International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Original Research Article

Pulmonary Functions in Smokeless Tobacco Users in Haryana

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Received: 05/05/2016 *Revised: 19/05/2016* *Accepted: 24/05/2016*

ABSTRACT

Background: Tobacco use is a public health concern worldwide as well as in India. The predominant use of tobacco is by smoke inhalation of cigarettes, pipes, and cigars or smokeless form. Smokeless tobacco is used in chewed, sniffed or sucked form. Tobacco smoking adversely affects the respiratory system causing chronic bronchitis, emphysema, chronic obstructive pulmonary disease, bronchial carcinoma. However the deleterious effects of smokeless tobacco on respiratory system are yet to be explored.
Aims & objectives: To study the adverse effects of smokeless tobacco on pulmonary function tests and compares them with smoked tobacco.
Methodology: The study was conducted among 30 tobacco nonusers or controls (Group I), 30 tobacco chewers (Group II) and 30 tobacco smokers (Group III). The pulmonary functions were conducted using the RMS Medspirometer. Pulmonary function parameters viz. FEV_1, FVC, FEV_1/FVC, $FEF_{25-75\%}$, PEFR and MVV were recorded and best of three readings were considered. Unpaired *t test* and *ANOVA* test were used for statistical analysis and statistical significance was set at $p<0.05$.
Result: All pulmonary function parameters viz. FEV_1, FVC, FEV_1/FVC, $FEF_{25-75\%}$ and MVV were reduced significantly ($p<.001$) in smokers (obstructive changes). Whereas in chewers all the pulmonary indices except FEV_1/FVC showed significant ($p\leq.001$) decrease (restrictive changes).However, on comparing the three groups, a highly significant ($p<0.000$) reduction was observed in all pulmonary function parameters in smokers except for PEFR.
Conclusion: We observed obstructive pattern in smokers in our study. It is attributed to inhalation of noxious substances. However, the changes in lung functions are restrictive in smokeless tobacco users. This may be due to abnormal gastro esophageal reflux and oxidative damage.

Key words: spirometer, gastric esophageal reflux, antioxidants, restrictive, obstructive.

INTRODUCTION

Tobacco use is a public health concern worldwide as well as in India. [1] WHO reported that tobacco smoking killed 100 million people worldwide in the 20th century and warned that it could kill one billion people around the world in the 21st century also. [2]

Smoking is a practice where tobacco is burnt and smoke is tasted or inhaled in the form of bidis, cigarettes, hookahs, pipes and cigars. [3,4] Of the 1.22 billion smokers worldwide, nearly 80% live in developing countries, where the burden of tobacco-related illness and death is heaviest. [5]

The use of tobacco without burning is referred to as smokeless tobacco (SLT). Smokeless tobacco is taken in several forms e.g. snuff/naswar (finely ground tobacco leaves), chewing tobacco (loose and

sweetened tobacco leaves), zarda/kiwan (paste), paan (betel quid) and khaini/mawa (tobacco with lime). [6] Prevalence of smokeless tobacco use is 26% which is far greater than smoking 14% among adults as reported by Global adult tobacco survey report of India. [7]

Smokeless tobacco indeed represents a health concern of growing magnitude. Due to increased awareness of the adverse effects of smoking, the use of smokeless tobacco has greatly increased. [8] It is presumed to be harmless and a less "social evil" by the users. As a consequence of its addictive qualities, the consumption of smokeless tobacco often becomes a lifelong habit with cumulative and deleterious effects on health. [9,10]

Tobacco smoking has extensive deleterious effects on the respiratory function and has been implicated in the etiology of respiratory diseases like chronic bronchitis, emphysema and bronchogenic carcinoma. [11]

Smokeless tobacco is an important risk factor for the development of various local oral pre-cancerous lesions like lichen planus, lichenoid lesions, leukoplakia, erythroplakia and cancers like that of mouth, throat, cheek, gums and lips. [12] Known systemic effects of SLT include hypertension, angina, congestive heart failure, Raynaud's phenomenon, or peripheral vascular disease. [13] But scanty literature is available on the adverse effects of SLT on lungs, hence, this study was undertaken.

MATERIALS AND METHODS

The present study was conducted in the Department of Physiology at Pt. B.D. Sharma PGIMS, Rohtak. The study population included a total of 90 male volunteers of age group 25-50 years, and was divided into three groups.

- Group I - 30 male volunteers who never used tobacco in any form (control group).
- Group II - 30 male volunteers who were chronic tobacco chewers (non-smokers), who chewed tobacco in the form of loose and sweetened tobacco leaves for a minimum of 10 pouch years in continuation with duration of 7 years or more.
- Group III - 30 male volunteers who were chronic smokers (non-chewers) for a minimum of 10 pack years in continuation with duration of 7 years or more.

The volunteers with any oral lesion, chronic cardiopulmonary, endocrine or metabolic disorder were excluded from the study. Cigarette smoking was quantified in pack years. [13] A standard package contains 20 cigarettes.

This can be translated into pack years as:

Pack years = number of packs per day x years smoked.

Example: 10 cigarettes per day = 1/2 pack for 10 years = 5 pack years (1/2 x 10 = 5).

Similarly, tobacco chewing was quantified in pouch years. [13]

This can be calculated as:

Pouch years = No of pouches per day x years of chewing

Example: 1 pouch per day for 10 years = 10 pouch years (1 x 10 = 10)

Informed consent was taken from every subject to undergo the whole procedure. All the tests were conducted from 10 am to 1 pm to avoid diurnal variation. Overnight abstinence from tobacco use in any form was recommended. Subjects were asked to avoid tea, coffee, carbonated drinks or heavy meals at least two hours before the test procedure. Pulmonary function tests were performed by using computerized RMS Med-spirometer. The whole procedure was explained in detail to each subject in his own language to allay any apprehension or fear. The basic parameters like age, weight, height and BMI of subjects were recorded.

The subject was asked to sit comfortably in a chair. Subject was instructed to breathe in fully by deep inspiration with nostrils closed by using nose clips. The lips were sealed around the sterile mouthpiece of spirometer and air was

forcefully expired out, as fast and as far as possible. Pulmonary function parameter viz. Forced vital capacity (FVC in litres), Forced expiratory volume in first second (FEV_1in litres), FEV_1/FVC (%), Forced expiratory flow rate $_{25\text{-}75\%}$ (MEFR in litres/second), Maximum voluntary ventilation (MVV in litres/minute) and Peak expiratory flow rate (PEFR in litres/minute) were recorded. Best of three technically satisfactory performances as per recommendations of American Thoracic society were recorded and interpreted. [14]

Statistical analysis

All the data obtained was analyzed by unpaired t test and ANOVA test by using Microsoft Excel 2010 software. P value less than 05 was taken as statistically significant and P value less than .001 was taken as highly significant.

RESULTS

There was no significant difference in the anthropometric parameters including age, height, weight and BMI of chewers, smokers and control as shown in Table 1.

The age range of volunteers was 25-50 years with a mean age of 35.8 years in control, 34.5 years in the chewers and 35.6 years in smokers. The statistical significance was determined by student's t test (unpaired). P value less than .05 was accepted as statistically significant.

Table 1: Physical characteristics of control, smokers and chewers

Variables	Control (Mean ± SD)	Chewers (Mean ± SD)	Smokers (Mean ± SD)	p value
Age (years)	35.8±2.6	34.55±7.94	35.6±6.7	>.05
Height (cm)	165±5.27	166.8±7.35	165.86±8.11	>.05
Weight (Kg)	62.9±8.05	64.4±6.12	62.93±7.5	>.05
BMI (Kg/m^2)	22.81±3.35	22.98±2.62	22.55±3.77	>.05

*SD = standard deviation, **p < 0.05 = significant

Table 2: PFTs in control and smokers

Pulmonary function tests (PFTs)	Control (Mean ± SD)	Smokers (Mean ± SD)	p value
FVC(litres)	3.17±.52	1.86±0.66	.0001
FEV_1(litres)	2.87±.41	1.51±.64	.0001
FEV_1/FVC(%)	96.24±11.0	80.8±15.2	.000
$FEF_{25\text{-}75\%}$(litres/second)	3.96±1.06	1.85±1.24	.000
MVV(litres/minute)	125.9±25.7	68.1±32.5	.000
PEFR(litres/minute)	7.05±1.98	3.36±1.97	.9784

*p< 0.05=significant, **p < 0.001= highly significant

Table 3: PFTs in controls and chewers

Pulmonary function tests (PFTs)	Control (Mean ± SD)	Chewers (Mean ± SD)	p value
FVC(litres)	3.17±.52	1.90±.51	.001
FEV_1(litres)	2.87±.41	1.73±.58	.001
FEV_1/FVC(%)	96.24±11.0	90.2±11.4	.84
$FEF_{25\text{-}75\%}$(litres/second)	3.96±1.06	2.31±1.47	.000
MVV(litres/minute)	125.9±25.7	74.4±30.7	.000
PEFR(litres/minute)	7.05±1.98	3.53±1.82	.000

*p<.05=significant, p<.001= highly significant

Table 2 shows PFTs in smokers and control. It is evident from the table that all the pulmonary function indices are showing a highly significant (p<0.001) reduction in smokers except for PEFR on applying student's *t* test (unpaired).

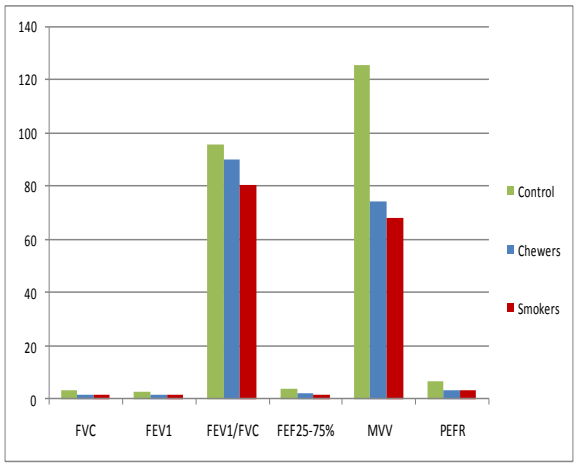
Table 3 shows PFTs among chewers and control. The mean value of all the pulmonary function tests (PFT) parameters is decreased in chewers. The impaired PFT in chewers had shown a highly significant (p<0.001) reduction in$FEF_{25\text{-}75\%}$, MVV and PEFR and a significant (p<0.05) reduction in FVC and FEV_1on applying student's unpaired (*t*) test. The decrease in FEV_1/FVC was not significant.

Table 4: PFTs in control, chewers and smokers

Pulmonary function tests (PFTs)	Control (Mean ± SD)	Chewers (Mean ± SD)	Smokers (Mean ± SD)	p value
FVC(litres)	3.17±.52	1.90±.51	1.86±0.66	.000
FEV_1(litres)	2.87±.41	1.73±.58	1.51±.64	.000
FEV_1/FVC(%)	96.24±11.0	90.2±11.4	80.8±15.2	.000
$FEF_{25-75\%}$(litres/second)	3.96±1.06	2.31±1.47	1.85±1.24	.000
MVV(litres/minute)	125.9±25.7	74.4±30.7	68.1±32.5	.000
PEFR(litres/minute)	7.05±1.98	3.53±1.82	3.36±1.97	.000

*p<.05=significant, **p<.001= highly significant

Comparison of PFTs among three groups is shown in Table 4. The mean of all PFT parameters is decreased in smokers among these three groups. On applying *ANOVA* test, the impaired PFT showed a highly significant (p<0.001) reduction in smokers.



Bar diagram showing PFTs among three groups

DISCUSSION

We observed significant reduction in all PFT parameters except for PEFR in smokers, indicating obstructive impairment. Several studies have shown similar obstructive changes in smokers. [15-19] However, Vyas et al showed somewhat different findings in their study with a significant reduction in FEV_1 but no significant difference in the FVC and FEV_1/FVC in smokers. [20] Similar results were observed by Malo, Angelo, Mahajan, Gupta et al. [21-24] Combustion products from smoked tobacco, apart from nicotine also yield tar, nitrous oxides, carbon monoxide, hydrogen cyanide, phenol and several carcinogenic products like benzopyrene, N-nitrosamine etc. Tar and related products are carcinogenic with other major health hazards like COPD. [25] The damage caused is influenced by the number of cigarettes smoked, filtered or not and method of tobacco preparation. Cessation of smoking reduces the risk of lung cancer mortality compared with that of the continuing smoker. [26]

Acid gastro-esophageal reflux disease (GERD) is a known culprit and risk factor for various respiratory disorders like idiopathic pulmonary fibrosis [27] chronic bronchitis, COPD and pneumonia [28] in smokers. The acid can cause throat irritation, postnasal drip and hoarseness, as well as recurrent cough, chest congestion and lung inflammation leading to asthma, bronchitis or pneumonia. [29] Unlike asthma and cough, in which the esophagobronchial reflex may play an important role, direct aspiration of gastric contents into the lung is thought to be the major pathophysiological mechanism in other respiratory disorders. [27]

We found significant reduction in all PFT parameters except FEV_1/FVC in chewers suggesting restrictive impairment. Pramanik has reported similar results in khaini users. [1] Smokeless tobacco products induce oxidative stress resulting from imbalance between formation of reactive oxygen species and antioxidants, contribute to chronic airway limitation. [30] These free radicles alter the cellular antioxidant defense system. Lam EWN et al have demonstrated the release of free radicle nitric oxide ($^{\bullet}NO$) from extracts and components of smokeless tobacco in human saliva of SLT users. [31] Some other workers have however, reported oxygen free radical (O_2^-) production in cells exposed to smokeless tobacco and nicotine. [31-34] An animal study conducted on the effect of aqueous extract of gutkha, has reported a

decrease in antioxidants like glutathione, superoxide dismutase, catalase and glutathione peroxidase and increase lipid peroxidation in lungs. [35]

An association between GERD and tobacco chewing is also assumed as a culprit in causing the pulmonary manifestations. As the pH of many SLT products is between five to seven, it may also contribute to abnormal acid reflux. [36] There are 2 different mechanisms by which GERD can cause pulmonary manifestations: (i) acid in the distal esophagus stimulating a vagal mediated esophageal tracheobronchial cough reflex and (ii) micro or macro aspiration of esophageal contents into the larynx and tracheobronchial tree. [37] We assume aspiration of tobacco products of like nicotine, nitrosamine compounds, nitrosonornicotine and 4-(methylnitrosamino) -1-(3-pyridyl)-1-butanone into the larynx and tracheobronchial tree by the second mechanism, also contributing to pulmonary pathology. Further studies are required to determine the association between esophageal pH, GERD and pulmonary impairment in tobacco chewers.

All the PFT parameters were significantly reduced in smokers followed by chewers. This implicated that tobacco consumption by smoking has more deleterious effects on lungs than smokeless form, though effects of smokeless form cannot be ignored.

CONCLUSION

Tobacco smoke contains noxious substances like tar, nitrous oxide, carbon monoxide, hydrogen cyanide, phenol and many carcinogenic products like benzopyrene, N-nitrosamine causing diseases like COPD, bronchogenic carcinoma etc. The adverse effects of SLT on lungs, however, need attention. Smokeless tobacco produces oxidative stress by increasing the formation of free radicles and decreasing the level of antioxidants and free radicle scavengers, which may result in chronic airway limitation. Antioxidant rich foods such as green leafy vegetables and fruits may help to reduce the oxidative stress caused by tobacco consumption. The treatment of acid reflux in SLT users may also help in reducing pulmonary manifestations. SLT should not be considered a safe alternative to smoking. Adequate measures are required to be taken to curtail the use of tobacco even in the smokeless form.

ACKNOWLEDGEMENT

We would like to express our sincere thanks to all the staff of Department of Physiology and Department of Chest and Tuberculosis, Pt BD Sharma, PGIMS, Rohtak and subjects of the study who have helped us in making this work a reality.

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How to cite this article: Gupta A, Goyal K, Gupta R. Pulmonary functions in smokeless tobacco users in Haryana. Int J Health Sci Res. 2016; 6(6):106-112.

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