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LIPID PROFILE IN PATIENTS WITH ORAL CANCER

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ABSRACT

Present study deals to evaluate lipid profile in oral cancer patients and correlate their lipid profile with carcinogenesis. Lipid profile was analyzed in newly diagnosed histologically confirmed-100 cases of oral cancer received at the Department of Oral Pathology, LUMHS, from July, 2013 to July 2014. The age ranges of 31 to above 70 years with Male: female ratios 2.12:1 were included. Study was statistically analyzed by SPSS 17. Out of 100 cases 46 %, 22 %, 12 % and 5 % had malignancy of buccal mucosa, tongue, retromolar trigone and hard palate respectively. Lipid profile was reported as Serum TC in 87%, LDLC in 86%, HDLC in 68% of cases were lowered as compared to controls. There was an inverse relationship between the serum lipid profile and carcinogenesis. A significant association was reported between hypolipidemia and oral cancer.

Keywords: Lipid Profile, Oral Cancer, Hypolipidemia

INTRODUCTION

Carcinogenesis leads to various biochemical changes in the body. Lipids might be associated with cancers because they play a significant role in the maintenance of cellular integrity **[1, 2]**. Hypolipidemia predisposes or effect of cancer is still unclear. Furthermore, antineoplastic therapies also influence lipid profile **[3]**. Risks of carcinogenesis and

associated mortality has been reported with low serum cholesterol **[4]**.

This study was undertaken to evaluate the correlation of serum Lipid Profile, in oral cancer patients and their correlation with healthy subjects. In addition, Tobacco carcinogens generates reactive oxygen species and free radicals, which causes high rate of oxidation/peroxidation of polyunsaturated fatty acids, in addition releasing peroxide radicals, leading to carcinogenesis.[5,6] But the findings of changes in serum lipid profile in oral cancer patients strongly warrant an in depth research.

MATERIALS AND METHODS

The present case control study comprises of newly diagnosed histologically confirmed-100 oral cancer cases received in the Department of Oral Pathology, LUMHS, between July 2013 to July 2014 after taking written informed consent. Ethical clearance was taken from institutional ethical committee.

Controls: Age and sex matched subjects who do not have any renal, hepatic or cardiac dysfunction.

Patients having any cardiac, renal and hepatic dysfunction & those on chemotherapy and Radiotherapy were excluded.

Lipid profile tests were performed from fasting blood sample. The measurements were done by using kit method on Analyzer Clinical Chemistry. Lipid profile estimations included measurement of serum total cholesterol, HDL, LDL and triglycerides.

RESULTS

In present study age range of patients was 31 to above 70 years, with maximum no. of cases (72%) in age group 41-60 years. Male: female ratio was 2.12:1 (68:32). Out of 100 cases- 66 were of normal BMI, 24 were overweight & 10 % were obese. The BMI of cases and controls were almost same (BMI range: underweight-16-18.5, normal-18.5-25, overweight-25-30, obese->30). Out of 100 cases 46 %, 22 %, 12 % and 5 % had malignancy of buccal mucosa, tongue, retromolar trigone and hard palate respectively. Rest 15% of the cases had malignancies of alveolus, tonsillolingual sulcus, soft palate, lip, pyriform fossa and epiglottis. Maximum numbers (40 %) of cases were stage III of TNM classification. Lipid profile was reported as Serum TC in 87%, LDLC in 86%, HDLC in 68% of cases were lowered. Out of 100 cases 76% were tobacco chewers, 11% were alcoholic, 9% were smokers and 4% were not having any addiction. No significant alteration of lipid profile seen with addiction of tobacco.

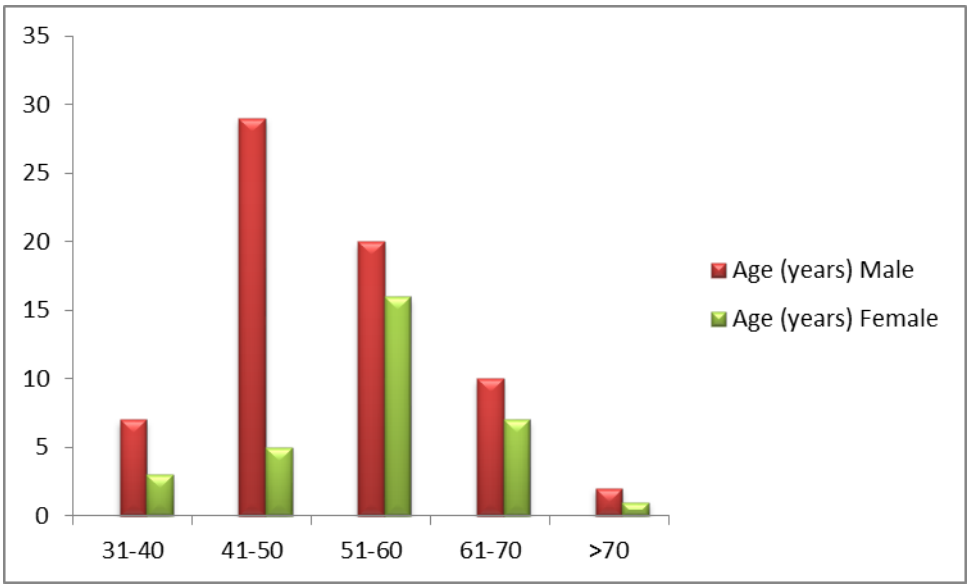

Figure 1: Age and Sex Wise Distribution of Subjects

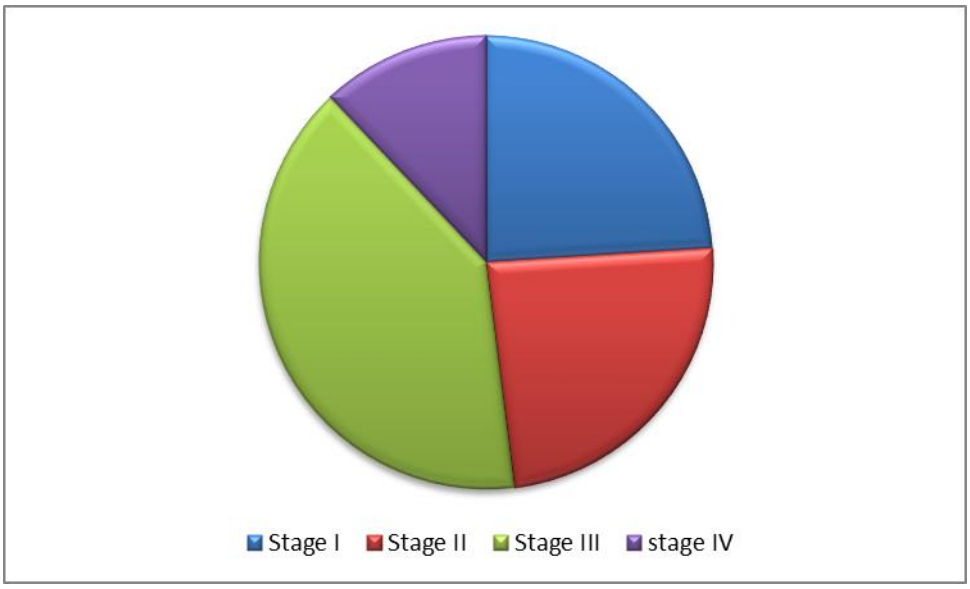

Figure 2: Showing Distribution of Cases According To TNM Staging

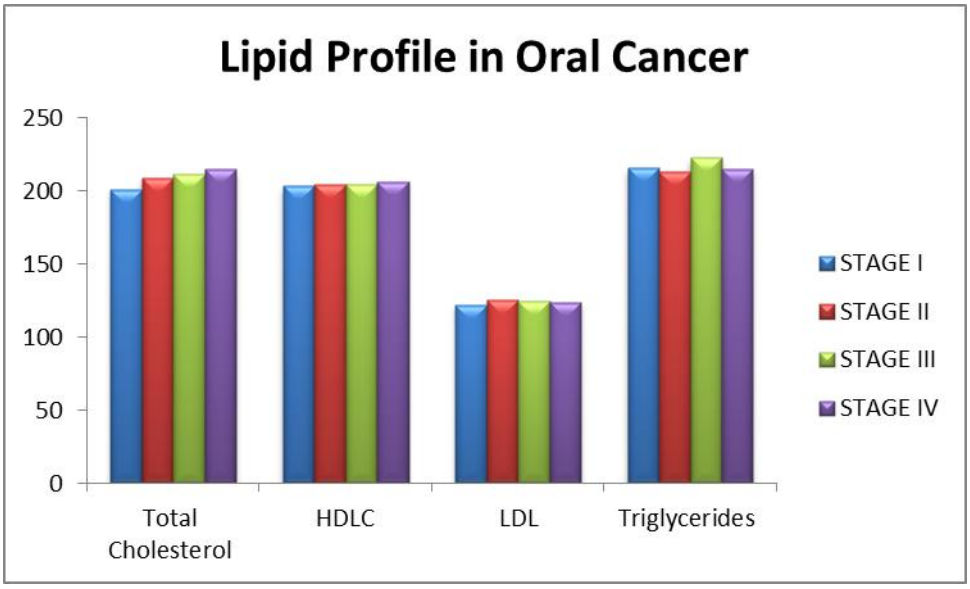

Figure 3: Showing Lipid Profile According To TNM Staging In Cases of Oral Cancer

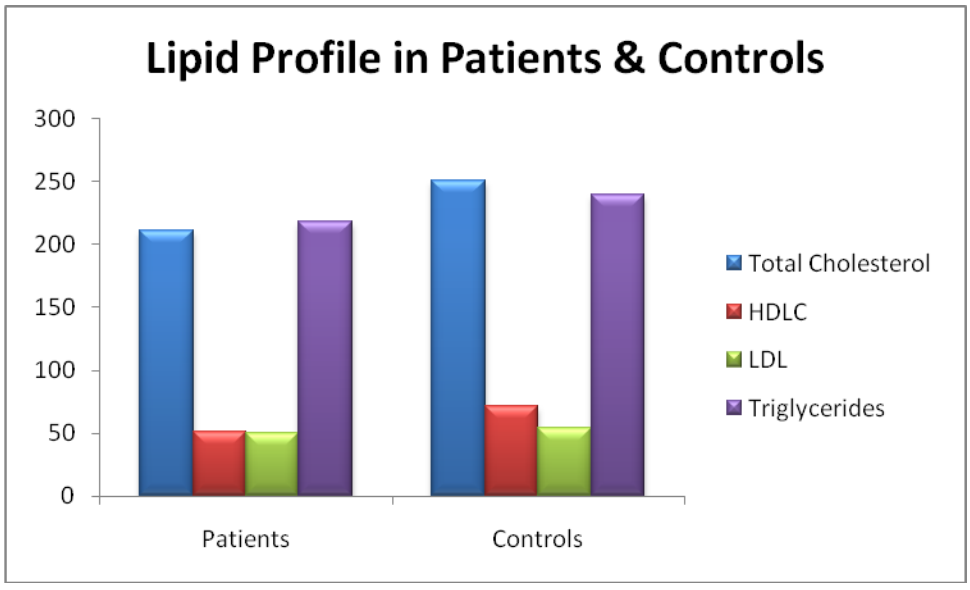

Figure 4: Showing Lipid Profile in Oral Cancer Patients & Controls

Table: 1 Showing Distribution of Cases

VARIABLES	STAGE I	STAGE II	STAGE III	STAGE IV
Total Cholesterol	**201.1±28.9**	**209.3±14.1**	**211.5±9.3**	**214.9±8.1**
HDLC	**203.2±12.1**	**204.8±24.2**	**204.4±9.3**	**206.5±11.3**
LDL	**122.3±7.27**	**125.6±3.2**	**124.9±8.41**	**124.1±6.4**
Triglycerides	**215.6±13.3**	**212.9±26.4**	**222.7±3.9**	**215.22.1**

Table 2: Showing Lipid Profile in Oral Cancer Patients & Controls

VARIABLES	PATIENTS	CONTROLS
Total Cholesterol	**211.3±6.5**	**250.7±11.6**
HDLC	**51.7±1.3**	**71.9±6.2**
LDL	**50.11±6.31**	**54.21±4.5**
Triglycerides	**218.33±8.3**	**239.5±5.6**

DISCUSSION

Oral cancer is highly prevalent disease in South East Asia in general and particularly it is one the most frequent problem in dental surgical sections of Sindh **[7]**. As in other studies[(8)], this study also reported oral cancer mainly effects old age groups particularly in fifth decade of life with higher males to females ratios **(Figure 1)** Studies have reported that, in Sindh, the oral cancer has very high incidence with high mortality **[9, 10]**. Lack of antioxidants and excessive production of peroxidation products of lipid have a vital role in carcinogenesis **[8]**. Disturbance in defensive action of antioxidants and peroxidation of lipid may be associated with curative and preventive chemotherapeutic treatment **[8]**.

Hyperlipidaemia a well documented risk factor for cardiovascular diseases, found highly prevalent in population of Sindh, but there was no data available to indicate hypolipidemia particularly in case of carcinogenesis. These facts insinuate us to investigate the serum lipid profile in oral cancer patients. This study emphasized correlation between changes in serum lipid profile carcinogenesis and, association of serum lipids with TNM staging of oral cancer. Hypolipidemia in general, particularly

decreased HDL, in case of oral cancer compared to controls may be under the influence of tobacco exposure, but this study did not highlight any significant difference between tobacco users and non tobacco users **[9, 10]**. Most cancers conditions were histopathologically diagnosed as epidermoid carcinoma and showed 68% were well-differentiated while remaining 32% were poorly differentiated epidermoid carcinoma. On the TNM Staging 40% were stage III cases. Although few studies reported incline in lipid profile in oral cancer. This study shown a significant decrease in serum lipid profiles in case of oral cancer compared to controls, specifically HDL was declined in case of cancer. In addition, no any significant difference in lipid profile level between different TNM Stages of oral cancer reported.

These biochemical changes may be observed because of lipid peroxidation that involves the oxidation of components of lipid bilayer of cellular structure. Lipid peroxidation, which may be provoked by tobacco, may play a key role in decreasing concentration of HDL and DNA damage in carcinogenesis **[10]**. Tobacco is a well known etiological factor of oral cancer **[11, 12]**. Studies showed that tobacco exposure enhance the production of free oxygen radicals and free nitrogen radicals through activation of inflammatory cascade or directly **[13, 14]**. Tobacco products induced production of reactive oxygen spices and free radicals, which increases the oxidation and peroxidation of unsaturated fatty acids.

Studies showed that either lack of defensive action of anti oxidants or overproduction can leads to lipid peroxidation, structural and functional change in protein, and DNA damage **[15]**. Free or reactive oxygen radicals and their generated lipid peroxidation products have been reported as significant role in carcinogenesis in oral cavity **[16]**. In addition peroxidation generates peroxide radicals which damage the cell membrane leads to carcinogenesis **[17]**. Formation or repair of cell membrane needs lipids which are supplied by the degradation or metabolism of major lipoproteins such as HDL, LDL, Vitamin C & E have defensive actions against peroxidation of lipid due to their antioxidant action **[18]**. Studies have reported lowered lipid profile in case of different cancers and their higher mortality rate **[19-22]**. Recycling of blood lipids keeps the lipid profile in normal range. In case of carcinogenesis change in lipid profile is more likely to be due to increased demand of lipid that utilize in biogenesis. It s unjustified that lowered lipid profile leads to carcinogenesis or produced as a result of it.

CONCLUSION

In the Present study of oral cancer shows inverse relationship between the serum lipid profile and carcinogenesis. Further studies should be carried out in large number of patients to confirm the role of these parameters with special attention to modifiable parameters in malignancies and their relation with staging and grading of cancer, which could be used as prognostic markers.

However, to what extent their dietary habits, nutritional status, and other physical activities led to these changes in lipids needs to be documented.

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