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ORIGINAL ARTICLE

LIPID PROFILE ESTIMATION IN PATIENTS WITH ORAL PREMALIGNANT DISORDERS, AND ORAL CANCER- A COMPARATIVE STUDY

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ABSTRACT:

Background: The changes in lipid profile have long been associated with cancer because lipids play a key role in maintenance of cell integrity. The present study evaluated alterations in serum lipid profile in untreated oral cancer patients and also in premalignant patients and compare with the controls. **Material and Methods**: The study included a total of 50 Patients who fulfilled the inclusion and exclusion criteria and who were further divided into 2 groups i.e. Habit associated premalignant and oral cancer group and control group. Serum lipid profile including Total cholesterol, HDL, LDL, VLDL and Triglyceride were analysed using semi automatic chem. 7 analyser. **Statistical Analysis**: Values of lipid profile obtained from the patients were evaluated statistically using one way ANNOVA and (unpaired) 't' test. **Results**: A significant decrease in serum total cholesterol and LDL levels were observed in cancer patients(P=0.047 and p=0.039) as well as significant increase in serum HDL levels were observed in patients with premalignant disorders(p=0.014) when compared with controls. **Conclusion**: The decrease in the levels of serum total cholesterol and LDL in oral cancer patients can be due to the reason that during carcinogenesis, there is greater utilization of lipids for new membrane biogenesis.

Key Words: Low density lipoproteins, High density lipoproteins, Very low density lipoproteins.

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INTRODUCTION

The use of tobacco and areca nut in various forms is very popular in our country and are the most important etiological factors for the development of many premalignant disorders like- leukoplakia, erythroplakia and oral submucous fibrosis and subsequently Oral cancer. Tobacco related mortality rate in India alone is among the highest in the world.[1]

Lipids are the major cell membranes components essential for various biological functions like cell growth and division. Tobacco carcinogens can induce generation of free radicals and reactive species, which are responsible for high rate of oxidation/ peroxidation of polyunsaturated fatty acids which can affect the cell membrane and might have involved in carcinogenesis. Due to the lipid peroxidation, there is a greater utilization of lipids including total cholesterol, lipoproteins and triglycerides for new membrane biogenesis. Thus lipid profile may be altered in patients with premalignant disorders and oral cancer and in patients with such habits.[2]

Changes in the circulatory lipid profile levels have been found to be associated in the aetiology of breast cancer and colorectal cancer.[3] However, only few reports are available on lipid profile in oral premalignant disorders, oral cancer and subjects with habit of tobacco. Thus the present study is designed to evaluate the serum lipid profile in patients with premalignant disorders associated with habit, oral cancer and compare with that of the controls.

MATERIALS AND METHODS

The study was undertaken in collaboration with the Medical OPD and Department of Oral Medicine and Radiology following the approval of the ethical committee of SGT University. The study comprised of patients reporting to the OPD of the SGT Medical Hospital for routine screening procedures of their individual needs and wherein when we encounter patients with oral findings who had existing lipid profile reports, they were further thoroughly examined in the Dental OPD between December 2014 to August 2016, around 400 patients were screened and a total of age and sex

matched 50 Patients who were untreated and were clinically confirmed were included in the study. Participants undergoing treatment for potentially malignant disorders and oral cancer or treated for the same and with systemic diseases were excluded. These patients were further divided into 2 groups namely.

- **Group I**: Subjects with habit related premalignant disorders or Oral cancer.(30 subjects)
  [For the ease of convenience **Group I** was further split into two:-
  - **I (A**) Premalignant (n=20)
  - **I (B)** Cancer (n=10)

- **Group II**: Subjects with no habit without lesion- control(20 subjects)

Once the values of lipid profile (CH, LDL, HDL, VLDL, TG) were obtained from patients they are noted on master chart and later evaluated statistically using one way ANNOVA and (unpaired)'t' test as described above**.** The results for all the tests, the confidence of interest was set at 95% with the "p" value of less than 0.005.

RESULTS

The study population comprised of 50 patients divided into 2 groups-

Group I (patients with premalignant and malignant disorders with Habit) consisting of 30 patients further divided into two Subgroups

- **Group I (A) Premalignant** [OSMF (10) and Leukoplakia (10)]
- **Group I (B) Malignant/Oral cancer** (10)

Group II (patients without lesion without habit) consisting of 20 patients. The obtained lipid profiles were compared among the groups.

Table 1: Age and Gender Distribution in Group I {I(A)+I(B)}

		Premalignant I(A)				Malignant (oral cancer) I (B)		Group II	
		OSMF		Leukoplakia					
		Male	Female	Male	Female	Male	Female	Male	Female
Age (In years)	< 30	7	0	1	0	0	0	3	0
	31-60	3	0	7	2	7	1	11	2
	61-90	0	0	0	0	1	1	3	1
Total		**10**	**0**	**8**	**2**	**8**	**2**	**14**	**3**

Table 2: Habit Distribution in Group I {premalignant group + oral cancer group}

Group I (A) (Premalignant)	Lesion	Bidi	Cigarette	Hookah	Betelnut	Gutka
	OSMF	0	0	0	5	5
	Leukoplakia	5	4	1	0	0
Group I(B) (Malignancy/Oral cancer)		7	3	0	0	0

Table 3: Mean serum values of subgroup 1A (premalignant) and group II (control) and their comparison

Lipid profile	Group	Mean	Std. Deviation	t-value	Sig.*
CH	I(A)	180.35	36.827	0.512	0.611
	II	173.00	52.531		
LDL	I(A)	100.80	30.080	-1.148	0.258
	II	113.08	37.273		
HDL	I(A)	54.61	11.523	2.562	0.014
	II	45.26	11.557		
VLDL	I(A)	47.56	53.415	1.427	0.162
	II	29.93	13.728		
TG	I(A)	190.60	163.080	1.174	0.248
	II	144.90	60.825		

*difference is significant if value <0.05

Table 4: Mean serum values of subgroup 1B (Oral cancer) and group II (control) and their comparison

Lipid profile	Group	Mean	Std. Deviation	t-value	Sig.*
CH	I(B)	162.60	74.307	-0.445	0.047
	II	173.00	52.531		
LDL	I(B)	93.18	50.112	-1.234	0.039
	II	113.08	37.273		
HDL	I(B)	48.01	12.644	0.617	0.542
	II	45.26	11.557		
VLDL	I(B)	33.48	21.227	0.533	0.598
	II	29.93	13.728		
TG	I(B)	157.30	112.949	0.394	0.697
	II	144.90	60.825		

*difference is significant if value <0.05

In the results, the age range of the present study was 19 to 85 years, in this 12 (24.00%) patients were in the age group of less than 30 years, 30 (60.00%) patients were in the age group of 30 to 60 years and 8 (16.00%) patients were in the age group of 60 to 90 years. The mean age in the present study was 45.15 years (35.9 in sub group IA and 62.8 in sub group IB) in Group I, and 48.3 in Group II. Upon gender wise distribution, there were total of 26 males and 4 females in group I, and total of 17 males and 3 females in group II. **(Table-1)**

In case of Oral submucus fibrosis, total number of patients were 10, betel nut (5), gutka (5), In case of leukoplakia, the total number of patients were 10, bidi (5), cigarette (4) and hookah (1). Incase of malignancy, the total number of patients were 10, bidi (7), cigarette (3), hookah 0. **(Table-2)**

When oral premalignant group was compared with the controls, the mean serum levels of HDL(p=0.014) was statistically higher in group I(A) when compared with group II. Also serum CH, VLDL and TG levels were also higher and serum LDL level was lower but all were statistically not significant**. (Table-3)**

When oral cancer group was compared with the controls, the mean serum levels of CH(p=0.047) and LDL(p=0.039) was statistically decreased in group I(B) when compared with group II. Also the mean serum levels of HDL, VLDL and TG were increased although they were statistically not significant. **(Table-4)**

DISCUSSION

Cell is the structural and functional unit of the living body. It is the basic unit of any tissue, which determines the type, the nature and function of the tissues. A group of different types of cells form tissues, and one or more types of tissues form organs. The cell membrane is composed of three substances that are proteins (55%), Lipids (45%) and Carbohydrates (5%).[3]

Lipids form an important constituent of these cell membranes, and cellular constituents, and are essential for various biological functions including cell growth & division. They are the main source of energy for the cell and they also act as key molecules for maintenance of membrane structure. Lipid anabolism and catabolism helps as a molecular integrator of energy homeostasis, membrane structure and function.[4]

The habit of tobacco chewing with betel quid, tobacco smoking and alcohol consumption are the most important etiological factors for the development of oral submucous fibrosis, leukoplakia and head and neck cancer. It is believed that tobacco carcinogens induce generation of free radicals and reactive oxygen species, which are responsible for high rate of oxidation/ peroxidation of polyunsaturated fatty acids. This peroxidation further releases peroxide radicals. This affects essential constituents of cell membrane and might be involved in carcinogenesis.[1]

In India, oral cancer (OC) is the leading cause of morbidity and mortality and may be preceded by clinically definable potentially malignant disorders. The lifetime risk for mortality from cancer in India for both males and females is 61%. According to statistics, the number of deaths in 2012 due to oral cancer is 36463 in males and 15361 in females that clearly indicates lack of awareness, education and probably even lack of resources for early detection especially in rural population[5]. As cancer is a multistep procedure which involves changes at micro and macro cellular levels lipids being one of them. The changes in lipid profile have long been associated with cancer because newly forming and fast proliferating malignant cells need many basic components such as lipids well above the normal physiological limits leading to diminished lipid stores. Cells fulfil these requirements from circulation, by synthesis through the metabolism or from degradation of major lipoprotein fractions like high density lipoprotein (HDL), low density lipoprotein (LDL) or very low density lipoprotein (VLDL).[6]

The mean age distribution of 50 patients among two groups were 44.87 yrs in Group I [sub Group I(A)-35.9 and sub group I(B)-62.8], and 48.30 in Group II. Gender wise distribution of 50 patients among three groups, there were 26 males and 4 females in Group I. In Group I, total number of Oral submucous fibrosis (OSMF) patients were 10, all of them were males, total number of leukoplakia patients were 10, out of them males were 8 and females were 2, total number of cancer patients were 10, out of them males were 8 and females were 2. In group II, 17 were males and 3 were females Thus, gender wise distribution shows male preponderance in all the groups which was in accordance to the study done by Aveu N et al. (2003)[7], Salonen L et al. (1990)[8] and Mehrotra R et al.(2010)[6]. The reason could be due to easy accessibility for males to use tobacco, areca nut and its products more frequently than females in our society and changing lifestyles of the youngsters.

When Group I was compared with Group II, there was statistically significant increased value of the serum HDL in pre malignant group when compared with group II, that was similar with the study of Dua S et al.(2011).[2] The possible mechanism for increase in the HDL levels was due to its scavenging effect on cholesterol levels and there was statistically significant decreased value of serum CH and LDL in cancer group when compared with group II which were similar to the study done by Kumar P et al. (2012)[9], Omoti et al. (2009)[10] and Kumar A et al.(2015)[11] this low level of cholesterol can be due to greater utilization of cholesterol in the proliferating tissues and blood compartments during the process of carcinogenesis and the possible reason for low level of LDL could be that it is more susceptible to oxidation in various pathologic conditions as a result its higher peroxidation takes place during oxidative stress when compared with other parameters.

Considering the observations of the present study, it is evident that there is inverse relation between serum cholesterol levels and cancer condition which was in accordance to the previous studies, This observation can

be explained on the basis that during progression of cancer there is greater utilization of lipids for new membrane biogenesis as it includes different lipoproteins containing apolipoproteins, which serve as ligands for specific receptors on cell membranes. Cellular uptake and regulation of cholesterol is mediated by these receptors located on the cell surface. Lipoproteins are then taken up and degraded by the cells which are needed for functioning of cells and simultaneously increased LDL receptors activity could be the possible factor for lower lipid values in patients with oral cancer.

Therefore from the study it could be concluded that alteration in lipid profile wards of changes which may be evident for prediction of conversion into oral cancer as previously stated. Further, studies with more number of cases with additional parameters with specific guidelines pertaining to the cases reporting to OPDs with habit of tobacco consumption or pre malignant disorders should be carried out so as to establish lipid profile as one of the investigative procedures to aid in early detection of oral cancer.

CONCLUSION

- There was no significant decrease in Serum CH, LDL, VLDL and TG in the premalignant group instead significant increase in the serum HDL was observed suggesting that hypolipidemia is a late change that occurs during carcinogenesis.
- Serum cholesterol and LDL were statistically lower in Oral cancer compared to controls which could be due to inverse relation between serum lipid profile and oral cancer as during carcinogenesis, there is greater utilization of lipids for new membrane biogenesis.

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