Research Article

Evaluation and analysis of abnormalities in serum lipid profile of oral cancer patients

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Abstract

Background: Oral cancer is the fifth most common cancer in the world with more than 3, 00,000 new cases diagnosed every year. Uncontrolled and excessive proliferation of cells in cancer needs many basic components like lipids well above the normal limits used in physiological process. Abnormalities in the level of lipids have been implicated as an important aspect of malignant transformation. Present study was carried out to evaluate changes in plasma lipid levels of patients with oral cancers. **Materials and Methods:** 50 age matched diagnosed cases of oral cancer and 50 healthy controls underwent detailed clinical history, physical examination and investigations. In this study, we measured and compared serum levels of lipid profile comprising of total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C) in both groups. **Results:** All the parameters of lipid profile were decreased in cancer group. This decrease was statistically significant when compared with control group. **Conclusion:** Our study points towards hypolipidemia in patients of oral cancer. Whether hypolipidemia at the time of diagnosis is a causative factor or is a result of the cancer remains unanswered.

Keywords: serum lipid profile, oral cancer.

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INTRODUCTION

Cancer is one of the five main causes of death in all societies, its relative position varying with age and sex[1]. Oral cancer is the fifth most common cancer in the world with more than 3, 00,000 new cases diagnosed every year[2]. In India it ranks first among all types of cancers in male patients[3]. The development of oral cancer is associated with chronic irritating factors such as tobacco, smoking, alcohol and betel nut; tobacco and betel nut being major factors in India[4]. The development of this malignancy as any other would require the uncontrolled and excessive proliferation of cells which would need many basic components well above the normal limits used in physiological process. One such component is lipid which forms major cell membrane components essential for various biological functions including cell division and growth of tissues. The increased requirement of lipids to fulfill the need of these new cells would be expected to diminish the lipid stores of the body. Abnormalities in the level of lipids have been implicated as an important aspect of malignant transformation and several authors have proposed that hypocholesterelomia is a predisposing factor for cancer development[5,6,7,8]. The present study was carried out to evaluate changes in plasma lipid levels of patients with oral cancers and to examine if any relation exists between the changes and increasing malignant potential.

MATERIALS AND METHODS

The present study was undertaken in the Department of Biochemistry, Government Medical College, Latur. The period of study was from January 2014 to August 2015. A group of fifty patients between age group of 21-60 years consisting of those diagnosed with oral cancer and

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confirmed by biopsy from indoor wards and OPD of the surgery department of Government Medical College, Latur was included in a cross sectional study. Fifty age and sex matched healthy controls were also included. Written informed consent was taken from the participants. Each patient underwent detailed clinical history, physical examination and investigations. In this study, we measured serum levels of lipid profile comprising of total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C). Fasting sample of 5 ml blood was collected in clean plain bulb after an overnight fast. (i.e. after 12 hours). The serum was separated and serum lipid profile was estimated on the same day.

Measurement of Serum Cholesterol

It was measured by using Autozyme new cholesterol enzymatic kit, manufactured by Accurex Biomedical private limited, Thane, India.

Measurement of HDL cholesterol

It was measured by using Autozyme HDL cholesterol enzymatic kit, manufactured by Accurex Biomedical private limited, Thane, India.

Measurement of Serum triglyceride

It was measured by using new triglyceride enzymatic kit manufactured by Transasia Biomedical private limited, Daman, India.

Measurement of Estimation of LDL

Following formulae were used:

$$\text{Srum VLDL} = \frac{\text{Serum TG}}{5}$$

Serum LDL = Serum total cholesterol - (serum VLDL + Serum HDL)

Statistical Analysis

Statistical data was recorded on Microsoft Excel program. Data was analyzed using primer software. The values were quoted in the form of mean ± standard error of mean wherever required. Data was compared using repeated measures one way ANOVA. The p value ($p< 0.05$) is considered as significant and the p value ($p< 0.001$) is considered as highly significant.

RESULTS

Table 1: Age wise distribution of cases and controls

Age in (Yrs)	Control (N)	Control (%)	Oral Cancer Cases (N)	Oral Cancer Cases (%)
21-30	5	10%	5	10%
31-40	10	20%	9	18%
41-50	17	34%	17	34%
51-60	18	36%	19	38%

N = Number of Cases

The above table shows age distribution in cases of oral cancer and controls. Most of subjects in control group (72%) and oral cancer (72%) were between the age of 41-60 years. The mean age of subjects in control group was 45.64 ± 9.06 years while that of patients with oral cancer was 45.98 ± 9.48.

Table 2: Sex Wise Distribution of Cases and Controls

Sex	Control (N)	Control (%)	Oral Cancer Cases (N)	Oral Cancer Cases (%)
M	30	60%	31	62%
F	20	40%	19	38%

N= Number of Cases

The above table shows sex wise distribution of cases and controls. The number of males in cases of oral cancer was 31 (62%) while in controls it was 30 (60%). The number of females in cases of oral cancer was 19 (38%) and while in controls it was 20 (40%).

Table 3: Comparison of lipid profiles among control and oral cancer group

Variables	Control [Mean ±S.D.]	Oral Cancer Group [Mean ±S.D.]
Total Cholesterol	177.14 ± 13.93	145.36 ± 10.06[a.]
Trigycerides	113.54 ± 11.58	106.50 ± 7.92[a]
LDL	112.66 ± 14.93	95.16 ± 10.80[a]
VIDL	23.62 ± 2.40	21.18 ± 1.61[a]
HDL	41.72 ± 6.18	28.76 ± 4.37[a]

N = 50 in each group. [a]p<0.01 when compared to control

Table no. V shows comparison among lipid profiles of control and oral cancer group. When the total cholesterol of cases of cancer(145.36 ± 10.06) was compared with that of the controls(177.14 ± 13.93), it was found to be less than that of controls and the difference was statistically highly significant ($p < 0.01$). The triglyceride level in cancer patients (106.50 ± 7.92) was markedly reduced when compared with controls (113.54 ± 11.58) and the difference was statistically highly significant ($p < 0.01$). When the LDL of cancer patients (95.16 ± 10.80) was compared with that of the controls (112.66 ± 14.93), it was found to be less than that of controls and the difference was statistically highly significant. ($p < 0.01$) The VLDL in cancer group (21.18 ± 1.61) was decreased when compared with control group (23.62 ± 2.40) with the difference being statistically highly significant. ($p < 0.01$). Similarly, the HDL in cancer group (28.76 ± 4.37) was decreased when compared with control group (41.72 ± 6.18) and the difference was statistically highly significant. ($p < 0.01$).

DISCUSSION

In this study, we estimated the lipid profile in 50 patients each of oral cancer and compared them with 50 age and

sex matched controls. The mean age of subjects in control group was 45.64 ± 9.06 years, while the mean age of patients with oral cancer was 45.98 ± 9.48. Most of the patients in the cancer as well as control group were of the age between 41-60 years. In our study, we observed that the total cholesterol and LDL cholesterol in cancer (145.36 ± 10.06 and 95.16 ± 10.80 respectively) was evidently decreased when compared with that of the controls (177.14 ± 13.93 and 112.66 ± 14.93 respectively).The difference was statistically highly significant ($p<0.01$). The triglycerides of cancer patients (106.50 ± 7.92) were reduced in comparison of the controls (113.54 ± 11.58) where the difference was statistically highly significant ($p<0.01$). The VLDL and HDL in cancer group (21.18 ±1.61 and 28.76 ± 4.37 respectively) was found to be decreased when compared with the controls (23.62 ± 2.40 and 41.72 ± 6.18 respectively) and the difference was statistically highly significant ($p<0.01$) in both these cases. Three main competing hypotheses can explain the inverse association between cholesterol concentrations and the incidence of cancer. Firstly, lower cholesterol values, even before the manifestation or detection of cancer, may be a result of the cancer process. Secondly, lower cholesterol values may precede the development of cancer but the association with cancer is secondary, i.e. cholesterol serves as a marker for some other causal set of variables. Thirdly, lower cholesterol values may precede the development of cancer and may be causally associated with the occurrence of some forms of cancer.[9] The "preclinical cancer effect" hypothesis that is, the metabolic depression of serum cholesterol by undiagnosed malignant neoplasms has received considerable attention as an explanation for the observed inverse association.[10,11] Malignant neoplasms are known to have protean physiological effects, which might include depression of blood cholesterol. In this regard, leukemic blood and bone marrow cells have been shown recently to have an elevated low density lipoprotein-receptor activity that correlated inversely with plasma cholesterol concentration.[12] Patients of leukemia with both a high LDL-receptor activity per cell and a high white-blood-cell count had the lowest cholesterol concentrations. During chemotherapy, cholesterol levels rose concomitantly with the disappearance from the peripheral blood of leukemic cells. Hypercholesterolemia in leukemia and other neoplastic disorders may be due to increased LDL-receptor activity in the malignant cells. It is suggested that this high uptake and degradation of LDL by malignant cells could be utilized to target neoplastic cells with LDL-bound chemotherapeutic agents.[12] Studies have found increased LDL catabolism in patients with prostate cancer, and some malignant cells have been shown to have increased LDL receptor activity.[13,14,15] The physiological reasons for the decrease are a matter requiring further investigation. A host response to a progressing tumor mediated by a substance such as tumor necrosis factor may be involved.[16,17] Physiological responses to tumor necrosis factor include anorexia, cachexia, and disturbances in lipid metabolism.[16] Two serum growth factors, human monocytic colony-stimulating factor and human granulocyte-macrophage colony-stimulating factor, have both been shown to lower blood cholesterol.[18,19] From this study, it was evident that there are definite underlying biochemical alterations in serum of patients with oral cancer when compared with the serum levels of patients apparently free of any disease process that is, healthy control groups. Thus, the identification and characterization of more enzymes involved in these pathways will be more important for the proper understanding of these alterations. It will assist researchers in the near future to identify the genes and enzymes of lipid metabolic pathways. The change in lipid levels may have a diagnostic or prognostic role in the early diagnosis and treatment of oral malignant lesions. Further studies along these lines may be useful for the prediction of prognosis and response to cancer treatment.

CONCLUSION

In conclusion our study points towards hypolipidemia in patients of oral cancer. Whether hypolipidemia at the time of diagnosis, is a causative factor or is a result of the cancer remains unanswered. Lower plasma lipid status however, may be a useful marker in the early neoplastic changes.

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