Pulmonary function, white blood corpuscles & haemoglobin levels in asymptomatic light smokers and non-smokers

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Abstract

Context: Smoking, a risk factor for several diseases has also been reported as the largest preventable cause of morbidity and mortality globally. Tobacco smoking is associated with a decrease in lung function in smokers when compared to non-smokers. **Aims:** To study and compare pulmonary function, WBC count and Hb levels in asymptomatic light smokers and non-smokers. **Settings and Design:** Case control study. **Methods and Material:** Patient relatives or subjects who had come for health check with a known history of smoking and apparently asymptomatic were recruited as our study subjects (n = 49). Healthy volunteers were also recruited from patient relatives. Subjects without any known history of smoking were selected as controls (n = 50). **Statistical analysis used:** Data was analysed using SPSS V-19. P value ≤ 0.05 was considered significant. **Results:** A statistically significant difference was found in total leucocyte count in smokers (P = 0.004) when compared to non-smokers. There was also a statistically significant increase in absolute neutrophil (P = 0.021), monocyte (P = 0.004) and eosinophil (P = 0.075) counts in smokers. FEV1/FVC (P = 0.005) and FEF 75 (P = 0.027) was significantly higher in non-smokers when compared to smokers. No significant difference was observed in haemoglobin concentration and differential leucocyte count, pulse rate, systolic pressure, diastolic pressure and rate pressure product among the two groups. **Conclusions:** The present study gives ample evidence for the damage to the respiratory system along with activation of immune system as evident from a significant lowering of small airway patency and increased white cell counts.

Key-words: Pulmonary function, White blood corpuscles, Smokers, Non-smokers,

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Introduction

Smoking, a risk factor for several diseases has also been reported as the largest preventable cause of mortality globally [1-3]. In a study on Industrial population in India, 40.2% of men and 14.9% of women has been reported to have the habit of using tobacco.

The authors have also reported smoking to be the major form of tobacco consumption [4]. Among tobacco users in Mumbai, cigarette smokers have been reported to have a relative risk of mortality of 1.39 [5]. Tobacco smoking has been reported to have caused 700,000 deaths in India in the year 2000 alone [6].

Manuscript received: 11th December 2016 Reviewed: 20th December 2016 Author Corrected: 26th December 2016 Accepted for Publication: 3rd January 2017 A decrease in pulmonary function is smokers have been well established. Studies have reported forced expiratory volume in 1 sec (FEV1) to be lowest in cigarette smokers [7]. A lower and a rapid decline in lung function in smokers when compared to age and height matched non-smokers has also been reported [8]. A longitudinal study on Norwegian men for seven years has reported a lower FEV1 in smokers as compared to non-smokers [9].

Studies from India have also shown the ill effects of smoking on lung function parameters. A study from rural Maharashtra has reported a decrease in all pulmonary function parameters in smokers [10]. Studies have also reported a decrease in FEV1, force vital capacity (FVC), FEV1/FVC in smokers [11, 12]. Haematological parameters have also been reported to be altered in smokers. An increase in total leucocyte count [13], an increase in neutrophils and eosinophils in smokers has been reported previously [14]. A dose response relation between smoking and white cell counts has also been reported [15]. High white blood cell (WBC) count has been reported to be a strong and independent predictor of coronary risk in patients of both sexes [16].

Most of these reported studies have been carried out on moderate and heavy smokers. The purpose of the planned study was to evaluate lung function, Hb and easily attainable markers of inflammation such as WBC count in light smokers and their by assess the cardiovascular risk associated with these apparently healthy individuals. Not many studies have reported cardiovascular risk associated with light smoking.

Subjects and Methods

The present study was carried out in the department of Physiology and Medicine outpatient department. Patient relatives with a known history of smoking and apparently asymptomatic were recruited as our study subjects (n = 49). Healthy volunteers were also recruited from patient relatives. Subjects without a known history of smoking were selected (n = 50). Subjects with a history of recent drug therapy, any disease affecting blood counts and pulmonary function was excluded from the study. The classification of study subjects as light smokers was based on pack years [17]. All the subjects included in the present study are males.

Results

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Written informed consent was collected from all the study participants. Institute Ethical committee clearance was also obtained. Necessary care was taken to comply with the World Medical Association Declaration of Helsinki regarding ethical conduct of research involving human subjects. A day prior to actual recording 5ml of venous blood was collected for the estimation of Hb concentration, total leucocyte count (TLC) and differential leucocyte count (DLC). Absolute values for each type of cell was then calculated (Cotter S - Hematology). Blood parameters were analysed using Sysmex XN-1000TM Hematology Analyzer (Sysmex America). On the day of recording, blood pressure was recorded using a mercury sphygmomanometer as per the recommendations of the JNC 7 guidelines [18].

Height and weight were measured and BMI was calculated as weight in kg divided by the square of the height in units of meter squared. Lung function parameters were assessed using RMS Helios 401(RMS, India) Spirometer connected to a Windows based computer. The parameters recorded were FEV1, FVC, FEV1/FVC, FEF25-75, FEF 25%, FEF 50% and FEF 75%. The recordings were based on the American thoracic society and European respiratory society task force report [19].

Statistical Analysis- Data are presented as the means \pm SD unless mentioned otherwise. IBM SPSS statistics (Version 22.0. Armonk, NY: IBM Corp) software for Windows was used in data analysis. Unpaired t test or Mann Whitney U test was used as appropriately for the comparison of the two groups. A 2-tailed P value ≤ 0.05 was considered statistically significant.

No statistically significant difference was observed in age, height, weight and body mass index between smokers and non-smokers' [Table 1]. No significant difference was also observed in haemoglobin (Hb) concentration and differential leucocyte count (DLC) among the two groups as shown in table 2. There was a statistically significant difference in TLC in smokers (P = 0.004) as compared to non-smokers although the increase was within the normal limits.

| | Smokers (50) | Non-smokers (50) | P value |
|--------------|--------------------|---------------------|---------|
| Age (yrs) | 29.98 ± 7.779 | 32.28 ± 6.809 | 0.119 |
| Height (mts) | 1.685 ± 0.074 | 1.683 ± 0.069 | 0.878 |
| Weight (Kg) | 66.940 ± 8.524 | 66.920 ± 11.900 | 0.992 |
| BMI | 23.494 ± 1.898 | 23.545 ± 3.414 | 0.928 |
| Height (mts) | 1.685 ± 0.074 | 1.683 ± 0.069 | 0.878 |

Table-1: Age & anthropometric parameters between smokers and non-smokers. Data expressed as mean ± SD.

BMI: Body Mass Index

Table-2: Haemoglobin concentration, Total and differential leucocytes count in smokers and non-smokers. Date expressed as mean± SD.

| | Smokers (50) | Non-smokers (50) | P value |
|------------------------------------|------------------------|--------------------|---------|
| Haemoglobin (g %) | 14.250 ± 1.375 | 14.380 ± 1.325 | 0.632 |
| WBC count (cells/mm ³) | 7744.00 ± 1813.122 | 6754.00 ± 1521.439 | 0.004 |

WBC: white blood corpuscles.

Table 3 shows the absolute leucocyte count in smokers and non-smokers. There was a statistically significant increase in neutrophil (P = 0.021), monocyte (P = 0.004) and eosinophil (P = 0.075) count in smokers as compared to non-smokers. No statistically significant different was observed in pulse rate, systolic pressure, diastolic pressure and rate pressure product [Table 4].

| Table-3: Absolute leucocy | vte count in smokers and | non-smokers. Data ex | pressed as mean ± SE. |
|---------------------------|--------------------------|----------------------|-----------------------|
| | , | | |

| | Smokers (50) | 95% CI | Non-smokers (50) | 95% CI | P value |
|-------------|----------------|-------------|------------------|-------------|---------|
| Neutrophils | 4093 ± 175 | 3756 - 4452 | 3570 ± 138 | 3300 - 3850 | 0.021 |
| Lymphocytes | 2485 ± 110 | 2270 - 2701 | 2341 ± 97 | 2165 - 2532 | 0.38 |
| Monocytes | 627 ± 26 | 578 - 672 | 524 ± 24 | 475 - 569 | 0.004 |
| Eosinophils | 400 ± 43 | 312 - 486 | 304 ± 31 | 242 - 377 | 0.075 |
| Basophils | 43 ± 7 | 39 - 57 | 40 ± 6 | 28 - 53 | 0.767 |

95% CI: 95% confidence interval

| | Smokers (50) | Non-smokers (50) | P value |
|-----------------------|--------------------|--------------------|---------|
| Pulse rate (BPM) | 72.56 ± 6.020 | 73.48 ± 8.664 | 0.539 |
| Systolic BP (mmHg) | 120.20 ± 7.910 | 119.40 ± 9.598 | 0.650 |
| Diastolic BP (mmHg) | 77.68 ± 6.967 | 77.84 ± 8.683 | 0.919 |
| Rate Pressure Product | 87.13 ± 8.200 | 88.05 ± 15.142 | 0.705 |

Bpm: beats per minute

Table-5: Spirometric parameters in smokers and non-smokers. Data expressed as mean± SD.

| | Smokers (50) | Non-smokers (50) | P value |
|----------------|--------------------|--------------------|---------|
| FEV1 (lts) | 2.86 ± 0.776 | 2.84 ± 0.618 | 0.844 |
| FVC (lts) | 3.30 ± 0.715 | 3.14 ± 0.585 | 0.309 |
| FEV1/FVC (%) | 86.80 ± 16.051 | 91.04 ± 15.827 | 0.005 |
| FEF25-75 (lts) | 3.70 ± 1.284 | 4.05 ± 1.180 | 0.203 |
| FEF 25 (lts) | 5.83 ± 2.607 | 5.91 ± 2.147 | 0.684 |
| FEF50 (lts) | 4.25 ± 1.612 | 4.50 ± 1.411 | 0.393 |
| FEF 75 (lts) | 2.12 ± 0.792 | 2.53 ± 0.899 | 0.027 |

FEV1: Forced expiratory volume at one second, FVC: Forced vital capacity, FEF: Forced expiratory flow.

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| | Smokers (50) | Non-smokers (50) | P value |
|--------------------------------|---------------------|---------------------|---------|
| Percentage predicted FEV1 | 86.22 ± 18.142 | 95.20 ± 17.985 | 0.001 |
| Percentage predicted FVC | 84.28 ± 10.435 | 88.60 ± 13.548 | 0.076 |
| Percentage predicted FEV1/FVC | 103.84 ± 18.668 | 108.46 ± 18.590 | 0.008 |
| Percentage predicted FEF 25-75 | 82.00 ± 24.669 | 94.12 ± 24.249 | 0.029 |
| Percentage predicted FEF 25 | 73.16 ± 32.985 | 74.42 ± 27.740 | 0.754 |
| Percentage predicted FEF 50 | 76.84 ± 31.236 | 78.56 ± 25.630 | 0.672 |
| Percentage predicted FEF 75 | 75.68 ± 24.672 | 85.80 ± 25.261 | 0.072 |

Table-6: Percentage predicted spirometric parameters in smokers and non-smokers. Data expressed as mean± SD

FEV1: Force expiratory volume in 1 second; FVC: Forced vital capacity; FEF 25-75: forced mid expiratory flow 25-75%.

Table 5 shows the lung function parameters in two groups. FEV1/FVC (P = 0.005) and FEF 75 (P = 0.027) was significantly higher in non-smokers when compared to smokers. None of the other pulmonary function parameters in absolute values were significantly different between the two groups. Percentage predicted values of FEV1 (P = 0.001), FEV1/FVC (P = 0.008) and FEF 25-75 (P = 0.029) were significantly higher in non-smokers as compared to smokers (Table 6).

Discussion

The present study was planned to evaluate the effect of smoking on lung function test, haemoglobin and white blood count in asymptomatic light smokers. Although no significant difference in Hb and DLC was observed, there was a statistically significant difference in TLC, absolute neutrophil, monocyte and eosinophil count, with the count being higher in smokers.

In contrast to our findings are studies which have reported an increase in Hb levels in smokers as compared to non-smokers [20, 12]. The authors have attributed this to hypoxic stimulation of Hb production in smokers owing to carbon monoxide present in cigarette smoke. Various other studies have reported an increase in leucocyte count, which are in line with the findings of the present study [22, 23]. TLC and leucocyte subsets as estimated by DLC have also been reported to be higher in smokers [24].

Most of these studies have employed DLC and the study subjects were mild to moderate smokers. An increase in WBC count in light smokers has also been reported previously [25]. Moreover, in the present study we have reported significance levels in WBC subsets based on absolute counts.

The presence of elevated WBC subsets gives a picture of allergic reaction.

Elevated markers of allergy have been previously reported in smokers. Along with an increase in TLC, the study also reported an increase in eosinophils and serum IgE levels [26]. This increase in WBC count has been considered to be an acute effect of smoking [14].

The probable reason for only a mild increase in WBC count in our study group could be due to the fact that our smokers were mostly light smokers. The same fact has also been highlighted by the reported dose response relationship between smoking and WBC counts [14, 24]. The increase in WBC count in smokers has also been attributed to activation of the inflammatory system [22], endothelial damage [21, 27] and also via increase in catecholamine release by nicotine present in cigarettes [14].

WBC counts have emerged as a marker of inflammation that is widely available in clinical practice [28]. Inflammation plays an important role in the development of cardiovascular disease. An elevated WBC counts in our study subset probably highlights the risk associated with these group of light smokers who were otherwise healthy.

No significant change in BP, pulse rate and RPP was observed in smokers when compared to non-smokers. Contrasting reports have been documented from various studies on the effect of smoking on BP and heart rate (HR). Heavy smoking has been found to be associated with persistent elevation in BP and increase in blood pressure variability [29]. Increase in BP and HR has also been reported in habitual smokers.

The authors have attributed these changes to the arterial wall stiffening effect brought about by nicotine [30].

FEV1/FVC ratio is the corner stone for the diagnosis of obstructive airway disease [31]. In the present study we did not find smokers falling into this disease category in spite of the fact that FEV1/FVC was significantly lower in smokers compared to non-smokers.

The percentage of predicted values of FEV1 and FEV1/FVC were also significantly lower in smokers. FEF25-75% is the average expired flow over the middle half of the FVC manoeuvre and is regarded as a more sensitive measure of small airways narrowing.

The predicted value for FEF 25-75% were also was significantly lower in smokers. The lower value for these parameters in our study of light smokers is probably an indication of increasing smaller airway obstruction in these people as compared to non-smokers.

Cigarette smoking has been well documented to have deleterious effect on the respiratory system. Injury to the lung is attributed to the harmful chemicals present in cigarette smoke [32].

A progressive decrease in expiratory flow rates with increasing cigarette usage has also been reported previously [33]. A lower FEV1, FVC, FEV1/FVC% and FEV1% predicted has also been reported in smokers [34, 35]. In contrast to our finding are the observations of no significant difference of FEV1/FVC in smokers and non-smokers [12].

Damage to the respiratory system even in the case of light smokers is clearly apparent from the present study. Mild changes if not properly taken care of will surely result in severe cases of obstructive disease.

In view of the rising incidents of death owing to tobacco smoking our results finds significance.

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