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Original article

Comparative Analysis of lipid profile in tobacco and non-tobacco abusers

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Abstract

Background and Aim: Premalignant lesions and conditions significantly play important role in the pathogenesis of oral squamous cell carcinoma. The main etiology for oral premalignant and malignant diseases is tobacco consumption. For lipid profiling mostly serum is analyzed. But it is being reported saliva can also be used as an alternative to serum for diagnostic purpose. Present study is aimed to compare and correlate the serum and salivary lipid profile in healthy individual and tobacco abusers.

Material and Methods: Total 90 cases are taken and divided into 3 groups A, B, C; 30 cases in each group. Group A comprises the healthy control individuals, Group B comprises the tobacco chewers, Group C comprises the tobacco smokers. . Fasting blood and unstimulated saliva sample collected and the lipid analysis (Total Cholesterol - TCHL, Triglycerides - TGL, High density lipid cholesterol - HDL, Low density lipid cholesterol - LDL, very low-density lipid cholesterol - VLDL) were done on an autoanalyzer based on spectrophotometric principle.

Results: Significant differences were appreciated in TCHL, HDL and LDL level when compared in saliva for Healthy individual's vs tobacco chewer's vs smokers as< 0.05. Moderate positive correlations between serum and saliva parameters were seen except for LDL level. Moderate

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positive correlation between serum and saliva parameters except LDL was observed between variables.

Conclusion: Saliva can be used as diagnostic tool for the analysis of lipid profiling. It is advantageous because of non-invasive technique for its collection. However, diagnostic value of saliva has to be determined in terms of sensitivity, specificity and reproducibility in larger samples and different disease setting.

Key Words: Oral Squamous Cell Carcinoma, Saliva, Serum, Total Cholesterol

Introduction

In the 16th century tobacco was first introduced by Portuguese in India now India became one of the world's top consumers. The World Health Organization (WHO) has attributed 4 million tobacco-related deaths every year which will rise to 8.4 million by 2020.[1] The threshold level of 5 mg of nicotine per day can be established and sustained addiction.[2] People are aware that tobacco-induced cancer is the increasing cause of death. But they are least aware that tobacco can also cause death due to coronary artery disease.[3]

Lipids are the biomolecules which are insoluble in water and soluble in solvents like chloroform and ether and these are heterogenous in nature and found in the cell membrane. [4,5] Lipids are the main component of cell membrane and helps in the maintenance of cell integrity. It also helps in cell division, cell growth and DNA stabilization.[4,5] The fatty acids in the diet are being converted into triacylglycerols by liver for fuel or as precursors. These triacylglycerols are then packaged with specific apolipoprotein into very low-density lipoprotein cholesterol (VLDL). These VLDL are transferred to muscles and adipose tissue through blood. Some VLDL gets converted into low density lipoprotein (LDL) by the loss of triacylglycerols. LDL helps in the transport of cholesterol to extra hepatic tissues through the specific plasma membrane receptors for LDL on these tissues. One more type of lipoprotein is High density lipoprotein cholesterol (HDL) helps in transfer of excess cholesterol back to the liver from the extrahepatic tissues.[6] Cholesterol is considered to play an important etiological role in coronary heart disease.[7] In literature it has been found to have inverse relationship of lipid profile to oral premalignant and malignant diseases.[8,9]

Premalignant lesions and conditions significantly play important role in the pathogenesis of oral squamous cell carcinoma.[10] The main etiology for oral premalignant and malignant diseases is tobacco consumption. The tobacco carcinogens cause oxidation/ peroxidation of polyunsaturated fatty acids by the production of free radicals and reactive oxygen species. The peroxidation of fatty acids in turn affects the constituents of cell membrane.[11] Smoking has been shown to alter lipid/lipoprotein levels. Komiya et al.[12] reported smokers with Brinkman index 554 (defined as the number of cigarettes smoked per day multiplied by duration of smoking in years) to have 1.657 times the odds of having abnormal triglyceride (TG) levels among Japanese males aged 24–68 years. Kuzuya et al.[13] also reported smokers to have lower levels of high-density lipoprotein (HDL), lower levels of low-density lipoprotein (LDL), lower levels of total cholesterol (TC), and higher levels of TG than nonsmokers.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

For lipid profiling mostly serum is analyzed. But it is being reported saliva can also be used as an alternative to serum for diagnostic purpose.[14] Saliva collections being noninvasive procedure makes it advantageous over serum. Therefore, present study is aimed to compare and correlate the serum and salivary lipid profile in healthy individual and tobacco abusers.

Material and Methods

The cases are obtained from the Out-patient department of the institution. Total 90 cases are taken and divided into 3 groups A, B, C; 30 cases in each group. Group A comprises the healthy control individuals, Group B comprises the tobacco chewers, Group C comprises the tobacco smokers. Patient with chronic habits were included. But patient with any systemic diseases, other premalignant disorders and malignant disease were excluded. Ethical approval was taken from the institutional ethical committee and written informed consent was taken from all the participants.

Serum sample

With all aseptic precautions, about 5 ml of venous blood was collected. The sample was then allowed to clot at room temperature. Later the sample was centrifuged at 3000rpm for 10 mins to separate the serum. Immediately the serum was used for the estimation of lipid profile by autoanalyzer using spectrophotometric principle.

Saliva sample

Before collecting the saliva sample, patient is asked to rinse mouth thoroughly to avoid any contamination by debris and exfoliated cells. The patients were asked to pool the saliva in the floor of the mouth and to spit in sterile containers provided to them. Then unstimulated saliva was collected and centrifuged at 10,000×g for 10 mins to avoid visible precipitates. After centrifugation the saliva sample was subjected to autoanalyzer based on photometric principle.

Statistical analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2007) and then exported to data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA). For all tests, confidence level and level of significance were set at 95% and 5% respectively.

Results

Mean TCHL, TGL, HDL, LDL and VLDL values in serum are given in (Table 1) for all three study groups viz healthy individuals, Tobacco chewers and among smokers whereas Mean TCHL, TGL, HDL, LDL and VLDL values in saliva are given in (Table 2) for all three study groups viz healthy individuals, tobacco chewers and among smokers. Significant differences were appreciated in TCHL, TGL level and VLDL level when compared in serum for Healthy individuals, tobacco chewers and smokers. Whereas for HDL level significant associations were seen between Healthy individuals and tobacco chewers only, and LDL significantly correlated between healthy individual's tobacco chewers and smokers as $p < 0.05$. Significant differences were appreciated in TCHL, HDL and LDL level when compared in saliva for Healthy individual's vs tobacco chewer's vs smokers as< 0.05. Moderate positive correlations between

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serum and saliva parameters were seen except for LDL level. Moderate positive correlation between serum and saliva parameters except LDL was observed between variables.

Table 1: Mean and standard deviation (SD) for three groups of all the parameters (serum)

Variables	Healthy Individuals (Mean±SD)	Tobacco chewers	Smokers
TCHL	215.2±42.5	148.25±4.74	171.47±18.22
TGL	136.5±39.4	86.24±28.20	142.9±22.47
HDL	50.98±13.14	35.10±4.40	38.45±4.99
LDL	136.97±29.45	96.35±6.78	105.26±12.74
VLDL	27.8±8.23	17.09±5.45	29.12±3.99

Table 2: Mean and standard deviation (SD) for three groups of all the parameters (saliva)

Variables	Healthy Individuals (Mean±SD)	Tobacco chewers	Smokers
TCHL	5.52±2.48	0.59±0.71	0.59±0.47
TGL	3.84±2.26	2.24±0.4	6.20±3.90
HDL	1.71±0.3	0.72±0.32	0.48±.2
LDL	3.30±1.92	0.89±0.50	1.12±0.80
VLDL	0.72±.36	0.50±0.36	1.25±0.90

Discussion

According to Brischetto et al.[15] in cigarette smokers blood high level of carbon monoxide damage the endothelium, facilitate the entry of cholesterol in the wall of the artery. Smoking affects the concentration of the plasma lipids and lipoproteins adversely. Relative anoxemia by the formation of carboxyhemoglobin in the tissues of the smokers including the myocardium. Platelet aggregation is facilitated by smoking. Nicotine from cigarette smoke may induce cardiac arrhythmias. In a habitual smoker, Nicotine enhances hormonal secretion of norepinephrine, epinephrine, growth hormone, and cortisolwhich occur 20 or more times a day. Activation of the adenyl cyclase of adipose tissue occurs which causes lipolysis of stored TG and free fatty acids flow into plasma. By binding to plasma albumin released free fatty acids (FFA) are transported to various tissues of the body which ultimately causesstimulation of hepatic TG and VLDL synthesis. In smokers, due to the effect of smoking the plasma free fatty acid level increases which decrease the plasma HDLc and increases plasma TG and VLDL8 risk of cardiovascular events are higher in smokers with low-tar cigarettes and smokeless tobacco in comparison to nonsmokers.[16]

In our study we had found decrease in mean of serum HDL from control group to tobacco chewers & smokers which were in accordance to the results of Khurana et al[17] who has also observed decrease in HDL from control group to tobacco chewers and smokers. We had also observed decrease in mean of serum TCHL & LDL from control group to tobacco chewers and smokers which was contrary to the results of Rao et al[18] (2012) who had reported increase in TCHL, TG & LDL from control group to tobacco chewers & smokers. VLDL in our study

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

decreases from control group to tobacco chewers but increase in smokers from control group which is contradiction to Khurana et al[17] (2000) study who observed increase in VLDL between control group and tobacco chewers & smokers.

We had observed statistically significant difference between control group and tobacco chewers in all the parameters which was in consistent to the results of Rao et al[18] (2012) who had also reported the same data. Whereas when control group was compared with the smokers, we observed statistically significant difference only in case of LDL, other parameters showed insignificant difference which was contrary to the results of Rao et al[18] (2012) who observed statistically significant difference in all the parameters. Haragopal et al[1] studied the effects of chewing tobacco in serum lipid profile among the South Indian population. Authors observed that the significant difference in cholesterol values between non-users and long-term tobacco chewers which was similar to our observations. Neki[19], Craig et al[20], Adedeji[21], Nagaraj et al[22] found decreased levels of HDL and Increase in the levels of LDL, VLDL, TG, TC in smokers in their study of the association between the lipid profile and chronic smoking.

When the comparison was done in case of serum values of tobacco chewers and smokers TCHL, TG & VLDL shows significant difference which was in contrary to results of Khurana et al[17] (2000) who had observed statistical insignificant difference. They concluded that tobacco abusing whether in chewing or smoking form has impact on lipid profile, thus can increase the susceptibility to cardiovascular diseases. We have also evaluated salivary lipid profile in all three groups of all the five parameters. We had observed a moderate correlation between serum and saliva in control group, tobacco chewers and tobacco smokers of TCHL, VLDL, TG & HDL whereas low and negative correlation was found in case of serum and salivary LDL. Our results were inconsistent with the study of Singh et al[5](2015) who had also observed moderate correlation between serum and salivary TCHL, TG, VLDL & HDL and low correlation between serum and salivary LDL. They suggested that some portion of plasma lipid gets filtered in saliva. They had stated that it could be possible due to several possible mechanisms. They concluded that saliva can be used as a diagnostic tool for lipid profiling.

Conclusion

Saliva can be used as diagnostic tool for the analysis of lipid profiling. It is advantageous because of non-invasive technique for its collection. However, diagnostic value of saliva has to be determined in terms of sensitivity, specificity and reproducibility in larger samples and different disease setting. Use of serum cotinine rather than self-reports improves understanding of the relationship between smoking and unfavorable lipid profile. Tobacco has always been highlighted as a carcinogenic agent in an attempt to prevent its usage by the people. It is high time to create awareness that tobacco causes cardiac diseases too.

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