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Lipid Profile and Lipid Peroxidation Level among Smokers and Non-Smokers at Kirtipur

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ABSTRACT

Objective: To determine the alteration in serum lipid profile and lipid peroxidation among smokers and non-smokers.

Methodology: A community-based, case-control study was conducted in Kirtipur Municipality, Nepal from May 2018 to October 2018. A total of 160 local participants aged from 20 to 55 years were engaged out of which, 80 were healthy smokers, and 80 were non-smokers. Questions related to socio-demographic information were taken. Also, venous blood was collected for biochemical analysis of Triacylglycerides (TAGs), Total Cholesterol (TC) and High Density Lipoprotein (HDL) via spectrophotometer. Lipid peroxidation product as Malondialdehye (MDA) was performed via Thiobarbituric Acid Reactive Substance (TBARS) method. Low Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL) was calculated using Friedewald's formula.

Result: In this study, male (80, 50.0%) had a mean age of 41±10.4, while female (80, 50.0%) had a mean age of 43±7.4 whilst majority of them were between the ages of 41 and 50. (104, 65.0 %). This study revealed that mean serum TC in non-smokers was 158.96±38.95mg/dl while it was significantly higher in smokers, i.e., 206.30±33.06mg/dl with p-value <0.001. Similarly, TAGs, LDL, VLDL, and TC were significantly higher (p-value<0.001) in smokers than non-smokers. In context of intensity and duration of cigarette smoking by participants, it showed significant (p-value < 0.05) association with an increased TAGs, TC, LDL and VLDL and decreased HDL level. MDA level increased significantly (p-value<0.001) in smokers than non-smokers.

Conclusion: Regular and chronic smokers showed significantly higher levels of TC, TAGs, LDL, VLDL, and MDA than non-smokers, suggesting that cigarette smoking alters lipid profile and enhances lipid peroxidation, increasing the risk of atherogenesis.

I. INTRODUCTION

One of the most hazardous yet prevalent human tobacco behavior is smoking cigarettes(1). Cancer, heart disease, stroke, lung disease, diabetes, and chronic obstructive pulmonary disease are all caused by it(2). More than 8 million people die each year from the tobacco epidemic, which is one of the greatest risks to global public health. This figure includes roughly 1.2 million fatalities from exposure to second hand smoke(3). Additionally, according to a published factsheet from 2018, Nepal's juvenile population (aged 13 to 17) and economically productive population (aged 30 to 69) both had smoking rates of 11.0% and 36.0%, respectively(4). According to previous researches, smoking cigarettes causes an elevation in blood cholesterol, Reactive Oxygen Species (ROS), and other adverse consequences that pose a health risk(5,6).

Cigarette smoking alters the lipid profile level resulting higher Total Cholesterol (TC), Triacylglycerides (TAGs), Very low-density lipoprotein (VLDL), low-density lipoprotein (LDL) levels, and lower High-density lipoprotein (HDL) level (7). In healthy non-smokers, aerobic metabolism results in the production of a small amount of ROS. However, excessive generation or poor clearance during smoking causes oxidative stress, resulting in lipid peroxidation, mutagenesis, and carcinogenesis(8). Lipid peroxidation is a well-known natural process in the body that generates small amounts of Malondialdehyde (MDA). Increased MDA levels are implicated in the development of ample of diseases, including lung cancer, asthma, diabetes, coronary heart disease, oral cancer, etc.(1).

There haven't been conducted enough studies in Nepal to access the association between lipid profile and lipid peroxidation levels among active smokers and healthy non-smokers. These blood parameters are directly related with cardiovascular system (8). In this backdrop, the

current study attempted to determine whether there is a difference in serum lipid profile and lipid peroxidation levels between smokers and non-smokers of Kirtipur Municipality, Nepal.

II. MATERIALS AND METHODS

2.1 Study Design and Site

A community-based case-control study was conducted in the Kirtipur Municipality from May 2018 to October 2018. All the biochemical assessments and laboratory experiments were carried out at the Department of Medical Laboratory Technology, Janamaitri Foundation Institute of Health Sciences, Hattiban, Lalitpur, Nepal.

2.2 Study Population and Criteria

This study entailed 160 volunteer participants aged from 20 to 55 years. Out of total participants, 80 were healthy active smokers who smoked 100 cigarettes in their lifetime with no clinical symptoms were considered as case. Other 80 healthy non-smokers were considered as control. In addition, exclusion criteria were set for those who had history of diabetes, endocrine disorder, hypertension, age above 55 years, obesity, renal disorder, coronary artery diseases and history of drug intake: β-blockers, lipid lowering drugs, steroids.

2.3 Sample Collection and Processing

After obtaining informed consent and administering a questionnaire, blood samples were collected. Every participant who approved to involve in this study had a 12-hour fasting blood sample drawn from a peripheral vein for lipid profile and lipid peroxidation testing. Serum TC, TAGs and HDL was measured spectrophotometrically by using semi-automated biochemistry analyzer i.e. Biolyzer 100 with commercially prepared reagents i.e. accurex. Lipid peroxidation product MDA was estimated by the Thiobarbituric Acid Reactive Substance (TBARS) method as described by Buege and Aust method.Serum Cholesterol was determined by CE-CO-PAP, enzymatic, end- point method. The expected value of serum cholesterol was<200 mg/dl.Serum, Triglyceride was evaluated by GPO-PAP, End point method. The expected value of serum triglyceride was<100mg/dl. Serum HDL was determined by enzymatic method. The expected value of serum HDL is 40-60 mg/dl. Serum LDL and VLDL was calculated by Friedewald formula.

2.4 Statistical Analysis

For data entry and patient coding, Statistical Package for the Social Sciences (SPSS) version 21 was used. For each set of tests, the mean and standard deviation were calculated. Independent T-test and ANOVA test were used to calculate the p-value for comparison of mean and standard deviation values and p-value less than 0.05 was considered as significant.

2.5 Ethical consideration

Informed consent was taken from each participant and ethical approval was taken from Nepal Health Research Council (NHRC) with reference number 3133.

III. RESULTS

3.1 Demographic characterization of smokers and non-smokers participants

In this study, a total of 160 people were enrolled, with 80 of them being smokers and the other 80 being non-smokers. Males (80, 50.0%) had a mean age of 41±10.4, while females (80, 50.0%) had a mean age of 43±7.4. The majority of participants were between the ages of 41 and 50. (104, 65.0 %).

As shown in Table 1, most of the participants in the study were engaged in their businesses, with Hindus accounting for the highest proportion (106, 66.3%). There was a significant relation in between gender of the participants with p-value <0.001 among smokers and non-smokers. In terms of occupation, there was a significant difference between unemployed people and people working in government services and business (p-value < 0.05). Duration of smoking among smokers was as follows: 15 smokers had been smoking for 1 year, 18 for 1-5 years, 16 for more than 15 years, and 31 for 6-15 years.

Table 1: Demographic characterization of smokers and non-smokers participant

Variables	Total numbers (%)	Number (%)		p-value
		Smokers (n=80, %)	Non-Smokers (n=80, %)	
Gender				
Male	80 (50.0)	61 (76.3)	19 (23.8)	Ref.
Female	80 (50.0)	19 (23.7)	61 (76.2)	<.001
Age-group				
20-30	23 (14.4)	10 (12.5)	13 (16.2)	Ref.

31-40	27 (16.9)	10 (12.5)	17 (21.3)	0.64
41-50	104 (65.0)	56 (70.0)	48 (60.0)	0.37
51-55	6 (3.8)	4 (5.0)	2 (2.5)	0.32
Occupation				
Unemployed	26 (16.3)	19 (23.8)	7 (8.8)	Ref.
Government Services	38 (23.8)	15 (18.8)	23 (28.7)	.008
Business	49 (30.6)	21 (26.3)	28 (35.0)	.012
Private services	47 (29.4)	25 (31.3)	22 (27.5)	.096
Religion				
Hindu	106 (66.3)	59 (73.8)	47 (58.7)	Ref.
Buddhist	52 (32.5)	21 (26.3)	31 (38.8)	0.07
Others	2 (1.3)	-	2 (2.5)	0.24

3.2 Comparison of Serum Lipid Profile parameters and MDA level among participants

According to Table 2, the lipid profile of study participants appeared to be higher in smokers than in non-smokers, while HDL appeared to be lower in smokers. In this study, there was a significant difference (p-value < 0.001) between smokers and non-smokers in all lipid profile parameters. Furthermore, smokers had higher serum MDA levels (lipid peroxidation) than non-smokers, and the relationship was significant (p-value < 0.001).

Table 2: Comparison of serum lipid profile parameters and MDA level among participants

Parameters	Smokers	Non-smokers	P-value
TC	206.30±33.06	158.96±38.95	<.001
TAGs	198.09±73.13	141.53±44.39	<.001
HDL	36.29±8.61	38.87±8.20	<.001
LDL	130.41±36.83	91.78±38.19	<.001
VLDL	39.61±14.62	28.30±8.87	<.001
MDA	2.75±1.57	1.14±0.83	< .001

3.3 Comparison of smokers' lipid profiles and lipid peroxidation levels with their smoking frequency and duration.

In this study, the level of lipid parameters and MDA showed up to be increasing and HDL decreasing with increasing daily use of cigarettes by smokers, with a significant relationship (p-value <0.05) in TAGs, cholesterol, LDL, and HDL level. Other parameters, however, showed no correlation. Besides, serum HDL levels decreased as smokers' smoking duration (in years) increased, while MDA levels and other lipid profiles appeared to increase. Similarly, a significant relationship (p-value <0.05) was found in serum cholesterol, LDL, and HDL levels, while others showed no relationship.

Table 3: Comparison of Lipid Profile and Lipid Peroxidation Level with Frequency and Duration of Smoking by Smokers.

Parameters	TAGs	TC	LDL	HDL	VLDL	MDA
No. Of Cigarette						
1-5	192.51±56.76	192.85±25.11	119.31±28.42	35.03±6.44	38.50±11.35	2.78±1.57
6-10	197.35±50.79	206.53±30.03	132.93±27.94	31.83±4.27	39.47±10.41	2.82±1.40

11-15	227.48±28.33	224.02±33.19	141.77±46.74	30.69±5.11	45.49±25.66	2.47±1.93
16-20	229.78±27.33	233.07±47.39	165.68±49.63	28.71±4.46	47.95±5.46	2.90±1.52
P-Value	<0.05	<0.05	<0.05	<0.05	0.15	0.82
Duration of Smoking in years						
<1	185.76±25.62	175.46±27.31	104.98±34.52	33.33±4.16	37.15±5.12	2.73±0.37
1-5	203.06±74.89	191.03±13.39	117.57±22.22	32.85±4.97	40.61±14.97	2.28±1.57
6-15	221.68±97.55	212.65±34.11	135.35±39.08	32.97±6.82	44.33±19.51	2.63±1.48
>15	223.81±32.04	215.43±36.13	143.14±38.95	32.33±5.54	46.96±6.40	3.07±1.69
P-value	0.37	<0.05	<0.05	<0.05	0.24	` 0.143

IV. DISCUSSION

Cigarette smoking is one of the predominant factors for variation in lipid profile and anti-oxidant levels in humans, resulting in lipid peroxidation. As a result, elevated serum MDA levels indicates an increased in ROS production, suggesting a possible role in atherogenesis, which leads to coronary heart disease(9).

This study revealed that mean serum TC in non-smokers was 158.96±38.95mg/dl while it was significantly higher in smokers, i.e., 206.30±33.06mg/dl with p-value<0.001. Similarly, TC, TAGs, LDL, and VLDL were significantly higher (p-value<0.001) in smokers as compared to non-smokers thereby implying a direct dose response relationship. Similar results were obtained from studies conducted in Srilanka and India with significant (p<0.05) elevation of TC, TAGs, LDL, and VLDL level and declination in HDL level among smokers(10,11). When it comes to the intensity and duration of cigarette smoking by participants, it is significantly (p-value <0.05) associated with an increase in lipid parameters except for TAGs in duration of smoking. In both cases, VLDL hasn't been significantly increased, but, significant (p-value <0.05) decrease in HDL level was seen. According to a review of 54 published studies, smokers had higher levels of TC by 3%, TAGs levels by 9.1%, VLDL levels by 10%, LDL levels by 1.7%, and lower levels of HDL by 5.7% when compared to nonsmokers(12).The following mechanism may explain the increase in lipid levels in smokers: Cigarette smoking causes nicotine absorption into the body, which causes lipolysis and the release of free fatty acids into the bloodstream to the liver, increasing hepatic TAGs and VLDL synthesis and thus increasing blood TAGs and VLDL concentrations(13). Further, our study found that smokers had a significantly lower level of HDL (p<0.05) than non-smokers. Several studies have found that smokers have high levels of homocysteine in their blood. Plasma homocysteine is negatively correlated with HDL, resulting in a decrease in HDL levels. Other studies have investigated that higher LDL and VLDL levels in the blood contribute to lower HDL levels(14).

In smokers, endogenous lipid peroxidation has been found higher than non-smokers. Likewise, our study concluded that MDA levels in smokers is significantly (p-value <0.001) higher than the non-smokers. This revealed that the smokers are more prone to oxidative burst resulting cardiovascular risks. According to studies conducted in India(15) and Nigeria(16), smokers are statistically more likely than non-smokers to have inflated MDA levels. Furthermore, in our study, participants' MDA levels appeared to increase with increasing intensity and duration of smoking though there was no significant association (p-value ≥ 0.05). Plethora of studies have demonstrated that smoking causes free radical assaults on polyunsaturated fatty acids in cell membranes, resulting in lipid peroxidation and the formation of end products such as MDA, ethane, and pentane. This event causes increased MDA levels in smokers(17). It is noted that peroxidised lipids are significantly associated with atherogenesis and its severity (18). Conclusively, rigorous rules addressing early intervention and smoking cessation, as well as their accurate execution and monitoring from the ground level,

are expected so that the general population is aware of the detrimental effects of smoking on health.

V. CONCLUSION

In the nutshell, our study concluded that smokers had substantially greater levels of serum TC, TAGs, LDL, VLDL, and MDA than non-smokers, suggesting that cigarette smoking alters the lipid profile and lipid peroxidation which is associated with increasing risk of atherogenesis.

Abbreviations
TC: Total Cholesterol
HDL: High-Density Lipoprotein
LDL: Low-Density Lipoprotein
MDA:Malondialdehyde
p-value: Probability value
ROS: Reactive Oxygen Species
TAGs:Triacylglycerides
TBARS:Thiobarbituric Acid Reactive Substance
VLDL: Very-Low Density Lipoprotein

Data availability
The data used to support the finding of this study is available from the corresponding author upon request to authors.

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Conflict of interest
Do not have any conflict of interest.

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