



## RESEARCH ARTICLE

### TO EVALUATE SERUM LIPID LEVELS IN ORAL CANCER: A PILOT STUDY

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#### ABSTRACT

**Introduction:** Lipids are the major cell membrane components essential for cell growth and division and the maintenance of cell integrity of normal and malignant tissues. Alterations in serum lipid profile have long been associated with malignancies.

**Aim:** The aim of this study was to estimate the serum lipid profile in oral cancer patients and to study the relationship and clinical significance of serum lipid level and risk of oral cancer.

**Material and Methods:** In this study total of 30 cases were taken, out of which 20 were of oral cancer and 10 were controls. Fasting lipid profile including cholesterol(C), triglycerides (TGL), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were analysed.

**Results:** Decrease levels of C, TGL, HDL, and LDL were observed in patients with oral cancer as compare to controls. VLDL values as such were not significant.

**Conclusion:** An inverse relationship was found between serum lipid profile and oral cancer which may have an early diagnostic or prognostic role in oral cancer.

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## INTRODUCTION

Oral cancer is the sixth most common cancer in the world. Head & neck cancer accounts for 30-40% of all malignant tumor in India and the most common malignant counterpart is oral SCC which is the tenth most common cause of death. By the time it is diagnosed oral cancer has already spread and therefore proves to be fatal. So early detection of oral cancer can improve treatment modalities and hence the prognosis (Day *et al.*, 2003). Development of oral cancer is a complex mechanism comprising of proliferation, apoptosis, and differentiation and interrelationship between these processes resulting in tumor development and progression. Newly uncontrolled proliferating tumor cells would require many basic components above the normal levels which are used in physiological process (Day *et al.*, 2003). One such component is lipid which is a major cell membrane component essential for various biological functions including cell division and growth of normal and malignant tissues. Storage of lipids decreases due to its increase use by the rapidly dividing cells (Chawla *et al.*, 2011; Gupta and Gupta, 2011). The present study was mainly undertaken to estimate, analyse and compare the levels of plasma total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL) and

triglycerides (TG) in patients with oral cancer and normal controls. Saliva, urine, blood based tests are more appealing because of its ease, economic reasons, advantages and possibility to repeat sampling and thus can be used for early diagnosis, predicting prognosis and monitoring the progression of disease (Lohe *et al.*, 2010).

## MATERIALS AND METHODS

A total of 30 patients were selected in the age range of 30-65 years. 20 patients were histopathologically confirmed as oral SCC. 10 healthy patients as normal control with no systemic disease were selected. Out of 20 patients selected 12 were of well differentiated SCC, 5 were moderately differentiated SCC and 3 were poorly differentiated SCC. 2 ml of fasting blood samples were collected from each subject, drawn from antecubital vein under aseptic conditions. Serum was collected after centrifugation and stored at 80°C until analysed. Lipid analysis was done in a fully automated analyser based on spectrophotometric principle using kits obtained from ERBA diagnostics. Written consent was taken from the patients. Detailed history including frequency, duration of habits, any signs and symptoms, histopathology and lipid profile estimation was recorded in all patients.

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## RESULTS

The study comprised of 30 subjects. They were divided into two groups. In Group I total number of patients were 20 (oral SCC) out of which 18 were males and 2 were females. Group II which was control group comprised of 10 patients out of whom **6 were males** and 4 were females. The Mean value, standard deviation and F and P values of all lipid values in oral cancer patients and controls were calculated and then compared.

**Table 1. Comparison between serum LDL levels (Group I) and controls (Group II)**

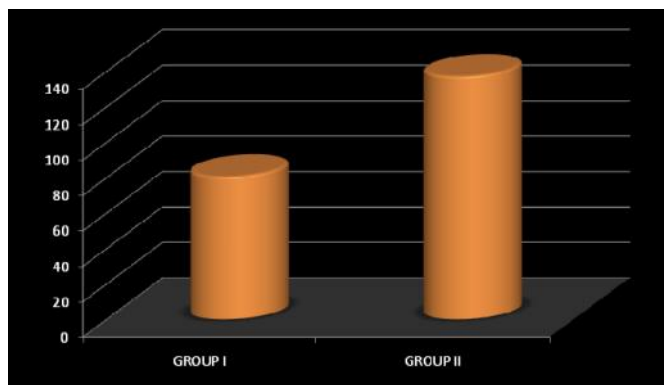
Groups	Mean	Standard deviation	F- value	P-value	Significance
Group I	80.53	3.917			
Group II	137.1	2.447	2.483	0.025	Significant

**Table 2. Comparison between serum HDL levels (Group I) and controls (Group II)**

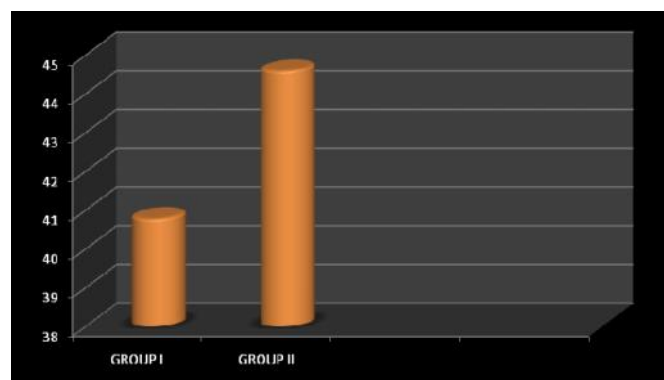
Groups	Mean	Standard deviation	F- value	P-value	Significance
Group I	40.78	1.607			
Group II	44.59	3.158	0.253	0.043	Significant

**Table 3. Comparison between serum TC levels (Group I) and controls (Group II)**

Groups	Mean	Standard deviation	F- value	P-value	Significance
Group I	138.94	6.686			
Group II	184.66	2.497	10.058	0.030	Significant



**Graph I. Comparison between serum LDL levels (Group I) and controls (Group II)**

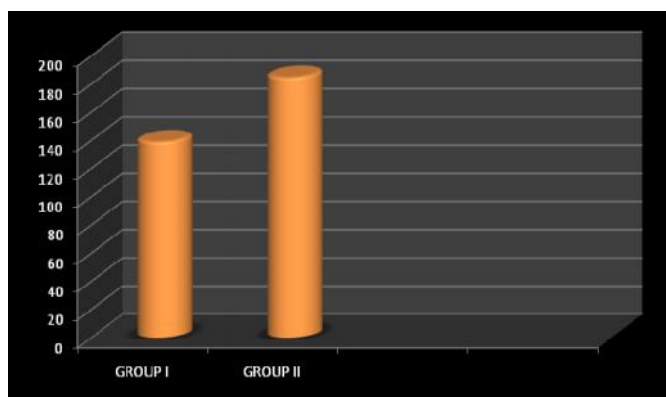


**Graph II. Comparison between serum HDL levels (Group I) and controls (Group II)**

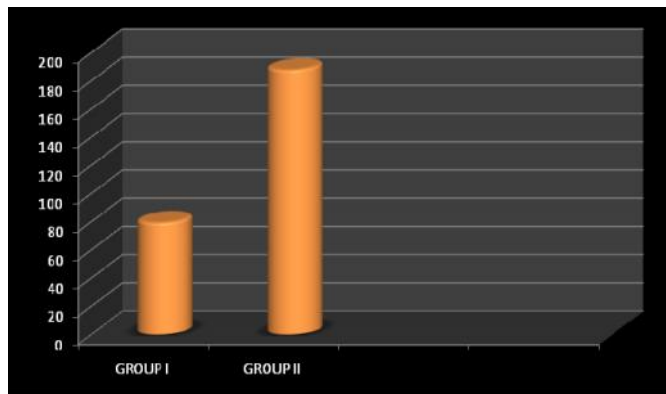
**Table 4. Comparison between serum TGL levels (Group I) and controls (Group II)**

Groups	Mean	Standard deviation	F- value	P-value	Significance
Group I	79.17	13.493			
Group II	187.71	3.224	1.557	0.011	Significant

There was a significantly lower level of mean serum LDL  $80.53 \pm 3.917$  (Table 1), HDL  $40.78 \pm 1.607$  (Table 2), TC  $138.94 \pm 6.686$  (Table 3) and TGL  $79.17 \pm 13.493$  (Table IV) found in all groups of oral cancer patients as compared to the controls LDL  $137.1 \pm 2.447$  (Table 1), HDL  $44.59 \pm 3.158$  (Table 2), TC  $184.66 \pm 2.497$  (Table 3) and TGL  $187.71 \pm 3.224$  (Table 4). *P* values were suggested to be significant in oral cancer patient as compared to controls ( $P < 0.05$  is significant)



**Graph III. Comparison between serum TC levels (Group I) and controls (Group II)**



**Graph IV. Comparison between serum TGL levels (Group I) and controls (Group II)**

## DISCUSSION

Lipids are high energy yielding molecules which include oils, waxes, fats, phospholipids and steroids. Fats and oils are made from two kinds of molecules: one glycerol and three fatty acids joined by dehydration synthesis known as triglycerides which are the major form of energy storage. For transport in plasma, triglycerides and cholesterol are packaged into lipoproteins, which are then taken up and degraded by cells to fulfill the demands for cellular functions.<sup>5</sup> Lipids in malignant tumors are not only necessary for providing the membrane constituents of proliferating cells but are also needed for energetic, biophysical and signaling pathways that leads to tumorigenesis. Dysregulated lipid metabolism is a hallmark of cancer and possible role of lipid in the etiology and prognosis

of cancer has been studied (Nomura and Cravatt, 2013; Nayak *et al.*, 2010). Lipids stores are decreased due to increased use of lipids by the malignant cells which can be beneficial for the early diagnosis of oral cancer. There are three main hypotheses to explain the inverse association between cholesterol concentrations and the incidence of cancer. Firstly, lower cholesterol values, even before the 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 (Kark *et al.*, 1982). Earlier studies have reported that low lipid levels may be due to the direct lipid lowering effect of tumor cells or some secondary malfunction of the lipid metabolism or secondary to antioxidant vitamins. An inverse relationship was found between serum cholesterol level and risk of cancer and further provided a basis for epidemiological research. This U or J shaped relationship indicates a higher mortality rate in both high and low serum cholesterol concentrations (Rose and Shipley, 1980). The habit of tobacco consumption is a well known etiologic factor in the pathogenesis of oral cancer. Tobacco is a carcinogen which induces generation of free radicals and reactive oxygen species and are responsible for oxidation and peroxidation of polyunsaturated fatty acids. Lipid peroxidation releases peroxide radicals mainly hydroxyl radical which leads to destruction of tissue by initiation and propagation of lipid peroxidation by detaching hydrogen from unsaturated fatty acids (Choi *et al.*, 1999). This affects essential constituents of the cell membrane and might be involved in carcinogenesis. Hypolipidemia may also result due to the direct lipid lowering effect of tumor cells or some secondary malfunction of the lipid metabolism or secondary to antioxidant vitamins (Gupta and Ray, 2003; Bose *et al.*, 2012). In the present study, a significant decrease in serum LDL, HDL, TC and TG levels were observed in oral cancer patients as compared to controls. The results of the present study also support that blood can be used as a non invasive diagnostic tool in assessing lipid profile levels in oral cancer and to support this concept are the findings of the earlier studies (Schatzkin *et al.*, 1987).

## Conclusion

Alteration in lipid levels may have a diagnostic or prognostic role in the early diagnosis or prognostication for oral cancer. Lower levels may act as a useful indicator for initial changes in neoplastic cells. Early detection is also secondary prevention leading to increased survival and reduced mortality. Result of the present study also support that blood based test can be used as a non invasive diagnostic tool in assessing lipid profiles in oral cancer patients.

Before a blood based diagnostic test can replace a more conventional one, its diagnostic value has to be determined in terms of sensitivity, specificity, correlation with established disease diagnostic criteria and reproducibility. However, a detailed study of cholesterol carrying lipoprotein transport and the efficiency of the receptor system may help in understanding the underlying mechanisms of regulation of plasma cholesterol concentrations in cancer.

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