International Blood Research & Reviews
3(1): 26-35, 2015, Article no.IBRR.2015.003
ISSN: 2321–7219

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Lipid Profile as a Marker of Pre-stage Cancer and Oral Cancer in Tobacco Users

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Authors' contributions

This work was carried out in collaboration between all authors. Authors DAT and RSR designed the study, wrote the protocol, and wrote the first draft of the manuscript. All managed the literature searches, analyses of the study performed and reviewed the patients data. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IBRR/2015/14140
Editor(s):
(1) Armel Hervé Nwabo Kamdje, University of Ngaoundere-Cameroon, Ngaoundere, Cameroon.
Reviewers:
(1) Anonymous, Afe Babalola University, Nigeria.
(2) Anonymous, Brazil.
Peer review History: http://www.sciencedomain.org/review-history.php?iid=647&id=28&aid=7006

Original Research Article

Received 20th September 2014
Accepted 3rd November 2014
Published 18th November 2014

ABSTRACT

Aim: To investigate the serum lipid profile in conventional smokers, reverse smokers and tobacco chewers and smokers and chewers associated with precancer and oral cancer.
Study Design: A cross sectional longitudinal study.
Place and Duration of Study: Department of Oral medicine and Radiology, Vishnu dental college, Bhimavaram, between October 2011 and November 2013.
Methodology: A total of 100 subjects with age range of 20-80 years were included and divided into five groups with 20 subjects in each group. The subjects in Group 1 were Conventional smokers, Group 2 were Reverse smokers, Group 3 were Tobacco chewers, Group 4 were Smokers and chewers and Group 5 with no habit of tobacco usage. Serum lipid profile levels were estimated and analyzed for all the five groups.
Results: Total cholesterol (TC) and HDL were significantly reduced in all forms of tobacco abusers of oral precancer group. There was significant decrease of TC, HDL in conventional smokers (Group 1) and Reverse smokers (Group 2), significant decrease of total cholesterol, triglycerides, HDL, LDL, VLDL in tobacco chewers (Group 3) and smokers and chewers (Group 4) of oral cancer

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group compared with the controls. Total cholesterol, triglycerides, HDL, LDL, VLDL were much lower in the oral cancer group compared with the control. Although these parameters were low in the precancer group compared with the controls and triglycerides showed non –significant p value.
Conclusion: An inverse relationship was obtained between serum lipid levels and oral precancer and oral cancer subjects. Thus the serum lipid profile can be used as an indicator to reflect the initial changes occurring in neoplastic cells, thus substantiating their use as a diagnostic adjuvant in monitoring the oral precancer and oral cancer condition.

Keywords: Serum lipid profile; forms of tobacco; oral precancer and oral cancer.

1. INTRODUCTION

Oral cancer -"*The Mysterious Myth*" is often a matter of health concern world-wide particularly in the regions of South East Asia. With its most devastating features, oral cancer is considered as the sixth most common cause of cancer death globally [1]. It is a well-documented fact that the combination of risk factors for occurrence of oral cancer includes tobacco, alcohol and betel quid. Geographic distribution of oral cancer is determined by these risk factors, hence its preponderance in South East Asia where consumption of tobacco and betel quid is high. In India, where chewing tobacco along with betel nut and reverse smoking (placing the lit end in the mouth) is practised, there is striking incidence of oral cancer among Indian population [2].

The survival rates for oral cancer patients will significantly be improved provided lesions are detected and treated at an early stage. The early detection of premalignant lesions of the oral cavity allows for treatment that may be sufficiently early to prevent their progression to an invasive carcinoma thus improving both the survival rate and quality of life. A key factor for the lack of improvement in the prognosis over the years is the fact that the significant proportion of oral cancers are not diagnosed or treated until they reach an advanced stage due to the absence of potential diagnostic marker.

Various scientific approaches were undertaken for the early detection of pre-cancerous lesions, conditions and oral cancer which included various non-invasive, genetic assays and bio-chemical assays. Researchers waved their paths in search of newer modality in detecting the oral cancer at an early stage. In recent years, emphasis has been placed on detecting biomarkers from body fluids for detecting cancer, predicting prognosis, and monitoring disease progression. Assessing the cellular condition or cellular proliferation by means of analysing the components useful in cellular membrane formation may act as a potential biomarker which will aid in diagnosing the cancerous condition at an early stage. One such biomarker may be "*Lipid profile"*. Changes in the lipid profiles have been seen in various diseased conditions including the oral cancer [3].

The seeds for assessment of oral cancer with regard to serum lipid profile were sown by Rose. In 1974, Rose reported an association of low cholesterol level with colon cancer [4]. Since then, numerous prospective studies have been undertaken to examine this particular association between cholesterol level and cancer showing increased risk of death from cancer in subjects with low plasma cholesterol level [5]. Studies have been reported alterations of cholesterol levels with different cancers such as breast [6], lung cancer, haematological cancers, ovarian cancers [7]. An inverse trend between low cholesterol and head and neck and oesophageal cancers has been reported [8].The question arises whether hypolipidemia predisposes to cancer or is at an effect of malignancy and the studies on altered lipid levels in oral cancer and precancer are few and conflicting. The present study was conducted to evaluate the implications of serum lipid profile association with precancer and oral cancer with tobacco use.

2. MATERIALS AND METHODS

A Case control study was carried out in the Department of Oral medicine and radiology. The study protocol was approved by the Institutional ethical review board and informed consent was obtained from each participant. The study subjects were selected from those who visited the Outpatient Department of Oral medicine and Radiology, Vishnu dental college, Bhimavaram, India. The study sample comprised a total of 100 subjects, each group comprising of 20 subjects.

Subjects were divided into five groups based upon habit of tobacco usage.

Group I - Conventional smokers
Group II - Reverse smokers
Group III - Tobacco chewers
Group IV - Both Tobacco smokers and chewers
Group V – Control group with no habit of tobacco use.

2.1 Inclusion Criteria

- Patients (Group I and IV) of 20 -80yrs age with history of tobacco use, presenting clinically with oral precancerous lesions or conditions, oral cancer and confirmed histopathologically were included.
- Control group included age matched patients with no habit of tobacco usage and clinically with no oral lesions.

2.2 Exclusion Criteria

- Subjects with history of drug treatment for Hyperlipidaemia.
- Subjects with history of known systemic diseases such as uncontrolled diabetes mellitus, Hypertension, thyroid disorder, and liver dysfunction associated with abnormalities in lipid profile.

3. METHODOLOGY

After taking thorough case history, informed consent from each participant was taken prior to venous blood collection and biopsy. An incisional biopsy was performed, if clinical evidence of oral precancer lesion and/or condition and oral cancer was present. After the confirmation of the precancerous state, 5 ml of fasting (12-14hrs) blood sample was collected in a sterile bottle and allowed to clot for about an hour at 37°C. The serum was then separated and stored at 4°C.

3.1 Lipid Analysis

The serum triglycerides were estimated by the GPO-PAP (Glycerol-3-phosphate Oxidase – Peroxidase), End Point Assay, Total cholesterol by CHOD-PAP (Cholesterol Oxidase – Peroxidase) and HDL-Cholesterol by PEG-CHOD-PAP (Polyethylene glycol, Cholesterol Oxidase – Peroxidase), End Point Assay with Lipid Clearing Factor (LCF). (Excel Diagnostics, India)

The LDL Cholesterol level was calculated by the Friedewald's equation.

VLDL= triglycerides/5
LDL=Total Cholesterol- HDL-VLDL.

The data collected was tabulated based upon the LDL, VLDL, HDL, total cholesterol and serum triglyceride levels separately for each of the five groups and subjected to statistical analysis. All the variables from the study were statistically analysed for the mean values, SD, standard error range, and *P*-value. Evaluation of results and statistical analysis was carried out using Student t-test and Chi square test, Anova test and post Hockey test. In all the above tests, *P*-value < .05 was taken to be statistically significant; *P*-value > .05 was taken to be statistically not significant, and *P*-value < .001 was taken to be statistically highly significant. The data were analysed using SPSS.15 software.

4. RESULTS

The study included 100 subjects, out of which 48 subjects were diagnosed as precancer and 32 subjects were diagnosed as oral cancer based on the histopathological diagnosis and 20 subjects were controls.

All the subjects selected in the study were within the age range of 20- 80 yrs. Maximum number of individuals in both precancer, oral cancer and control group were in the age range of 40 -60 yrs (Figs. 1 and 2). Among all the oral precancer group individuals**,** 31(64.5%) were males and 17 (35.5%) were females. Among all the oral cancer group, 21 (65.6%) were males and 11 (34.4%) were females. In control group with no habit of tobacco, 14(70%) were males and 6(30%) were females (Figs. 3 and 4).

The oral cancer and oral precancer were subdivided into four subgroups based upon the habit of tobacco use. Group 1: conventional smokers, Group 2: reverse smokers, Group 3: tobacco chewers and Group 4: smokers and chewers.

The serum lipid profile in the form of TC, HDL, LDL, VLDL, and triglyceride were estimated in each and every subjects of this study.

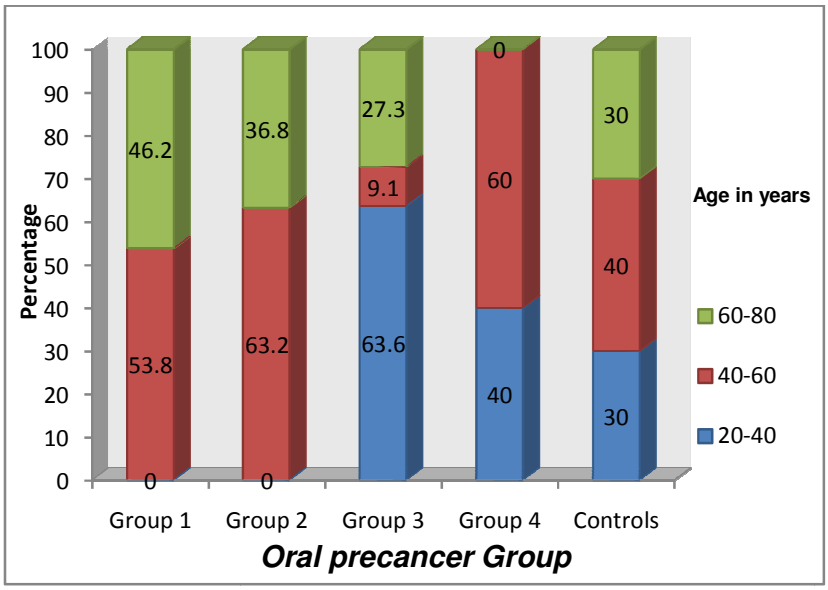

Fig. 1. Age distribution of patients studied among oral precancer group and control group

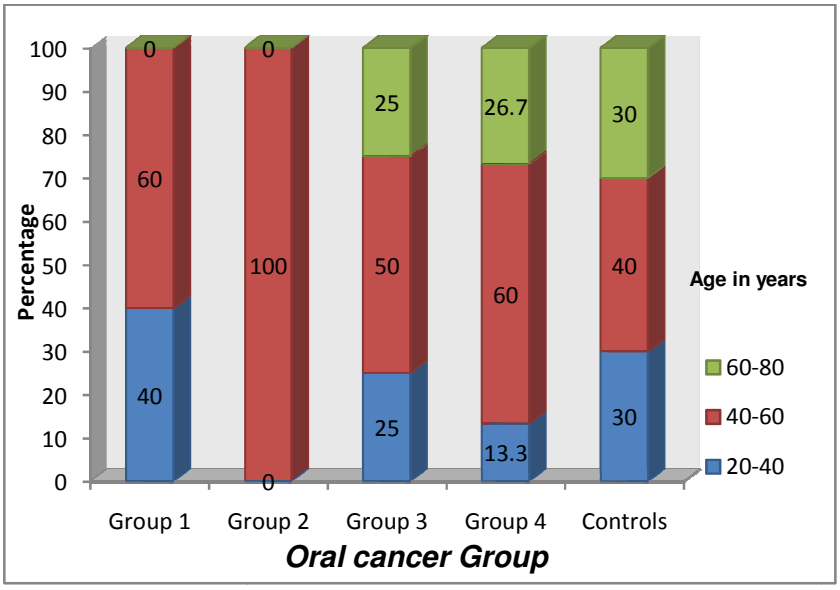

Fig. 2. Age distribution of patients studied among oral cancer group and control group

Among 48 subjects studied in oral precancer group, the commonest lesion is found to be Leukoplakia which is observed in 20 (41.7%) individuals with (*P* value < .001). Erythroplakia (*P* value = .088), and Erythroleukoplakia (*P* value = .064) showed positive correlation but there was no statistically significance between the groups. 8 (16.7%) individuals displayed monomorphic palatal lesion (*P* value= .002) and 6 (12.5%) individuals displayed multimorphic palatal lesion (*P* value =.022) between groups (Table 1).

Among 32 subjects studied in oral cancer group, 20% of individuals in Group 1 presented lesions on Alveolus, floor of the mouth and tongue. 3(75%) individuals in Group 2 presented lesions on palate. In Group 3, 4(50%) individuals had presented with lesions on buccal mucosa, buccal vestibule and retromolartrigone. 4(26.7%) individuals in Group 4 presented lesions on buccal mucosa and buccal vestibule (Table 2).

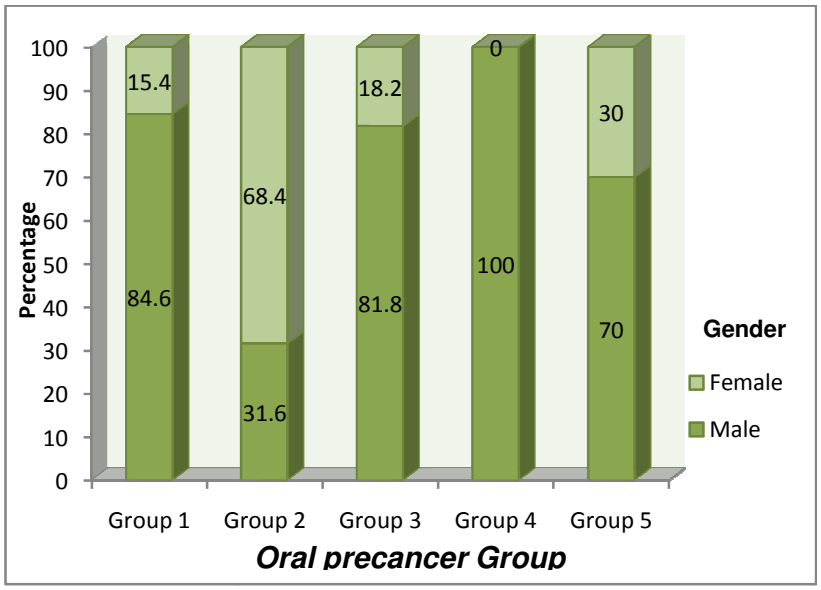

Fig. 3. Gender distribution of subjects among oral precancer group and control group

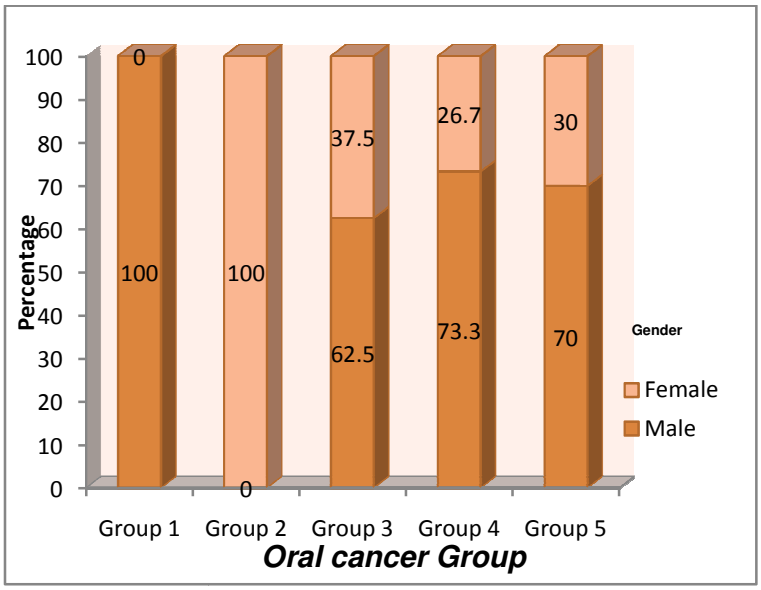

Fig. 4. Gender distribution of subjects among oral cancer group and control group

4.1 Serum Lipid Status in Oral Precancer and Control Group

The serum lipid profile in the form of TC, HDL, LDL, VLDL and triglyceride were estimated in each and every subject of the study. The mean serum lipid profile values of oral precancer and control groups are shown in (Table 3). Group 1 subjects were found to show significantly lower levels of total cholesterol (*P* value <.001) and HDL (*P* value =.036). Group 2 subjects showed significantly lower levels of total cholesterol, HDL (*P* value < .001) and LDL (*P* value = .001). Group 3 subjects were found to show significantly lower levels of total cholesterol, HDL (*P* value < .001) and VLDL (*P* value = .046). Group 4 subjects showed significantly lower level of total cholesterol (*P* value =.001) and there was no statistically significant correlation between other variables, when compared with the control group. Triglycerides showed non-significant p value among all the groups when compared with the control group.

Table 1. Lesions involved in oral precancer group using Fischer exact test

Lesion involved	Pre cancer group				Total	*P* value
	Group 1	Group 2	Group 3	Group 4		
Leukoplakia	10(76.9%)	4(21 %)	5(45.5%)	3(60%)	20(41.7%)	<0.001**
Erythroleukoplakia	2(15.4%)	0(0%)	3(27.3%)	0(0%)	5(10.4%)	0.064+
Erythroplakia	1(7.7%)	1(5.3%)	3(27.3%)	2(40%)	7(14.6%)	0.088+
Monomorhic palatal lesion	0(0%)	8(42.1%)	0(0%)	0(0%)	8(16.7%)	0.002**
Multimorphic palatal lesion	0(0%)	6(31.6%)	0(0%)	0(0%)	6(12.5%)	0.022*
Total	13(100%)	19(100%)	11(100%)	5(100%)	48(100%)	-

Table 2. Site wise distribution of lesions in oral cancer group

Lesion	Oral cancer group				Total
	Group 1	Group 2	Group 3	Group 4	
Buccal mucosa, Buccal vestibule, retromolar trigone	0(0%)	0(0%)	4(50%)	2(13.3%)	6(18.8%)
Buccal mucosa, Buccal vestibule	0(0%)	1(25%)	2(25%)	4(26.7%)	7(21.9%)
Alveolus	1(20%)	0(0%)	0(0%)	2(13.3%)	3(9.4%)
Alveolus and floor of mouth	1(20%)	0(0%)	0(0%)	2(13.3%)	3(9.4%)
Alveolus and tongue	1(20%)	0(0%)	0(0%)	2(13.3%)	3(9.4%)
Tongue	1(20%)	0(0%)	1(12.5%)	2(13.3%)	4(12.5%)
Palate	0(0%)	3(75%)	0(0%)	0(0%)	3(9.4%)
Maxillary antrum	1(20%)	0(0%)	1(12.5%)	1(6.7%)	3(9.4%)
Total	5(100%)	4(100%)	8(100%)	15(100%)	32(100%)

Table 3. Distribution of mean serum lipid profile levels in oral precancer group, control group and their statistical P-value comparisons using student t test

Pre cancer group	Total cholesterol	Triglycerides	HDL	VLDL	LDL
Group I	159.15±10.91	117.38±14.3	39.92±2.25	22.85±3.02	96.38±11.82
Group II	149.32±9.24	116.47±19.04	38.79±2.94	22.95±3.82	87.68±9.5
Group III	150.82±8.02	105.36±16.98	37.64±2.69	20.64±3.56	92.55±10.56
Group IV	162±13.64	110±15.18	41.4±2.3	21.8±3.11	98.8±8.61
Group V	184±11.37	123.15±20.74	43.15±3.9	24.5±3.91	102.85±13.14
Significance with group V					
• Group I	<0.001**	0.901	0.036*	0.704	0.496
• Group II	<0.001**	0.783	<0.001**	0.670	0.001**
• Group III	<0.001**	0.083+	<0.001**	0.046*	0.119
• Group IV	0.001**	0.603	0.787	0.574	0.951

4.2 Serum Lipid Status in Oral Cancer and Control Group

Mean distribution of serum lipid profile in oral cancer group and control group are shown in (Table 4). The serum total cholesterol levels were found to be low showing highly significant results with (*P* value < .001) among all the groups. Group 1 subjects were found to show significantly lower levels of total cholesterol, HDL (*P* value < .001), LDL (*P* value =.018) with Triglycerides and VLDL showing a non –significant p value when compared with the control group. Group 2 subjects showed significantly lower levels of total cholesterol (*P* value < .001), HDL (*P* value = .002), VLDL (*P* value = .008), LDL (*P* value = .006) with triglycerides showing a non –significant P value when compared with the control group. Group 3 and 4 subjects were found to show significantly lower levels of Total cholesterol, HDL, LDL (with *P* value < .001), triglycerides (*P* value=.011) and VLDL (*P* value =.003) when compared with the control group.

4.3 Serum Lipid Status in Precancer, Oral Cancer and Control Group

The difference in mean serum lipid profile levels in oral cancer, oral precancer and control were subjected to Student's t–test and P–values were calculated (Table 5). The difference of mean values of total cholesterol, HDL, triglyceride, VLDL, LDL were found to show lower levels

which are significant, wherein total cholesterol and HDL showing significant result with (*P* value < .001) and triglyceride, VLDL, LDL showing a significant result with (*P* value < .05) in oral cancer group when compared to precancer group. The difference of mean values of total cholesterol, HDL, showed significantly lower levels with (*P* value < .001).VLDL and LDL levels also showed significant values with (*P* value < .05) followed by Triglycerides showing non- significant levels (*P* value =.097) in precancer group when compared with the control group. The difference of mean values of all lipid parameters Total cholesterol, Triglyceride, HDL, VLDL, LDL were found to show significantly lower in oral cancer group when compared to control group with *P* value < .001.

5. DISCUSSION

Lipids are the major cell membrane components essential for various biological functions, including cell growth and division for the maintenance of cell integrity of normal and malignant tissues [9]. Cholesterol and triglycerides are important lipid constituents of cell which are essential to carry out several vital physiological functions. The lipids synthesized in the liver and the intestine has to be transported to various tissues to accomplish their metabolic functions. Because of their insolubility, they are transported in the plasma in macromolecular complexes called lipoproteins [10]. For transport in plasma, triglycerides and cholesterol are packaged into lipoproteins, which are then taken up and degraded by cells to fulfill demands for cellular functions. Thus Triglycerides, Cholesterol, LDL–cholesterol and HDL–cholesterol, VLDL constitutes *Serum Lipid Profile.*

In some malignant diseases, blood cholesterol undergoes early and significant changes. Low levels of cholesterol in the proliferating tissues and in blood compartments could be due to the process of carcinogenesis [10]. The question arises whether hypolipidemia is a predisposing factor or result of cancer. Several observational and clinical studies showed that low serum cholesterol has been associated with increased mortality from cancer [11]. The possibilities for decreased serum cholesterol levels include decreased synthesis or increased catabolism. Several authors proposed that hypocholesterima is a predisposing factor in cancer development. No causative relation has been established so far, however authors believe that hypocholesterima is infact the result rather than cause of cancer [4,7].

The present study comprises of 100 subjects in which the subjects were equally distributed into five groups with 20 subjects in each group. Out of 100 subjects studied, 48 subjects were diagnosed as precancer and 32 subjects were diagnosed as oral cancer base on histopathological diagnosis. The remaining 20 subjects were included in the control group. The peak age incidence of patients were in the age range of 40 - 60 yrs in both oral precancer as well as oral cancer.

The results and observations of serum lipid profile assay in the present study compared the statistical p values between groups with oral precancer and control group. Group 1 subjects showed significant decrease of total cholesterol and HDL. Group 2 subjects showed significantly lower levels of total cholesterol, HDL and LDL.

Table 4. Distribution of mean serum lipid profile levels in oral cancer and control group and their statistical P-value comparisons using student t test

Oral cancer group	Total cholesterol	Triglycerides	HDL	VLDL	LDL
Group I	142±7.68	104.4±12.22	34.8±3.11	19.8±2.59	87.4±9.29
Group II	138±5.72	98.25±23.16	36±3.16	18±2.58	84±2.31
Group III	131.13±4.26	100.38±4.53	33.25±2.55	19.5±0.76	78.38±6
Group IV	141.73±5.89	102.2±18.22	36.47±2.64	20.07±3.67	85.2±5.16
Group V	184±11.37	123.15±20.74	43.15±3.9	24.5±3.91	102.85±13.14
Significance with Group V					
• Group I	<0.001**	0.243	<0.001**	0.055+	0.018*
• Group II	<0.001**	0.101	0.002**	0.008**	0.006**
• Group III	<0.001**	0.031*	<0.001**	0.007**	<0.001**
• Group IV	<0.001**	0.011*	<0.001**	0.003**	<0.001**

Table 5. Distribution of mean serum lipid profile levels in oral precancer, oral cancer and control group and their statistical P-value comparisons using ANOVA test with Post Hoc Tukey test

	Pre cancer	Oral cancer	Controls	Pre-cancer vs oral cancer	Pre cancer vs controls	Oral cancer vs controls
Total cholesterol	153.65±10.86	138.66±7.18	184.00±11.37	<0.001**	<0.001**	<0.001**
TGL	113.50±17.22	101.59±15.13	123.15±20.74	0.009**	0.097+	<0.001**
HDL	39.10±2.81	35.34±2.95	43.10±3.89	<0.001**	<0.001**	<0.001**
VLDL	22.27±3.51	19.63±2.86	24.50±3.91	0.003**	0.041*	<0.001**
LDL	92.31±10.88	83.69±6.51	102.85±13.14	0.001**	0.001**	<0.001**

*+Suggestive significance (P value: 0.05<P<0.10), *Moderately significant (P value: 0.01< P ≤ 0.05), ** Strongly significant (P value: P≤0.01)*

Group 3 subjects showed significantly lower levels of total cholesterol, HDL and VLDL. Group 4 subjects showed only decrease of total cholesterol and there was no statistically significant correlation between other variables, when compared with the Group 5 (control group). Triglycerides showed non-significant difference among all the groups. (Table 3). Lohe V et al. [12] have found significant decrease of Total cholesterol, HDL in oral precancer and there is non –significant difference in subjects between no habit of tobacco and with the habit of tobacco, although both serum lipid levels and tobacco habit showed significant correlation with oral precancer independently. Kumar P et al. [13] have observed significant decrease of serum total cholesterol and HDL in oral precancer subjects with the tobacco habits as compared with controls with out habit of tobacco consumption, where as in the present study a significant decrease was observed in the total cholesterol and HDL in all forms of tobacco abusers with oral precancer.

The results and observations of serum lipid profile assay in the present study compared the statistical p values between groups with oral cancer and control group. Group 1 subjects showed significantly lower levels of total cholesterol, HDL, LDL and Triglycerides and VLDL were found to be statistically significant. Group 2 subjects showed significantly lower levels of total cholesterol, HDL, VLDL and LDL with triglycerides showing a non-significant p value. Group 3 subjects and Group 4 subjects were found to show significantly lower levels of all lipid parameters when compared with the Group 5 (control group**),** (Table 4). Patel PS et al. [10], have observed significant decrease of serum total cholesterol, triglycerides, HDL, VLDL in oral cancer patients with tobacco habit as compared with controls. The lower levels of serum cholesterol and other lipid constituents in patients might be due to increased utilization by neoplastic cells for new membrane biogenesis. Alexopolus CG et al. [14] have found non-significant difference in serum triglyceride levels between controls and patients, where as in the present study there was decrease of triglyceride levels in oral cancer patients as compared with the controls.

The decreased lipid levels in the present study were attributed to the fact that tobacco carcinogens which induces generation of free radicals and reactive oxygen species are responsible for lipid peroxidation of poly unsaturated fatty acids. Because of lipid peroxidation, there is a greater utilization of lipids for new membrane biogenesis. Cells fulfil these requirements from circulation either by synthesis through the metabolism or from degradation of major lipoprotein fractions such as VLDL, LDL or HDL [10,15,16].

Chawda J et al. [16], Goyal S et al. [17] have found significant decrease of only Total cholesterol, HDL in oral precancer, where as in the present study, the total cholesterol, HDL, showed significantly lower levels, VLDL and LDL also showed significant values followed by Triglycerides showing non-significant levels in oral precancer group when compared to control group (Table 5). As oral precancers are localised, that they do not require greater need of utilization of lipids for membrane biogenesis, they do not cause significant change as frank cancers do.

Nydegger et al. [7] observed a decrease in α - lipoprotein and cholesterol levels and was possibly due to increased catabolism of α – lipoprotein and cholesterol, decreased synthesis of α–lipoprotein and cholesterol by liver as the

synthesis is effected by tumor metabolites. In neoplastic tissue, an increased low density lipoprotein activity in tumor cells may produce hypocholesteremia. Schatzin A et al. [18] and Chyou AH et al. [19] have observed an inverse relation between lower serum cholesterol level and head and neck and as well as oesophageal cancers. Sherubin EJ et al. [20] observed a significant decrease of all lipid parameters in patients with oral cancer. The results are strengthen by the present study which also shows lower levels of serum levels of total cholesterol, Triglycerides, HDL, VLDL, LDL in oral cancer group when compared with the control group. Total cholesterol, HDL showed significantly lower levels, and Triglycerides, VLDL, LDL showed significant result in oral cancer group when compared with precancer group (Table 5).

This decrease in the level of plasma lipids can be attributed to the fact that 75% of the plasma cholesterol is transported in the form of LDL. Body cells sequester cholesterol from LDL fraction of lipoproteins. LDL receptors are necessary for metabolizing circulatory LDL and nearly 80% of the plasma LDL is cleared by LDL receptors. LDL receptor activity is several folds higher in rapidly proliferating malignant cells than non-dividing cells. This high activity of LDL receptors in proliferating tissues attributes for the lowered plasma LDL and TC levels due to their increased utilization for new membrane biogenesis [10]. This is in accordance with the previous studies, that low HDLC is an additional predictor for cancer. It might be a consequence of disease that is mediated by utilization of cholesterol for membrane biogenesis [20,21].

The levels of of serum cholesterol, triglyceride, HDL, LDL, VLDL were declined in patients with oral cancer as compared with the levels in patients with oral precancerous lesions. Thus it can be stated that lower plasma lipid status may be a useful indicator for initial changes occurring in neoplastic cells.

6. CONCLUSION

Serum lipid profile is used to measure the systemic health status of an individual. Nevertheless, it also depicts the other side of the oral health in individuals affected by oral precancer and oral cancer associated with the habit of tobacco of various forms.

In accordance with the few studies documented in the literature, the present study aimed to evaluate the levels of serum lipids and successfully fulfilled to establish a fact that, there exists an inverse relationship between the levels of serum lipids and oral precancer and oral cancer. The obtained results thus forecast the use of serum lipid profile as an indicator to reflect the initial changes occurring in neoplastic cells, thus substantiating their use as a diagnostic adjuvant in monitoring the oral precancer and oral cancer condition.

Although the role of tobacco has been established as an etiological factor for oral cancer and precancer, it may have an indirect and overall significant association with serum lipid profile. Hence, the results of the present study reveal that the low plasma lipid status in oral cancer patients can aid as a useful indicator to assess the course and prognosis of the disease at an early stage.

CONSENT

The authors declare that written informed consent was obtained from the patients before being recruited for this research.

ETHICAL APPROVAL

All author(s) declare that all procedure have been examined and approved by Institutional ethical review board, Vishnu dental college, Bhimavaram, India.

ACKNOWLEDGEMENTS

I would like to acknowledge all the subjects who had showed their willingness to participate in the study.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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