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Original Research Article

Impact of Cigarette Smoking on Perceived Stress and Serum Cortisol among Medical Students

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ABSTRACT

Cigarette smoking is one of the prime causes of increased mortality and morbidity in developed countries and the prevalence is increasing in developing world. Poisoning the body with carbon monoxide causes physical illness like emphysema, chronic bronchitis, arterial damage. The nicotine levels in plasma correspond to blood cortisol levels. The purpose of this study was to compare the serum cortisol and the degree of perceived stress among smokers and non smokers. This study included 30 students (15 smokers, 15 non smokers) from 1st year and similar group was chosen from 3rd year medical students. Body mass index, waist circumference and resting blood pressure were assessed. Perceived stress was assessed using Presumptive stressful life event scale. Serum cortisol level was estimated by Electrochemiluminescence immunoassay on a HITACHI 917 analyzer. The data was statistically analyzed using students t test and chi-square test. P value <0.05 was considered as statistical significance. The 1st year non smokers showed significantly higher stress score than smokers (p value=0.009). In the 3rd year group we found smokers had significantly higher serum cortisol levels (p value=0.007). This study concludes that smoking was not taken up to reduce stress level. Also it shows that smoking causes metabolic changes leading to hypercortisolism.

Keywords: Cigarette smoking, Serum cortisol, Electrochemiluminescence immunoassay, Hypothalamo-Pitutary-Adrenal Axis.

INTRODUCTION

The health consequences of cigarette smoking are an important cause of increased mortality and morbidity in developed countries and the prevalence is increasing in the developing world as well. Atherosclerotic cardiovascular disease due to smoking is a major cause of death. The risk of cancer is greater in smokers than nonsmokers, particularly for lung cancer. [1] Smoking has multiple effects on pituitary, thyroid, adrenal, testicular and ovarian function, calcium metabolism and the action of insulin associated with important clinical

implications. [2] These effects are mainly mediated by the pharmacological action of nicotine, toxins such as thiocynate and carcinogens. Smoking contributes to the development insulin resistance and hence type 2 diabetes mellitus. [3] The effect of smoking on the various metabolic and biological process in the body including secretion of hormones are mediated chiefly through behavioral and pharmacological actions of nicotine but also occur as a result of increased physical effects of stress on body caused by smoking. Smoking increases heart rate and blood pressure as a result of constriction of blood vessels. It increases the concentration of fatty acids in blood and also the ability of blood platelets to adhere to each other and to the walls of blood vessels. Carbon monoxide in smoke reduces the oxygen carrying capacity of the blood. Nicotine causes stimulation and sedation of central nervous system depending upon the dose. [4] The nicotine levels in the plasma correspond to the blood cortisol levels. [5] It is well known that psychiatric stress also induces rise in cortisol levels. [6] In view of above mentioned facts the effect of smoking on alteration in the serum cortisol in smokers, and their correlation with psychological stress factors was of interest.

MATERIALS & METHODS

This study was carried out among students studying in 1st year and 3 rd year in Kasturba Medical College, Mangalore. 15 students from 1st year with a history of smoking for 6-9 months and 15 students from 3rd year with smoking duration of at least 3-4 yrs where selected. 15 students in each group who were age matched and non smokers served as controls. Students on any sort of medication, or with acute inflammatory response within previous 2 weeks were excluded from the study. Upon obtaining their consent for participation, their height, weight and blood pressure was recorded. Body mass index was calculated. Stress level was assessed using presumptive stressful life event scale, modified by Gurmeet Singh et al [7] to suit the student population. 5 ml of venous blood was collected from 1st year students, early morning between 7- 7:30 am. Samples were immediately analyzed in the biochemistry laboratory using HITACHI 917 analyzer for serum cortisol. Serum cortisol was assessed using Electrochemiluminescence immunoassay. The results obtained were statistically analyzed using students T test to compare the parametric data among the groups. Chi square test was used to for association of stressors in the stress scale. P value less than 0.05 was considered significant.

RESULTS

Table 1- Comparison between smoker and non smokers of 1st year students.

PARAMETER	NON SMOKER(15)	SMOKER(15)	P VALUE
AGE (YEARS)	19.00±0.92	19.93±0.96	0.110
BMI(KG/M^2)	20.51±2.59	21±2.31	0.592
WAIST CIRCUMFERENCE	79.60±6.76	78.26±3.26	0.594
SYSTOLIC BP	119.06±8.64	116±8.45	0.334
DIASTOLIC BP	76±8.28	78.93±10.41	0.400
SERUM CORTISOL	13.18±3.81	15.53±5.30	0.174
STRESS SCORE	371.33±136.8	243.06±169.33	0.009*

P value <0.05 was considered significant*

In the 1st year student group serum cortisol was slightly higher in smokers but did not reach statistical significance. The non smokers however showed higher level of stress score.

Table 2- Comparison between non smoker and smokers in 3 rd year students

PARAMETER	NON SMOKER(15)	SMOKER(15)	P VALUE
AGE (YEARS)	20.86±0.63	20.93±0.88	0.815
BMI(KG/M^2)	21.66±3.01	22.8±3.01	0.312
WAIST CIRCUMFERENCE	77.33±7.65	82.41±14.31	0.505
SYSTOLIC BP	116.53±5.78	118.93±7.85	0.239
DIASTOLIC BP	74.80±5.22	78.26±5.28	0.349
SERUM CORTISOL	13.09±4.9	17.79±3.83	0.007*
STRESS SCORE	174.2±14.8	222.06±151.19	0.361

P value<0.05 = significant*

Among the 3rd year group serum cortisol was significantly higher in smokers than non smokers(p value=0.007).

Table 3- Comparison between non smokers among 1st year and 3 rd year students

PARAMETER	1st YEAR	3rd YEAR	P value
AGE (YEARS)	19.00±0.92	20.86±0.63	0.001*
BMI(kg/m^2)	20.51±2.59	21.66±3.01	0.27
WAIST CIRCUMFERENCE	79.60±6.76	77.33±7.65	0.397
SYSTOLIC BP	119.06±8.64	116.53±5.78	0.355
DIASTOLIC BP	76±8.28	74.80±5.22	0.639
SERUM CORTISOL	13.18±3.81	13.09±4.9	0.957
STRESS SCORE	371.33±136.8	174.2±14.8	0.001*

P value<0.05 = significant*

Among the non smokers of both groups, stress score was significantly higher(p value=0.001).

Table 4- Comparison between smokers among 1st and 3rd year students

PARAMETERS	1st YEAR	3rd YEAR	P value
AGE (YEARS)	19.93±0.96	20.93±0.88	0.006*
BMI(kg/m^2)	21±2.31	22.8±3.01	0.078
WAIST CIRCUMFERENCE	78.26±3.26	82.41±14.31	0.327
SYSTOLIC BP	116±8.45	118.93±7.85	0.333
DIASTOLIC BP	78.93±10.41	78.26±5.28	0.827
SERUM CORTISOL	15.53±5.30	17.79±3.83	0.192
STRESS SCORE	243.06±169.33	222.06±151.19	0.885

P value<0.05= significant(*)

The 1st year and 3rd year group showed age difference which was significantly higher in 3rd year with p value=0.006. No other appreciable difference was found in other parameters.

Table 5 -Comparison of combined non smokers and smokers from both the groups

PARAMETERS	NON SMOKER	SMOKER	P value
BMI(kg/m^2)	21.09	21.90	0.269
WAIST CIRCUMFERENCE	78.46	80.34	0.421
SYSTOLIC BP	117.8	117.46	0.868
DIASTOLIC BP	75.4	86.55	0.104
SERUM CORTISOL	13.13	16.66	0.004*

P value<0.05 is considered significant(*)

Collective comparison among the two groups showed that Smokers had a significant higher cortisol level (p value=0.004)

DISCUSSION

Many studies have associated smoking with psychological stress. [8] Many smokers report that smoking helps them alleviates stress. [9] On the other hand some studies report that smokers experience higher levels of stress than non smokers given a particular situation. [10] Smoking helps to reduce emotional disturbance during acute stress. Paradoxically nicotine activates the system that mediates the stress response and may prolong the hormonal and cardiovascular response to stress. This may represent a break down in body's ability to cope efficiently with stress and may contribute to smoker's susceptibility to acute stress. One of the significant responses to smoking is the activation of the Hypothalamo-pitutary-adrenal (HPA) axis. A consequent increase in HPA mediated hormones, particularly cortisol occurs. HPA is primarily used in body's response to physical and mental stress. It is hypothesized that prolonged activation of

this axis can be detrimental to the health and may provide a link between mental and physical stressors. [11] The effects of habitual smoking on the cortisol levels are mixed, with some studies indicating no relationship, [12] whereas others reporting increased cortisol secretion. [2] Chronic activation of the HPA axis and the consequent hypercortisolism may have adverse outcomes on the health of the individual. Kirschhbaum et al [13] studied responses to repeated psychological stress in a subpopulation of healthy non smoking men and found that, all subjects did not react equally to the same stressors. They described them as low responders and high responders based on their cortisol levels. The low responders quickly adapted to the everyday stressors and their cortisol level which had risen on first day of exposure to the stressor fell on consecutive days, whereas the high responders had a significant elevation of cortisol levels on each day of their exposure.

In the current study we found that 1st year students who smoked for 6-9 months and less than 5 cigarettes per day, had higher cortisol levels than their non-smoking counterparts but not statistically significant. Stress score was significantly higher among non smokers. This also proves that psychological stress was not the cause for smoking in student population. Among the students who smoked for longer duration the cortisol levels were significantly higher. When the smoking groups of the 1st and 3rd year were compared the cortisol levels were not significantly different although it was slightly higher in senior students. Their finding show that cortisol levels are higher among smokers and this is in agreement with earlier reports. [2,10,14] The levels tend to increase with longer duration of smoking although the senior students were also smoking less than 5 cigarettes per day. The raised cortisol levels in the smokers not reporting higher stress levels than their non smoking counterparts support the hypothesis that smoking is responsible for activation of the HPA axis and the resulting hypercortisolism.

CONCLUSION

This study reveals that smoking is not taken up to reduce stress as in commonly perceived. Also in the absence of confounding psychological stress among the smokers, higher cortisol level in them attributed to the effect of nicotine on HPA axis causing hypercortisolism. The effect of nicotine on cortisol secretion becomes demonstrable as early as 6 months of mild persistent exposure (<5 cigarettes/day, inconsistent) and perpetuates with the duration of exposure even with low dose (up to 4 years, <5 cigarettes/day, inconsistent).

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