38.10%) smokers; and, group 3 (35 years or older) with 113 individuals including 44 (38.94%) smokers. Smokers in group 1 had a 4.19% lower PEFR than nonsmokers (P=0.077). Smokers in group 2 and 3 had PEFRs 7.83% and 15.50% lower than those of nonsmokers respectively (P<0.001). Smokers had much lesser PEFR than predicted values as compared to nonsmokers (P<0.05 for all age groups). Smoking load increased with age. There was a significant negative correlation between the number of pack years and PEFR (R= -0.359; p<0.001).

Conclusion: Smokers had lower peak expiratory flow rates as compared to nonsmokers; the difference being directly related to the amount of smoking. (Rawal Med J 2014;39:381-385).

Key words: Smoking, peak expiratory flow rate, peak flow meter, pulmonary function tests.

INTRODUCTION

Smoking a leading preventable cause of death in adults globally and one in every five deaths in American adults is attributable to smoking.[1] In Pakistan, there is a smoker in at least 55% of the households and we lose around 60000 people due to smoking every year.[2] It is a well-known fact that smoking accelerates the age related decline in FEV_1 and quitting smoking can stop this increased loss in FEV_1.[3] Unfortunately, giving up smoking is extremely difficult especially considering the absence of proper smoking cessation services in our country.[4] Chronic obstructive pulmonary disease (COPD) is the most important non-malignant pulmonary effect of smoking and is characterized by reduced FEV_1.[5] Demonstration of a reduced FEV_1 much before COPD becomes clinically evident, can serve as a good motivational factor for smokers to quit.

A cross-sectional study on 11875 healthy asymptomatic Japanese males aged between 35 and 74 years showed that current smokers had a more rapid decline in FEV1 than non-smokers and those who quit smoking had slower decline in FEV1 than those who continued to smoke.[6] Several other international studies have also proved that smoking reduces FEV1.[7] This study was carried out to compare peak expiratory flow rates (PEFR) amongst otherwise healthy male adult smokers and nonsmokers from Pakistan, using this parameter as a surrogate marker of FEV_1.

METHODOLOGY

This cross sectional study was carried out simultaneously at 1 Mountain Medical Battalion (Bagh, Azad Kashmir) and PAF Hospital Rafiqui, Shorkot, Pakistan from January to March 2014 after obtaining approval from Ethics Review Committees of both institutes. Healthy male adults

accompanying patients attending medical outdoor clinics of these hospitals were selected by non-probability convenience sampling after they volunteered and provided verbal consent. Exclusion criteria included females, asthmatics, respiratory tract infections or patients unable to comprehend the technique of using peak flow meter. Hand held Mini-Wright peak flow meter was used to measure PEFR in a standing position. Height, weight and chest circumference at the level of nipples were then measured. At the end, history of smoking was obtained and quantified in terms of pack years using the formula: Number of pack years= (Number of cigarettes smoked daily/ 20) x Years of smoking. Individuals were classified as smokers even if they had quit smoking during last six months.

Statistical analysis was carried out using STATA version 12. Parametric data is shown as mean±SD and compared using independent samples t-test. Nonparametric data is shown as median and range, and compared using Mann-Whitney U test. For all tests of comparison, a P value?0.05 was considered significant. Predicted PEFR for each patient was calculated using the following formula: Predicted PEFR= exp ((0.544*ln(Age))-(0.0151*Age)-(74.7/Height)+5.48). Linear regression analysis was done to determine the relationship between smoking load (pack years) and PEFR.

RESULTS

The number of individuals enrolled during the study period was 376. There were 129 smokers and 247 non-smokers. The mean height of smokers and non-smokers was 172.21± 5.26 and 171.88± 5.61 cm, respectively (P=0.572).

Table 1. Anthropometric measurements of study participants.

		Height (cm)	Weight (kg)	Chest Circumference (cm)
Group 1	Smokers	173.41± 6.82	66.00± 9.33	89.67± 9.86
	Non smokers	171.79± 4.93	66.07± 8.47	88.82± 6.50
	P value	0.245	0.972	0.670
Group 2	Smokers	172.36± 4.87	68.91± 9.73	91.71± 4.93
	Non smokers	172.03± 6.19	70.41± 9.45	92.33± 6.94
	P value	0.725	0.362	0.528
Group 3	Smokers	171.23± 4.44	70.27± 11.34	93.80± 10.56
	Non smokers	171.78± 5.67	73.93± 9.70	95.38± 6.11
	P value	0.562	0.081	0.315

The mean weight of smokers and non-smokers was 68.72±10.27and 69.86±9.67 kg, respectively (P=0.298). The mean chest circumference of smokers and non-smokers was 91.96±8.46 and 91.95±7.04 cm, respectively (P=0.986). In this overall population, the smokers were older than non-smokers (median ages 31 vs. 30 years and range 46 vs. 47 years; p=0.031). To adjust for this age difference, data analysis was performed by stratifying the individuals into three age based groups.

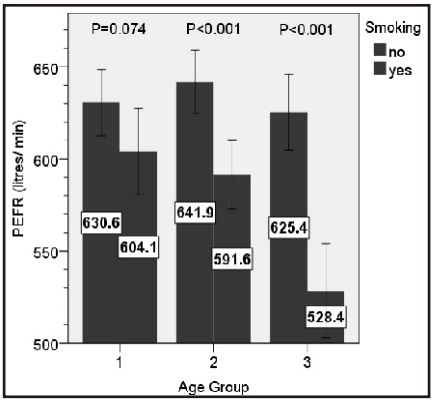


Fig 1.Comparison of peak expiratory flow rates amongst smokers and non-smokers.

(Error bars show 95% confidence intervals for the means)

Age group 1 (18-26 years) consisted of 116 individuals including 29 (25.00%) smokers. In age group 2 (27-34 years), there were 147 individuals including 56 (38.10%) smokers. There were 113 individuals in group 3 (35 years or older), 44 (38.94%) of whom smoked.

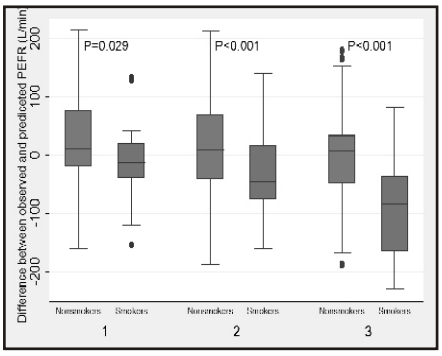
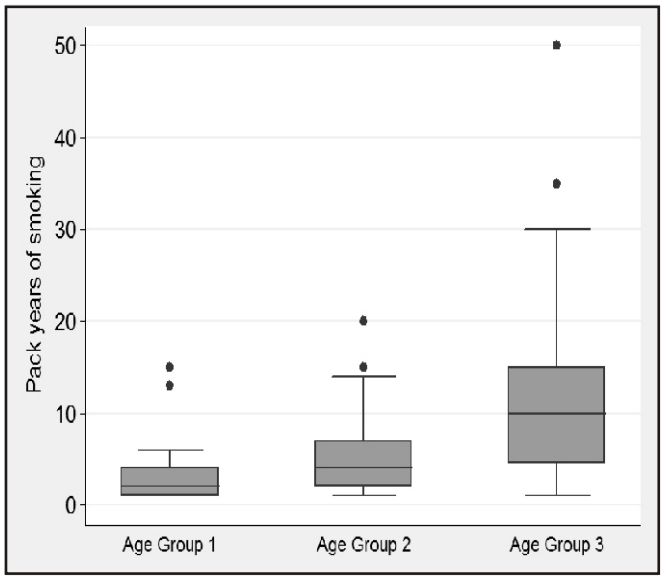


Fig 2. Differences between observed and predicted PEFR in relation to smoking status.

In all these groups, smokers and non-smokers had similar ages, height, weight and chest circumference (Table 1). Smokers had a 4.19% lower PEFR than nonsmokers in group 1 but this difference was statistically insignificant. However, smokers had PEFRs 7.83% and 15.50% lower than

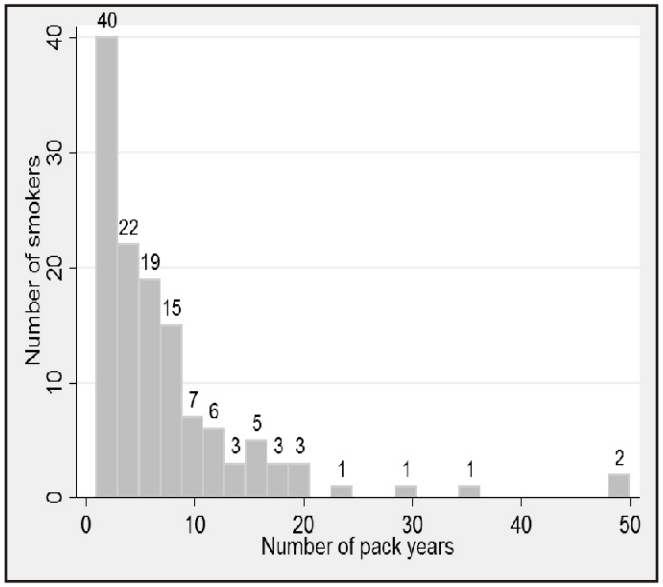
those of nonsmokers in Group 2 and 3 respectively, the differences being statistically significant (Fig 1).

Fig 3. Smoking load in different age groups.



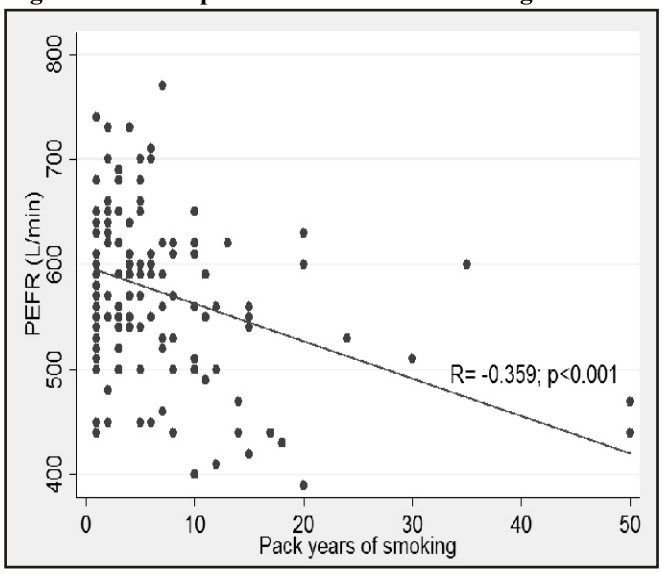
In all the three age groups, PEFR recorded in smokers was much lesser than the predicted PEFR as compared to nonsmokers (Fig 2). Figure 3 shows that the older individuals had smoked more since the median (2,4 and 10 years for groups 1, 2 and 3 respectively) as well as range of pack years of smoking (14, 19 and 49 years for groups 1, 2 and 3 respectively) was greater in older age groups.

Fig 4. Smoking according to pack years.



The frequency of smokers with various pack years of smoking is depicted in the histogram in Fig 4. Linear regression analysis (Fig 5) showed a significant negative correlation between the number of pack years and PEFR (R= -0.359; p<0.001).

Fig 5. Relationship between PEFR and smoking load.



DISCUSSION

This study has demonstrated a reduced PEFR in smokers as compared to nonsmokers. Moreover, the difference between predicted and observed PEFR was also greater in smokers as compared to nonsmokers. These results have substantiated the findings of some other studies done in the recent past. For example, a study done on 80 Indian adults demonstrated a 30% lower PEFR in smokers as compared to nonsmokers.[8] Similarly, Ajay et al found a 29.4% reduction in PEFR amongst smokers in another study done in India.[9] Another cross sectional survey done on 1290 healthy adults in Lahore demonstrated a lower PEFR amongst smokers across different age groups.[10] However, this was not the case in group 1 of this study where the difference between PEFR amongst smokers and nonsmokers was statistically insignificant. This coupled with the finding that the decline in PEFR is directly related to the smoking load in a given patient makes us believe that there might be a threshold (of smoking load) beyond which the reduction in PEFR becomes clinically apparent.

Smoking is very well known to produce airway

inflammation, which extends throughout the respiratory tree and involves the lung parenchyma as well. The exact constituents of cigarette smoke responsible for this are still not clearly identified.[11] Irrespective of this, inflammation in the early stages is associated with an increase in mediator cells such as neutrophils, macrophages and mast cells both within the airway lumen and the airway wall. The resultant edema and proteoglycan deposition produces an increased thickness of submucosal and adventitial tissue thereby compromising airflow.[12] In the later stages, there is progressive fibrosis of airway walls related to smooth muscle hypertrophy and hyperplasia with remodeling. Smoking also increases the number of mucosal goblet cells, resulting in increased secretion of mucus, formation of mucous plugs and obstruction of airways.[13] Cigarette smoke increases bronchial smooth muscle tone, mediated through cholinergic vagal pathways, and this bronchoconstricting effect explains the acute reduction in PEFR immediately after smoking.[14]

PEFR has been used as a surrogate marker of FEV_1 in this study. There are a couple of reasons for this. Firstly, spirometers are not available at the setups where this study has been carried out. Secondly, peak flow meters are portable and convenient to use. Moreover, there is enough literature to suggest a fairly good correlation between PEFR and FEV_1.[15] In a study done on Korean patients with mild asthma, the weighted kappa coefficient for the agreement between FEV1 % and PEFR % was 0.74, indicating excellent agreement between the two measurements.[16]

This study has strong clinical repercussions. Its findings can be linked to the tip of an iceberg in the sense that most of the smokers have much more grave pulmonary consequences than just asymptomatic airflow limitation. It's just a matter of time before the ill effects become clinically apparent, but the individuals would already have lost a lot by then. Smoking is very common in Pakistan and unfortunately, many of the smokers are not aware of the long term ill-effects.[17] Nearly half of the smokers have thus never tried quitting. Others even deny or underestimate the hazards of smoking.[18] This study has identified a very easy means of demonstrating lung damage even in otherwise asymptomatic healthy smokers. The results can be used to educate the masses as well as to convey a very strong message to the general public regarding hazards of smoking.

CONCLUSION

PEFR is lower in smokers as compared to non-smokers. In addition, pack years are negatively correlated with PEFR. This information can thus serve as a motivational factor for smokers to quit.

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Conception and design: Abdul Rehman Arshad
Collection and assembly of data: Abdul Rehman Arshad, Ghulam Abbas Khan Niazi
Analysis and interpretation of the data: Abdul Rehman Arshad, Ammad Akram
Drafting of the article: Ammad Akram Chaudhary, Ghulam Abbas Khan Niazi
Critical revision of the article for important intellectual content: Abdul Rehman Arshad
Statistical expertise: Abdul Rehman Arshad
Final approval and guarantor of the article: Abdul Rehman Arshad
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