

ORIGINAL ARTICLE

PEAK EXPIRATORY FLOW RATE IN ASTHMATIC SMOKERS AND
ASTHMATIC NON-SMOKERS: A COMPARATIVE ANALYSIS

Fatima Qayyum, Sidra Qayyum Malik*, Amber Tasnim Qureshi, Afreen Diju**

Department of Medicine, *Cardiology, Ayub Teaching Hospital, Abbottabad, Pakistan, **Family Medicine, Primary Health Corporation, Qatar

Background: Cigarette smoking and bronchial asthma can be a dangerous mix. The additive decline in pulmonary function can be assessed by a peak flow meter. This study was conducted with the objective of comparing the PEFR of asthmatic patients who smoke with the PEFR of asthmatic patients who do not smoke. **Methods:** This cross-sectional study was conducted in the Department of Medicine, Ayub Teaching Hospital, Abbottabad, Pakistan. A total of 278 patients fulfilling the inclusion criteria from 1st January to 31st August 2018 were included in the study by consecutive non-probability sampling. **Results:** The mean age of the subjects was 49±12.75 years. Female patients were 153 (55%) and males were 125 (45%). Mean PEFR in asthmatic patients who were smokers was 378.22±72.82 L/min and in asthmatic non-smokers it was 456.31±45.93 L/min. The difference in PEFR between two groups was statistically significant ($p=0.0001$). **Conclusion:** The PEFR of asthmatic smokers is significantly low from asthmatic non-smokers. We suggest that cigarette smoking quit program should be a part of disease management.

Keywords: Asthma, Peak Expiratory Flow Rate, smokers

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INTRODUCTION

Bronchial asthma is a chronic lung disease with an increasing global burden.¹ In Pakistan, there are 15 million children and 7.5 million adults suffering from asthma.² From being an atopic illness, the lifetime risk of bronchial asthma is 33.9%.³ Apart from the respiratory distress and exacerbations experienced by patients, this debilitating illness has a range of effects on a person's quality of life including financial burdens,⁴ absenteeism from school and workplace, hospital admissions⁵ and anxiety disorders⁶.

Cigarette smoking is an increasing behavioural problem.⁷ Not only is cigarette smoking observed commonly among healthy adults but it is also seen among patients with bronchial asthma.⁸ The impact of cigarette smoking on health is very vast that includes premature deaths, respiratory diseases, hypertension, myocardial infarctions and various cancers.^{9,10} On respiratory system, it facilitates the penetration of allergen across the respiratory epithelium¹¹, causes oxidative damage to the epithelium and continued exposure in the long run, results in the re-patterning of the epithelium of the small airways¹². All these modulatory changes cause a decline of pulmonary functions as a result of which asthmatic patients suffer from increased hospital admissions, poor response to corticosteroids and non-remission of symptoms.¹³

The pulmonary function can be assessed on the bedside via a peak flow meter by which the peak expiratory flow rate (PEFR) is estimated. The peak flow measurements can be obtained by this device in a manner that is less time consuming, less costly, easy to use and not dependent on trained staff.¹⁴ PEFR

estimation by peak flow meter is comparable to the record of spirometer results.¹⁵

This study was conducted to compare the peak expiratory flow rates in asthmatic patients who are smokers with asthmatic patients who do not smoke. The difference in the PEFR between asthmatic smokers with asthmatic non-smokers will provide a documented evidence of the smoking effects on airways. This will highlight the role of cigarette smoking quit as a part of disease management plan and to have in hospital services for the practical application of such plan.

MATERIAL AND METHODS

This cross-sectional study was conducted in the Department of Medicine, Ayub Teaching Hospital Abbottabad from 1st January to 31st August 2018. Consecutive non-probability sampling was used. The sample size was calculated using the WHO software for sample size calculation. The demographic, clinical and outcome data was recorded on a pre-specified case report form from patients fulfilling the inclusion criteria, i.e., male and female patients aged between 20 to 60 years, patients diagnosed with bronchial asthma for at least 5 years and taking treatment for chronic stable asthma [short acting beta 2 agonist (SABA), long acting beta 2 agonist (LABA), inhaled steroids and/or leukotriene receptor antagonist (LTRA)], and patients who have been smoking cigarettes for at least two year were included in the study. Patients with chest wall deformity, neuromuscular abnormalities, bronchogenic carcinoma, interstitial lung disease, metastatic lung disease, pneumothorax, patients with infective exacerbation of asthma, patients taking NSAIDs, beta blockers, patients who recently started smoking

cigarettes (<2 years) or who have recently quit cigarette smoking (<2 years) were excluded. PEFR among asthmatic smokers and asthmatic non-smokers was determined by using peak flow meter.

Data was analysed on SPSS-18. Quantitative variables like age, duration of asthma, duration of smoking and mean PEFR were described in terms of Mean±SD. Categorical variables like gender and smoking status were described as frequencies and percentages. The PEFR was stratified by age, gender, smoking status and post-stratification independent sample *t*-test and one way ANOVA was applied in which $p < 0.05$ was taken as significant.

RESULTS

A total of 278 patients were included in the study. Mean age of the patients was 49±12.75 years and the age range was 18–60 years (Table-1). Female patients were 153 (55%) and male patients were 125 (45%). The mean duration of asthma was 9±5.36 years. The patients who smoked cigarette were 83 (30%) while non-smokers were 195 (70%). Mean duration of smoking was 14±6.62 years with 21 (25%) patients having history of smoking of up to 10 pack years and 62 (75%) patients having history of 11–20 pack years. The PEFR in young patients was higher as compared to old aged patients decreasing from 486.17±41.20 L/min in second decade to 458.36±45.24 L/min in third decade, 410.91±93.27 L/min in fourth decade and 372.18±68.77 L/min in the fifth decade (Table-2). Mean PEFR in asthmatic patients who were non-smokers was 456.31±45.93 L/min compared to mean PEFR in asthmatic smokers in whom it was 378.22±72.82 L/min ($p=0.0001$).

Table-1: Age group of patients

Age (Years)	Frequency	Percentage
18–30	47	17
31–40	86	31
41–50	83	30
51–60	62	22
Total	278	100

Table-2: Stratification of peak expiratory flow rate

Age (Years)	PEFR (Mean±SD)
18–30	486.17±41.20
31–40	458.36±45.24
41–50	410.91±93.27
51–60	372.18±68.77

One-way ANOVA was applied ($p=0.0047$)

DISCUSSION

Bronchial Asthma was common amongst female patients with a female to male ratio of 1.22:1 in our study. DS Postma in his study similarly found that there is a higher prevalence of asthma in boys than in girls before puberty but a higher prevalence in women than men in adulthood.¹⁶ The change in prevalence could be attributed to the hormonal changes that occur at the time

of puberty making adult asthma more common in females.¹⁷ Zein JG *et al* accounted that the probability of asthma increased with increasing age until about 45 years of age thereafter which it increased at a much slower rate.¹⁸ In their study it was greatest between 18 and 45 years of life. This increased prevalence with age is most probably due to the lung aging that begins after 8 years of life causing an increased stiffness in chest wall and decreased elastic recoil of lung.

Cigarette smoking, which is one of the leading causes of preventable death and a major risk factor for cardiovascular diseases¹⁹, chronic obstructive pulmonary disease and cancers, is prevalent amongst patients of bronchial asthma more than it is among non-asthmatic population. In this study, among 278 patients, 83 (30%) were smokers whilst 195 (70%) were non-smokers. Cerveri *et al* found that smoking was prevalent in subjects with bronchial asthma although it was less frequent when compared with the rest of the population (26 vs 31%).²⁰ The difference is probably due to the respiratory difficulty faced by the asthmatics that hinders them from acquiring smoking habit.

However, combined cigarette smoking and asthma can be dangerous mix and the declined pulmonary function can be determined by PEFR estimation. In this study, the mean PEFR of patients with bronchial asthma who did not smoke was 456.31±45.93 L/min. However, when bronchial asthma was combined with cigarette smoking the mean PEFR declined to 378.22±72.82 L/min ($p=0.001$). Similar were the findings of Medabla *et al*²¹ who conducted a cross sectional study in Andhra Pradesh, India to compare the PEFR of non-smokers and the PEFR of age matched cigarette and cigar smokers. Amongst 123 patients enrolled for their study, the 64 non-smokers had a PEFR of 513.43±87.59 L/min while 49 age matched cigarette smokers had a PEFR of 409.79±90.31 L/min whereas it was 288±42.89 L/min in cigar smokers. Smoking causes bronchial irritation and precipitates acute episodes. It also increases bronchial responsiveness and sensitizes the airways to several allergens.

Sawant *et al*²² in their study conducted in Hyderabad, India compared the PEFR of 50 smokers with 50 BMI and age matched non-smokers. They concluded that the prevalence of abnormal PEFR was 84% among smokers compared to 60% among non-smokers. The risk of abnormal PEFR in smokers was 3.33 times more than in non-smokers and this association was statistically significant ($p<0.05$). Moreover, in that study, the magnitude of PEFR reduction was more in smokers who had longer pack years smoking history. Ukoli *et al*²³ found that the duration of smoking of more than 24 months caused a significant reduction in PEFR. This is most likely due to the inflammatory cascade bringing structural wall

changes of the bronchial tree to decrease the pulmonary function. Longer duration of smoking caused greater decline in PEFr.

CONCLUSION

The PEFr of smoker asthmatics is significantly lower than non-smoker asthmatics. Longer duration of smoking causes greater decline in the PEFr.

LIMITATIONS

Our study was limited to a convenience sample of adult patients of bronchial asthma who had to visit medical OPD/ward and it did not include stable patients of asthma in society. This study did not include younger asthmatics below 18 years. It focused only on cigarette smoke and did not include other forms of tobacco use.

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Address for Correspondence:

Dr. Sidra Qayyum Malik, Trainee Medical Officer, Department of Cardiology, Ayub Teaching Hospital, Abbottabad, Pakistan. Cell: +92-333-5039715

Email: doctor.sidra165@gmail.com

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