ALTERATION IN LIPID PROFILE LEVEL - AN INDICATOR FOR ORAL PRECANCEROUS LESIONS AND CONDITIONS

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ABSTRACT
Oral cancer is major health problem in developing countries and accounts for 30-40 percent of all cancers in Indian subcontinent and patients with oral precancerous condition have also been reported to show a significant tendency to develop cancer. The aim of this study was to find status of lipid profile in patients with oral precancerous lesion and condition which can be used in early diagnosis of oral precancerous lesion and condition. A randomized case control study was done among patients grouped into three categories 10 in each. Normal healthy individuals with no habit of tobacco chewing or smoking, Normal healthy individuals with habit of tobacco chewing and smoking and patients with clinically diagnosed oral precancerous lesion condition. Their fasting blood samples were analysed for plasma total cholesterol, HDL, triglycerides and LDL. The study showed significant decrease level of HDL levels (p<0.05) were observed in patients with oral precancerous lesion and condition.

KEYWORDS: Oral Cancer, Lipid Profile, Oral Potentially Malignant Disorders

INTRODUCTION:
Lipids are the major cell membrane components essential for various biological functions including cell growth of normal and malignant tissues, Cholesterol and triglycerides are important lipid constituents of the cell. Cholesterol is essential for the maintenance of structural and functional integrity of all biological membranes. Lipids are carried in the body fluids with the help of lipoproteins. Cellular uptake and regulation of cholesterol is mediated by lipoprotein receptors especially located on the surface of the cells. Cholesterol is an essential constituent of lipoprotein fractions like LDL, HDL and VLDL. 75% of the plasma cholesterol is transported in the form of LDL. Body cells sequester cholesterol from LDL fraction of lipoproteins. Alterations in circulatory cholesterol levels is associated with malignancy. Oral cancer being one of the most common cancers, constitutes major health problem in developing countries and accounts for 30-40 percent of all cancers in Indian subcontinent and patients with oral Precancerous condition have also been reported to show a significant tendency to develop cancer, our study aimed to investigate the role of altered lipid profile in early diagnosis or prognostication of oral pre malignant lesions and conditions.

MATERIALS AND METHOD
To investigate alterations in lipid profile in patients of oral pre malignant lesions and conditions randomized case control study was conducted at patients visiting the O.P.D of DR.BR AMBEDKAR INSTITUTE OF DENTAL SCIENCES & HOSPITAL, PATNA included in the study. They were further grouped into three categories 10 in each. Normal Healthy Individuals with no Habit of tobacco chewing or smoking, Normal Healthy Individuals with the Habit of tobacco chewing or smoking, Patients clinically diagnosed as Oral Precancerous Lesion (leukoplaxia) or Condition (OSMF) with habit of tobacco chewing or smoking included in the study with their concern. Venipuncture was carried out and 2ml of fasting blood samples were collected from subjects, into EDTA containing vacutettes. Samples centrifuged at 2000 r. p. m. for 15 minutes, plasma was collected and analyzed for the lipid levels. Plasma cholesterol levels were estimated using cholesterol kits obtained from Kamini Life Sciences Pvt. Ltd. (Hyderabad, AP). The estimation of cholesterol was done by modified CHOD-PAP method. 5μl of plasma sample was mixed with 500μl of working reagent that contained cholesterol oxidase, cholesterol esterase, peroxidase, 4-amino antipyrine, surfactant, phenol, buffer, preservatives and stabilizer. The mixture was incubated at 37°C for 10 minutes and absorbance was read by semi auto analyzer at 505nm. Total cholesterol was estimated by Phosphotungstate method. 10μl of plasma sample was mixed with 1000μl of triglycerides assay reagent containing Pipes buffer, lipoprotein lipase, 3,5 Dichloro-2-hydroxybenzenesulfonate, magnesium ion, ATP, lipase, peroxidase, glycerol 3 phosphate oxidase, and detergents. The mixture is then incubated for 10 min. at 37°C and absorbance was read by semi auto analyzer at 505nm. The value of VLDL cholesterol can be calculated by Friedewald calculation, if the value of triglycerides is known VLDLC= Triglycerides / 5 Similarly LDL cholesterol can be calculated based on Friedewald's equation LDL= Total cholesterol -(VLDLC)+(HDL). The data was compiled in MS excel worksheet and analysed in SPSS Software version 10.0. Frequency, Mean and standard deviation calculated and data were analysed by Student's unpaired t test and anova.

RESULTS:
The findings about lipid profile in control group and in patients of oral precancerous have been shown in table 1. Plasma lipid profile of patients with no tobacco habit under control group shows slightly higher Total cholesterol level (186± 44.04mg/dl), HDL cholesterol level (50.49± 6.49mg/dl), LDL Cholesterol level (103.82± 41.38mg/dl), VLDL Cholesterol (32.06± 10.88mg/dl) and Triglycerides level (160.35±54.43mg/dl) as compared to controls with tobacco habit with Total cholesterol level (168.48±28.12 mg/dl), HDL cholesterol level (45.62± 6.29 mg/dl), LDL cholesterol level (89.20± 25.25 mg/dl), VLDL cholesterol level (26.65± 10.41mg/dl) and finally the triglycerides level (168.48±52.06mg/dl). Lipid levels of controls with no tobacco habit and controls with habit of tobacco were also slightly higher as compared to patients with oral precancerous lesion or condition with Total cholesterol level (163.97±45.71mg/dl), LDL cholesterol level (97.60±43.33mg/dl), HDL cholesterol level (24.35±8.91mg/dl) and Triglycerides level (121.77±44.63 mg/dl) except for HDL cholesterol which is significantly high in controls without tobacco habit as compared to patients with oral precancerous lesion or conditions (42.29±7.02mg/dl). As evident in table 1, except for HDLC no significant inter-group difference seen for any of the lipid Profile components. Mean HDLC was found to be maximum in controls in no habit of tobacco followed by controls with Habit of tobacco and than the patients with Oral precancerous lesion and condition.

| TABLE 1 |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Control WHT (n=10) | Control NHT (n=10) | OPC (n=10) | Statistical Significance | | | |
| Total cholesterol | MEAN 168.48 | SD 28.12 | MEAN 136.65 | SD 44.04 | MEAN 163.97 | SD 45.71 | P 0.6897 | 0.420 |
| HDLc | 45.29 | 6.29 | 50.49 | 6.49 | 42.29 | 7.02 | 3.8088 | 0.035 |
| LDLc | 89.20 | 25.25 | 103.82 | 41.38 | 97.60 | 43.33 | 3.082 | 0.686 |
| VLDLc | 26.65 | 10.41 | 32.06 | 10.88 | 24.35 | 8.91 | 1.526 | 0.236 |
| TGL | 148.25 | 52.06 | 160.35 | 54.43 | 121.77 | 44.63 | 1.524 | 0.236 |

INDIAN JOURNAL OF APPLIED RESEARCH
Table 1 shows that except for HDLC no significant inter-group difference was seen for any of the lipid profile components. Mean HDLC was found to be maximum in controls with no habit. Of tobacco followed by controls with habit of tobacco and than the patients with oral precancerous lesions and condition.

DISCUSSION:
Cholesterol and triglycerides, important lipid constituent of cell, are essential to carry out several vital physiological functions including membrane biogenesis. In some malignant diseases cholesterol undergoes early and significant changes. Low cholesterol levels in the proliferating tissues and in the blood compartments could be due to the process of underlying premalignant or malignant disease. In our study we observed a non significant decrease in the mean cholesterol level in subjects with oral precancerous conditions as compared to controls. This is similar to previous findings of Chyoun et al (1992) 5, Monika E et al (2000) 6, Patel PS et al (2004) 7, Mehrotra Ravi et al (2009) 8 who also found decrease in total cholesterol level in cancer and precancerous condition. It is due to increased lipid peroxidation which is induced by carcinogens that involves the oxidation of unsaturated fatty acids present in cell membranes leading to tissue injury. Thus there is more utilization of cholesterol in new membrane biogenesis by cells leading to low plasma cholesterol level. We also found a significant decrease in plasma HDLC in patients with oral precancerous lesion or condition as compared to controls with no tobacco habit. This is in accordance to the findings of Halton et al (1998) 9, Patel PS et al (2004) 7, Ravi M et al (2009) 8, Quadir et al (2006) 10 concluded that low HDLC levels is an additional predictor of cancer and might be consequence of disease that is mediated by utilization of cholesterol for membrane biogenesis. Low HDLC levels is an additional predictor of cancer and might be consequence of disease that is mediated by utilization of cholesterol for membrane biogenesis. We found non significant difference in plasma triglycerides, LDL and VLDL levels in patients with oral precancerous lesion or condition compared to controls which similar to the findings of Alexopoulos et al (1987) 11, Patel PS et al (2004) 7 observed a significant difference. This may be explained as, nearly 80% of the plasma LDL is cleared by LDL receptors. The individuals having defective or defective LDL receptors remove plasma LDL at a much lower rate and have considerably elevated levels. Also intra group comparison of subjects with no tobacco habit was done with subjects with tobacco habit in control group, a non significant increase in plasma cholesterol level, LDL, HDLC, VLDL and TGL were observed in subjects with no tobacco habit as compared to subjects with habit of tobacco in control group which is similar to findings of Patel et al (2004) 7, K. Chattopadhayay and B.D. Chattopadhayay (2008) 12 also found low HDLC levels in their study on effect of nicotine on lipid profile. This may be due to the fact that tobacco carcinogens generate free reactive oxygen species and lipid peroxides, leading to tissue injury due to elevated lipid peroxidation further damaging the cellular structural blocks like lipids, proteins and DNA. Thus inducing more utilization of lipids for repair from degradation of major lipoproteins fractions like VLDL, LDL, HDL and TGL.

CONCLUSION
The study has shown a non significant difference in lipid profile between subjects with tobacco habit and subjects without tobacco habit. The study has shown a significant reduction in HDL cholesterol component of lipid profile in patients with oral precancerous lesions or conditions. As there is a significant change in HDLC of plasma lipid profile of oral precancerous condition patients, the HDLC component of plasma lipid profile may be helpful for the diagnosis of Oral precancerous lesions and conditions.

REFERENCES