RESEARCH ARTICLE

EFFECT OF SMOKING ON SELECTED BLOOD PARAMETERS

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ABSTRACT
Background: Smoking is not only a physical recidivism, but also a psychological inveteracy. Chronic effects of cigarette smoking on haematological parameters, with a good control side by side, were studied by very few. Evectics (including deaddiction) should be based on harm profile.
Aims & Objectives: To find out age-wise Hb, BP, leucocyte (total/ differential) count, monocyte count, and eosinophil count in smokers.
Materials and Methods: Study was carried out on a group of 108 otherwise healthy subjects from medical college campus – including 36 each in non-smoker, light-smoker and heavy smoker group – further subdivided into 9 each in 4 age ranges of 21-30 years, 31-40 years, 41-50 years and 51-60 years. Data regarding age, sex, height, weight and various blood parameters were noted as per standard methods.
Results: Chronic effects of smoking on cardiovascular system were found. SBP of both light and heavy smokers were significantly less than control group. DBP of light smokers and non-smokers did not differed very much. TLC and eosinophil count was higher while monocyte count was lower in smokers (insignificantly low in light smokers).
Conclusion: The TLC and eosinophil count rise, while BP and monocyte counts fall in smokers. Smoking may lead to isolated systolic hypertension. In Evectics, the improvement/ outcome can be measured by assessing the rate/ extent of normalization of these parameters.
Key Words: Blood Pressure; Age; Sex; Weight; Height; Smoking

Introduction

Recidivism is the tendency of a person to return back to a known bad behaviour and inveteracy is that behavioural outcome, i.e. recurrence of a known vice. There are more than 1 billion smokers in the world with an increased/ decreased/ again increased smoking habit. Worldwide, more than 3 million people currently die each year from smoking, and more than one third have cardiovascular events that often determine permanent disability of affected subjects.[1]

In USA, Current cigarette smoking prevalence among all adults aged ≥18 years has decreased 42.4% since 1965, but declines in current smoking prevalence have slowed during the past 5 years (declining from 20.9% in 2005 to 19.3% in 2010).[2] As a reason, there can be the concept of gender empowerment and smoking being advertised as a sign of liberation.

In a study by World Health Organization (WHO), the gender empowerment measure (GEM) has been shown to be positively and significantly correlated to gender smoking ratio (GSR). In addition, the GEM was the strongest predictor of the GSR (β, adjusted: 0.47; $P < 0.0001$), after controlling for gross national income (GNI) per capita and for Gini coefficient.[3]

But in India, tobacco consumption continues to rise, even though the evidences mount regarding its hazards. Young people are smoking earlier and more heavily. A recent study of mortality associated with smoking in India (2008), estimates that at least 930,000 adult deaths in India could be attributed to smoking, and that this would rise to over one million annually from 2010.[4]

Nicotine may mask the effects of carbon monoxide on arterial wall for a long time. Adverse effects, usually, will appear when they will be of structurally severe degree so that to induce stable hypertension.[1]

It is also well established that current smokers are characterized by increased white blood cell (WBC) counts and increased tumor necrosis factor-a (TNF-a). Light smoking is associated with an increase in WBC counts, while heavy smoking is responsible for TNF activation.[5]

Unfavourable blood rheology is closely associated with cigarette smoking and may reflect increased cardiovascular risk in smokers. The study results also suggest that such risk can be reduced after only 3 months

of smoking cessation.[6]

Smoking influences the immune system (i.e., neutrophils, macrophages, lymphocytes, and dendritic cells) in many ways, including through continual exposure to oxidative stressors. As a consequence, a low-grade systemic inflammatory response is evident in smokers as confirmed by numerous population-based studies.[7]

Elevated levels of CRP, interleukin 6, and tumor necrosis factors have been reported. Moreover, several studies have shown that rheological alteration and circulatory procoagulant and hypofibrinolytic activity occurred in current smokers.[7,8]

As an inference, it has been thought that smoking may, in part, explain the changed level of blood pressure, total and differential leucocyte count (TLC), eosinophil count and monocyte count.

Materials and Methods

Sampling

After due permission from ethical committee and written informed consent, the present study was carried out on a group of apparently healthy 108 subjects from medical college – including 36 each in non-smoker, light-smoker and heavy smoker group – further subdivided into 9 each in 4 age ranges of 21-30 years, 31-40 years, 41-50 years and 51-60 years. The mean height in cm. was 167.5 ±4.5 and mean weight in kg was 56.3 ± 8.68. There was an unequivocal history of being chronic smokers in 72 subjects, remaining was a non-smokers' group of 36 subjects having neither history of cigarette smoking nor tobacco chewing.

Classification of Subjects

a. *Non-Smokers:* 36 subjects, having no history of cigarette or tobacco chewing, were included in this group.
b. *Smokers:* Subjects with history of smoking at entry. Only cigarette smokers were included and were further divided into 2 sub-groups according to the amount of smoking. *(i) Light smokers (LS):* those who smoked less 10 cigarettes/ day. (ii) *Heavy smokers (HS):* those who smoked more than 10 cigarettes/ day.[11]

Procedure

Each individual was asked to take rest for ½ an hour prior to the beginning of tests, to avoid psychological and exertion effect on base line parameters like blood pressure etc. Blood pressure – mercury type of sphygmomanometer was used to measure blood pressure. CHEP, 2014 (Canadian Hypertension Education Program (CHEP) 2014 Recommendations, table.1) was used as the reference.[9] Pulse rate was recorded and blood was taken by finger prick for estimation of haemoglobin, total leucocyte count (TLC), differential leucocyte counts (DLC), eosinophil count (EOS) and monocyte count (MON). Haemoglobin, total leucocyte count (TLC), differential leucocyte count (DLC), eosinophil count and monocyte count were done on Hemoscreen device from Pixcell Medical Technologies.

Results

Mean and standard deviation of age, height and weight of light and heavy smokers and non-smokers were as shown in table 1. In different subgroups, the subjects were kept the same to avoid effect size variation. As shown in table 2, age and height were not significantly different among the groups. The weight was significantly less in light smokers and heavy smokers were also heavy-weight. As shown in table 3, with intensity of smoking, the suppression of systolic blood pressure was quite significant ($p < 0.01$). Similar, but less significantly ($p<0.05$), suppression in diastolic blood pressure was seen only in heavy smokers. This might mean that smoking can lead to isolated systolic hypertension. Compared to non-smokers, difference of diastolic blood pressure was insignificant among light smokers – though difference of diastolic blood pressure was significant among light/none smokers (0.05), if compared to heavy smokers.

As shown in table 4, mean total leucocyte counts (TLC) was highest in heavy smokers and lowest in non-smokers – all intergroup differences being statistically significant ($p<0.05$). Eosinophil increased with intensity of smoking and the increase was highly significant (0.01). As shown in table 4, absolute monocyte (MON) counts showed highly significant ($p<0.01$) negative relation in heavy-smokers, when compared to non-smokers, though monocyte count was not significantly decreased in light smokers.

Table-1: Grouping of subjects as per age and smoking habits

Group	20-30 Years	31-40 Years	41-50 Years	51-60 Years	Total
Non smokers	9	9	9	9	36
Light smokers	9	9	9	9	36
Heavy smokers	9	9	9	9	36
Total	27	27	27	27	108

Table-2: Mean and SD of various demographic parameters (age, height, weight), as per their smoking habits

Group	Age	Height	Weight
Non smokers	39.99 ± 2.52	166.99 ± 4.36	57.44 ± 9.46
Light smokers	40.10 ± 2.58	167.91 ± 4.65	52.08 ± 7.45
Heavy smokers	39.99 ± 2.52	166.99 ± 4.36	59.49 ± 9.15

Table-3: Blood pressure in smokers and non-smokers

Blood Pressure (mmHg)	Non smokers (Mean ± SD)	Light smokers (Mean ± SD)	Heavy smokers (Mean ± SD)
Systolic	121.50 ± 1.30	118.55 ± 2.52	116.55 ± 2.89
Diastolic	78.47 ± 2.24	78.38 ± 2.36	76.38 ± 2.34

Table-4: Haematology parameters with mean and SD in different smoking group

Group	Total Leucocyte Count	Absolute EOS Count	Absolute MON Count
Non smokers	6305.16 ± 60.51	150.54 ± 35.45	218.78 ± 45.86
Light smokers	6944.44 ± 48.27	179.51 ± 44.21	212.20 ± 22.07
Heavy smokers	8195 ± 80.13	228.19 ± 102.59	155.55 ± 56.69

Discussion

Most of the studies on smoking are concerned with acute effects of smoking. Chronic effects of cigarette smoking on haematological parameters with completely balanced grouping are studied by very few. As the difference of weight among non-smokers and light-smokers is negative (light smokers are also light weight), the statistical outcome could be internally compensatory.

On the other hand, heavy smokers were also heavy-weights – the health damage might have been synergistic. But as none of the participant was abnormally obese or cachectic, chances of getting the parameters significantly altered are minimal.

In epidemiologic studies of blood pressure, smokers have been found to have lower BP than non- smokers. Our result are generally consistent with above, as our data showed that mean SBP of heavy smokers and light smokers were lower than that of non-smokers and the difference was statistically significant. Accordingly, mean SBP of light smokers were more than heavy smokers.

Contrary to some findings reporting high blood pressure due to smoking, this finding is explained by "masking phenomenon" – in smokers, initially blood pressure is lowered under stress till cardio-vascular modalities are damaged enough to bounce back and show hypertension despite continued smoking. Thus lowering of BP in heavy smokers is expected to be more aggressive and light smokers, having less lowering, are expected to remain higher on that count.[1]

Altered immune cells (eosinophils, monocytes) also go with related researches.[5,10] Damage was worst for eosinophils and least for leucocytes as a whole (latter due to partial internal compensation e.g. increase in eosinophils and decrease in monocytes).

Conclusion

The result of this study showed that there is negative association of smoking with blood pressure and monocytes while leucocyte count and eosinophil counts was increased in smokers. Smoking not only affects the humeral immune system but also the cellular immune mechanism as the number of EOS was increased and MON was decreased. In Evectics, the branch of medical humanity dealing in rehabilitation of addicts, the improvement/ outcome can be measured by assessing the rate/ extent of normalization of these parameters.

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