Original Research Article

Effect of cigarette smoking on various biochemical parameters in patients attending OPD of RMMCH

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Abstract

Background: Smoking harms nearly every organ of the body and diminishes a person's overall health. Millions and millions of peoples have health problems due to the habit of smoking. It is now a burden worldwide because smoking addiction of teenagers increasing immensely. Globally, smoking is not only a leading cause of cancer as well as various heart diseases. Smoke contains several carcinogenic pyrolytic products like Polycyclic Aroma0tic Hydrocarbons (PCAH), acrolein etc. are irreversible binds to DNA, causes genetic mutation and cancer.
Aim: To evaluate the effect of smoking on Blood biochemistry parameters.
Materials and methods: Totally 68 subjects were included in the study. 34 current smokers who came from in and around Chidambaram to the RMMC and Hospital who fulfilled the inclusion criteria were selected as an experimental group. Another 34 non-smokers of the same age group were included separately in this study as a control group. So a total of 68 respondents were contacted for the study. The primary data were collected during the 2016-2017 period of around one year. biochemical parameters such as blood glucose, blood urea, and lipid profile were analyzed using standard methods.
Results: While comparing the mean Blood sugar, Serum Cholesterol, Triglycerides, HDL, and LDL values, it was found to be more in smokers compared to that of non-smokers and this was significant at 5% level for Blood sugar and at 1% level for other values like cholesterol, TGL, HDL, and LDL. The mean Blood urea value had no significant association with smoking.
Conclusion: Subchronic and acute exposure to tobacco smoke and various tobacco smoke constituents have been shown to elicit a wide variety of cardiovascular effects in animal models. These effects include decreased oxygen-carrying capacity, resulting in ischemia, platelet activation,

endothelial damage, altered lipoprotein levels and increased arterial wall thickness which can promote atherosclerosis, and thrombosis. Ischaemia, atherosclerosis, and thrombosis increase the risk of myocardial infarction and other serious cardiovascular effects.

Key words

Nicotine, Lipid Profile, Blood Glucose Level, Serum Urea.

Introduction

The link between smoking and pulmonary diseases was first recognized in the 1870s, but it was not until 1964 that the US Surgeon General's report warned of a potential relationship between smoking and emphysema. In every population for which prevalence data are available, airflow obstruction is more common among smokers [1]. In the most multi-variety analysis, cigarette smoking is the only statistically significant predictor of airflow obstruction after adjustment for the effects of age and initial forced expiratory volume in one second (FEV1) [2]. Altogether, cigarette smoking accounts for about 80% to 90% of COPD cases in the United States. Respiration as the term is generally used includes two processes: external respiration- the absorption of O2 and removal of CO2 from the body as a whole; internal respiration - the utilization of O2and production of CO2 by the cells and the gaseous exchanges between the cells and their fluid medium [3]. The respiratory system is made up of a gas exchanging organs, the lungs, and a pump that ventilates the lungs. The pump consists of the chest wall, the respiratory muscles which increase and decrease the size of the thoracic cavity, the areas in the brain that control the muscles and the tracts and nerves that connect the brain to the muscles [4]. There is strong evidence that an adverse relationship links smoking exposure and metabolic and physiologic response of different structures of heart and blood vessels [5]. Changes in intracellular metabolism as a consequence of smoking exposure may be determined particularly on RNA structure particles like mitochondria, ribosomes, and lysosomal vacuoles Also, intracellular DNA-metabolism that is related to genetic control may be altered by smoking exposure [6]. In an attempt to clarify several unsolved and widely debated questions on the relationship between molecular and biochemical damage from smoking and cardiovascular system, this review describes those mechanisms which harm the cardiovascular system as a consequence of smoking exposure on a structural level, the biochemical and functional characteristics of modified cells, the type of response as well as the relationship between smoking and cardiovascular pathology [7].

Materials and methods

Totally 68 subjects were included in the study. 34 current smokers who came from in and around Chidambaram to the RMMC and Hospital who fulfilled the inclusion criteria were selected as an experimental group. Another 34 nonsmokers of the same age group were included separately in this study as a control group. So a total of 68 respondents were contacted for the study. The primary data were collected during the 2016-2017 period of around one year. biochemical parameters such as blood glucose, blood urea, and lipid profile were analyzed using standard methods.

Method of sample selection

Obviously, the sample did not include female smokers or children which are uncommon in this part of the country and hence it was not included in the sample size. The selected sample size is justified by their habit of smoking and full cooperation in the survey.

The test and the control group were assessed for their general health including height, weight, to be normal. Blood samples were collected and blood sugar, urea, lipid profile, were analyzed.

Inclusion criteria

- Smokers of the age group above 20.
- Smokers of shorter as well as longer duration (chronic smokers).
- Smokers with smoking-related bronchitis.

Exclusion criteria

- Smokers of the age group less than 20 and more than 50.
- Smokers with major respiratory problems except for smoking-related bronchitis.
- Smokers with cardiac problems.

For categorical variable chi-square test was used. P value of <0.05 was considered as statistically significant.

Results

Table - 1 shows the comparison between smokers and nonsmokers in relation to biochemical variables. While comparing the mean Blood sugar, Serum Cholesterol, Triglycerides, HDL, and LDL values, it was found to be more in smokers compared to that of nonsmokers and this was significant at 5% level for Blood sugar and at 1% level for other values like cholesterol, TGL, HDL, and LDL. The mean Blood urea value had no significant association with smoking.

Table – 1: Comparison between smokers and non-smokers in relation to biochemical variables.

Blood Biochemistry Variables	**Smokers (Experiment Group) N=34**		**Non-smokers (Control Group) N=34**		**Mean Diff**	**'t' value**	**P value**
	SD	**Mean**	**SD**				
Blood Urea (mg%)	28.00	5.30	25.47	4.92	2.53	11.44	**0.07 NS**
Blood Sugar (mg%)	102.85	14.49	91.65	8.47	11.2	52.40	0.01*
Cholesterol (mg%)	173.38	19.15	157.74	19.78	15.64	13.61	0.00**
TGL (mg%)	9532	1827	92.97	14.89	235	52.06	0.00
HDL (mg%)	47.65	5.63	44.94	2.8	2.71	8.07	0.00**
LDL (mg%)	95.03	20.32	109.68	9.76	-14.65	7.84	0.00*

(*significant at 5% level, **significant at 1% level, NS-Non significant)

Discussion

The impact of cigarette smoking on glucose metabolism and its regulation is complex. The increase in concentrations of plasma glucose and HbA1c in smokers in this study suggested disturbance in glucose homeostasis [8]. The prolonged action of catechins and their derivatives are due to their re-entry into circulation after the first phase of reactions by enterohepatic circulation from gut to liver which might have contributed to glucose homeostasis. Reports also revealed that colon microflora help in degradation of catechins into smaller assimilable forms facilitating reabsorption from the gut [9]. Besides, smoking directly affects b-cells and suppresses insulin secretion in smokers which were attributed to the effects of nicotine on b-cells and influences on the secretion of insulin through nicotinic acetylcholine receptors on b-cells and that nicotine increases apoptosis of islet b-cells [10]. Cigarette smoking leads to cardiovascular diseases for which lipid parameters are considered to be reliable markers. Higher cholesterol, LDL-C, VLDL-C, triglycerides with a decrease in HDL-C were reported in smokers in this study and it is in agreement with earlier studies. In green tea consuming smokers, these parameters (cholesterol, triglycerides and other lipoprotein patterns) were found to be close to normal range suggesting either no or reduced cardiovascular risk [11]. While comparing the mean values of blood urea, blood sugar, serum cholesterol, HDL and LDL between smokers and non-smokers it

was found. that except blood urea all other mean values were increased in smokers compared to nonsmokers which were significant at l% level [12]. This may be due to the reason that smoking can interfere with the sugar and lipid metabolism and so increase the risk of developing coronary artery disease. Our inference regarding the Blood sugar in smokers is supported by other studies conducted by various researchers [13]. According to Rose JE, et al. (2000) the risk for impaired fasting glucose and type 2 diabetes increased in smokers in a dose-dependent manner as the number of cigarettes smoked per day and the number of years of exposure (the long term effect of cigarette smoking for current smokers) increased [14]. They also added that past smokers were associated with a non-significantly higher risk for impaired fasting glucose compared with nonsmokers but was not associated with the risk for type 2 diabetes [15].

Conclusion

We also recommend that biochemical verification is used in most or all studies of smoking cessation in special populations, such as adolescents, pregnant women, and medical patients with smoking-related diseases. There are circumstances under which the added precision gained by biochemical verification is offset in such a way that its use is not required and may not be desirable.

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