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Dyslipidemia as a Risk Factor in Oral Potentially Malignant Disorder and Oral Cancer Patients

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Abstract:- BACKGROUND: Dyslipidemia has long been linked to patients with cancer because lipids performance important part in preserving of cell veracity. AIM: To assess and compare the levels of Serum lipid profiles in oral potentially malignant disorders and oral cancer patients. MATERIAL AND METHOD: The present study involves 75 participants who were categorized into three groups. Group 1: Oral cancer consists of 25 subjects with Histopathological confirmed diagnosis of oral cancer. Group 2: Oral precancer consists of 25 subjects with Histopathological inveterate diagnosis of oral potentially malignant. Group 3: Controlled group consists of age and sex matched 25 healthy subjects. RESULTS: Substantial lower levels of mean serum HDL are found in the subjects of OPM and OC group as compared with control group; though, the mean serum CHO, LDL, TG's, VLDL levels were not significant. CONCUSION: This study concluded that on Lipids assessment HDL was decreased significantly, associated to other lipid profiles. Though, the increasing sign of low HDL-C is an early diagnostic marker for oral potentially malignant and oral Cancer diseases.

Keywords:- *Dyslipidemia, Oral Potentially Malignant, Serum HDL, Serum Lipid Profiles, Cholesterol, LDL, TG's VLDL*

I. INTRODUCTION

Cancer is one of the formidable health problems facing mankind today.[1]The word cancer was coined by Greek Physician Hippocrates. Oral cancer is a major problem in many portions of the globe.[2]On the Indian subcontinents, it is noticed that, because of social, traditional and environmental aspects and acceptance of compelling behaviour like chewing tobacco and betel nut.[1] It is estimated by WHO as the sixth most common cancer around global, two-third of these cases occurring in evolving nations. The highest incidence rates for oral cancer in the world were found in the area of South Asia. India has constantly been cited as the country with the maximum prevalence in the world.[3]In India alone over 100,000 cases are recorded each year.[4]

" Lipids are a diverse cluster of water insoluble (hydrophilic) organic fragments that can be removed from by non-polar diluents." They are the chief source of energy for the body. Examples: Fats, Oils, Steroids, Waxes and related components. Lipids are localized primarily to three compartments in the body as Plasma (Fatty acids), Adipose tissue (Triglycerides), Biological membranes (Cells).[5] Lipids are important for several purposes including cell division, development of the normal and malignant tissues. Basically, the progress of malignancy involves the unrestrained and extreme production of cells. The afresh founding cells would require many basic mechanisms well above the normal limits, used in biological process. The amplified necessity of lipids to fulfil the requirement of these new cells would be anticipated to reduce the lipid stores. So, discrepancy in blood cholesterols level in the analysis and treatment of several diseases may be expected in diseases like cancer, heart diseases. The research of changes in serum lipid profile of patients with oral precancer and oral cancer may help to know the increased malignant potential.[6]Cholesterol at either advanced or inferior level can be worrying. Health matters related to advance than the normal levels have acknowledged much communal consideration because of their association to occurrence of heart disease. Although inferences of reduced cholesterol heights endure uncertain.[8]But lower blood sterols have been associated with various cancers like Acute lymphoblastic leukaemia in children's, Myelodysplastic syndromes, Lipoprotein disorders, Hypocholesterolaemia in acute myelogenousleukemia, oesophageal cancer. Low serum cholesterol level also related to increase risk of cancer occurrence and mortality. The resolution of this present study is to estimate lipids heights in serum of oral cancer and oral precancer patients to identify the predictive consequence in cancer.

II. MATRIALS AND METHODS

The study subjects consist of 75 individuals who were divided into three groups based on the methodology and materials used. Group 1: mouth cancer is made up of 25 patients whose diagnosis of mouth cancer was confirmed by histopathology. Group 2: Oral precancer comprises 25 participants who have been diagnosed with oral precancer through histopathological.

- *Sampling:*

The study was carried out at Annamalai University's Department of General Biochemistry and Rajah Muthiah Dental College and Hospital's Department of Oral and Maxillofacial Pathology. All patients' personal and demographic information was entered into pre-designed

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Performa with their previous informed consent. Following informed consent, biopsy samples from patients suspected of having oral cancer and precancer were taken, and their samples were examined histopathologically. Blood samples from patients with precancer lesions and/or conditions, mouth cancer patients (oral squamous cell carcinoma), and healthy individuals are included in the sample.

- *Sample Collection:*

Each participant had a venupuncture in the cubital fossa to extract two milliliters of blood, which was then placed into a standard, sterile, disposable glass vacutainer tube. To avoid external damage, the samples were stored in ice packs and allowed to coagulate in an incubator at 370 degrees Celsius for approximately one hour. To separate the serum, the blood was centrifuged for five minutes at 3000 revolutions per minute (rmp). It was then kept in an ultra-low temperature freezer at -81 degrees Celsius until analysis.

- *Safety Test Tube:*

2 ml vial tube, 2 ml syringe, hot air oven, refrigerator, hot water bath, spectrophotometer, UV-VIS, and measuring tube are among the materials used to estimate the length of a lid. Pre-assembled kit for biochemical serum profile, A) A cholesterol test kit. cholesterol-enzymatic auto-enzyme (Accurex Biomedical Private limited). B) HDL, or high-density lipoprotein. HDL cholesterol autoenzyme.

A. Materials Used for Estimation of Lipids:

Sterile test tubes, 2ml Syringe, 2ml vial tube, Hot air oven, Refrigerator, Hot water bath, Spectrophotometer, UV-VIS, Measuring tube. Pre-available biochemical serum profile kit**, A**) *Cholesterol kit.* Auto enzyme new cholesterol-enzymatic, (Accurex Biomedical Private limited). B) *High Density Lipoprotein (HDL).* Auto enzyme HDL cholesterol recipitating reagent (Accurex Biomedical Private Limited), C) *Triglycerides kit (TGL).* Auto enzyme New Triglyceride enzymatic, (Accurex Biomedical Private Limited**),** blood samples of precancerous, cancerous and normal control patients. Vacutainer tubes and syringes for collecting samples, Laboratory centrifuge, Spectrophotometer, Ultra-low temperature freezer.

B. Method

The lipid analysis was done on a chemical analyser (Erba chem. 5x analysis) based on Spectrophotometric principle. The analysis was made by an enzymatic photometric test using a wavelength of *546 nm* and an optical path of 1cm and is known as "CHODPAP". By using ultra violet (UV) "spectrophotometer" (systronic company type 117, systronic, Ahmadabad India) the serum lipid profile in the form of TC, HDL, VLDL, LDL and Triglycerides was analysed on the same day of the withdrawal of blood.

- *Serum Total cholesterol (TC)* was estimated by mixing 0.01ml serum sample with 1ml of working reagent. Mixture was incubated at 37^0c for 10 minutes and the absorbance of assay mixture was measured by "spectrophotometer" at *510nm* against blank using distilled water. The values were expressed as *mg per dl.*

- *Serum Triglycerides (TLG)* was estimated by mixing 0.01ml serum sample with 1ml of triglycerides assay reagent. Mixture was incubated at 37^0c for 10 minutes and the absorbance of assay mixture was measured by "spectrophotometer" at *510nm* against blank using distilled water. The values were expressed as *mg per dl.*

- *Serum High density lipoprotein (HDL)* was estimated by mixing 0.2ml serum sample, 0.2ml HDL precipitating reagent, followed by 10mins incubation at room temperature. Mixture was centrifuged at 2800gms for 10mins to obtain a clear supernatant. Supernatant (0.1ml) obtained was mixed with 1ml of working cholesterol reagent. Mixture was incubated at 37^0c for 10 minutes. Absorbance of assay mixture was measured by "spectrophotometer" at *510nm* against blank using distilled water. The values were expressed as *mg per dl.*

The *Low-density lipoproteins (LDL)* cholesterol was estimated by calculation.

Calculation:

$$\text{LDL-C concentration (mg per dl)} = \frac{(OD2 - OD1)\ \text{Sample}}{(OD2 - OD1)\ \text{Calibrator}} \times \text{Calibrator concentration (mg per dl)}$$

The LDL cholesterol values were expressed as *mg per dl.*

Very low-density lipoprotein (VLDL) Cholesterol is calculated by "Friedewald equation" (Text book of biochemistry by Tietz, 4th edition 2006).

Calculation:

$$\text{Very Low-Density Lipoprotein (VLDL)} = \frac{\text{Triglycerides}}{5}$$

VLDL Cholesterol is expressed by *mg per dl.*

Normal lipid profile valves:

- Serum cholesterol (SC) 140-200mg/dl.
- High density lipoprotein (HDL) 45-65mg/dl.
- Low density lipoprotein (LDL) 70-130mg/dl.
- Very low-density lipoprotein (VLDL) 10-40mg/dl.
- Triglycerides (TGL) 45-165mg/dl.

III. RESULTS

In the present study, out of 75 subjects (25 oral precancer, 25 oral cancer and 25 controls), 41(54.66%) were males and 34(45.33%) were females with age range of 19-80 years with mean age of 56.32 ±12.28 (mean ± SD).

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The oral cancer group comprised of 13(52%) males and 12(48%) females out of 25 subjects, with age ranges of 35-86 years with mean age 56.32 ± 12.28 years.

The oral precancer group comprised of 15 (60%) males and 10 (40%) females out of 25 subjects with age ranges of 17-71 years with mean age 43.16 ±12.49 years.

The control group comprised of 13(52%) males and 12(48%) females out of 25 subjects, with age ranges of 32-80 years with mean age 56.32 ± 12.28 years.

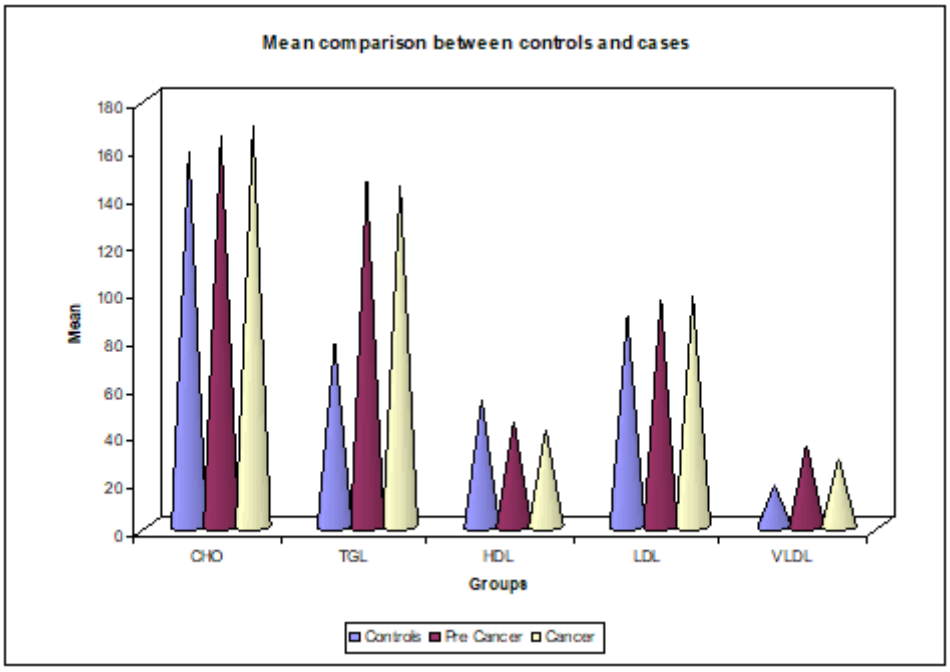
All the subjects studied were between the ages of 17-86 years within the mean age of 56 ±12 (mean ± SD).

The serum lipid profile in the form of TC, HDL, LDL, VLDL and Triglycerides were estimated in each and every subject of this study. The mean serum lipid profile values of oral precancer, oral cancer and control groups are shown in Table 1. Significant lower levels of mean serum HDL is found in the subjects of OPC and OC group as compared with control group; however, the mean serum CHO, LDL, TG's VLDL levels were not significant.

Table 1 Comparison of Mean, SD for Serum Lipid Profile in Between Controls, Oral Precancer, Oral Cancer and Overall, by ANOVA Test.

	No.	CHO	TGL	HDL	LDL	VLDL
Controls	25	158.32 ±14.38	77.08± 30.30	53.68± 5.25	88.56± 11.11	17.84± 5.33
Pre Cancer	25	165.20± 32.49	145.16± 54.69	44.64± 23.47	96.16± 29.74	34.64± 27.69
Cancer	25	168.56 ±24.20	143.44± 84.56	41.40± 2.78	96.80± 24.24	29.00± 16.89
Overall		164.03± 24.85	121.89± 67.86	46.57± 14.75	93.84± 23.06	27.16± 19.99
ANOVA	F	1.11	10.22	5.18	0.99	5.08
	P	0.34, NS	<0.001, S	<0.001, S	0.38, NS	<0.001, S

P < 0.001, Significant in all groups; P-Value, Probability value



Graph 1: Comparison of Mean, SD of Serum Lipid Profile in Between Controls, Oral Precancer, Oral Cancer and Overall is Done by ANOVA Test. TGL HDL and VLDL Showed Statistically Significance (p < 0.001). The CHO and LDL Showed no Significance.

IV. DISCUSSION

For a long time, it has been proposed that changes occur on the cell surface when a normal cell becomes cancerous. Malignant transformation of a cell has been linked to a number of cell-membrane alterations, including altered glycolipids, modifications to surface enzymes, and other phenotypic alterations. All biological processes, including the growth and division of cells in both healthy and malignant tissues, depend on cholesterol to remain structurally and functionally intact. Either high or low cholesterol levels might cause problems. Because of their connection to the prevalence of heart illnesses, the health risks associated with higher-than-normal levels have drawn a lot of public attention. On the other hand, the consequences of lower cholesterol levels are still unknown.

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Many malignancies, including head and neck tumours, have been linked to lower blood lipid levels **(Bryne M et al., 1992)**[8]

As well as oesophageal cancers **(Dabelsteen E et al., 1996)**.[9] The oral precancerous and cancer groups had significantly lower HDL levels than the control group, according to the findings and observations of the serum lipid profile assay used in this investigation (p value < 001). This is in line with research by Lohe VK et al., 2010)7, Patel PS et al., 1989)6, and Chawda et al., 2011)10, which discovered lower HDL levels in individuals with OSCC.

Patients with tumours may have lower serum HDL levels as a result of their illness, which is most likely caused by increased cholesterol use for the synthesis of new membranes and the buildup of esterified cholesterol in tumour cells. Patients who smoke and likely have lower levels of HDL, the good cholesterol, showed a reduction in mean serum HDL levels.

The minor increases in the other lipid profile markers, such as CHO, TGL, and VLDL, are not statistically significant. Studies by Patel et al. (1989)6, Lohe et al. (2010) 96, and Chawda et al. (2011) 10 that demonstrate a drop in the same do not support this. This can be due to the patients' genetic makeup and eating patterns. It is well established that eating habits, lipid intake, and absorption are all adversely affected by oral cancer. It follows that individuals with oral cancer should have low serum lipid levels; nevertheless, human plasma cholesterol levels are also regulated by the interaction of genes and hormones.

Alexopoulos et al. speculate that the carcinogenesis process may be to blame for the low cholesterol levels found in blood compartments and growing tissues. Low cholesterol was observed in cancer patients, but it was unclear if this was a result of the disease or its cause.

We observed significant variations in mean ($P < 0.001$) across groups in our study of precancerous patients, such as oral lichen planus and oral submucous fibrosis. The paired 't test' indicates a substantial drop in LDL and CHO levels in oral submucous fibrosis ($P \leq 0.001$). This is in line with a study by Nayak et al. (2010)11, who discovered that individuals with oral submucous disease had significantly lower HDL and CHO levels, with a mean of ($P < 0.001$). They also suggested that estimation of lipid profile can be considered as a good marker for increased turnover.

Serum lipid profile levels in precancerous and cancer patients can vary for a variety of factors, including age, body mass index, nutrition, exercise habits, and alcohol intake. Methological variations may possibly be the cause of the fluctuations in the five lipid parameters. As a result, alterations in lipid levels might be diagnostic in the early detection of oral cancer.

According to our findings, biomarkers such as HDL-cholesterol have demonstrated promise in the detection of individuals with oral precancerous lesions and oral cancer. Analysing these markers may also aid in the late-stage detection of metastatic cancer, for which HDL-cholesterol may offer practical biochemical indices for the clinical evaluation of the disease's invasiveness and dissemination in oral cavity malignancy. The malignancy is also indicated by the decreasing HDL levels. More case-controlled research on sizable populations utilizing these biomarkers will yield more empirical evidence that can be used to treat oral cancer.

V. CONCLUSION

In conclusion, the goal of the current study was to compare the serum lipid levels of patients with oral cancer and oral precancer to those of controls. After running the ANOVA test on the study's data, statistically significant findings regarding blood lipid levels in oral precancer and oral cancer were discovered.

HDL was found to be lower on lipid estimate, which is highly typical of all malignancies. To obtain additional scientific data that can aid in the early identification of oral precancer and oral cancer, a case control study including a broader and diverse population can be conducted on the combined usage of HDL, TGL, CHO LDL, and VLDL.

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