Lipid profile level in oral cancer and its significance on histological grade

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**Abstract**

**Introduction:** Oral cancer is one of the commonest cancer in India. Its early detection can prevent morbidity and mortality. Aim of this study is to estimate the lipid profile level in oral cancer and its significance on histopathological grade.

**Material and method:** 70 patients with established oral squamous cell carcinoma were taken in the study and their lipid profile analysed.

**Result:** Lipid profile had an inverse relationship with oral cancer with no significant relation with histopathological grade.

**Conclusion:** Lipid profile estimation is a cost effective method to detect oral cancer.

**Key Words:** Lipid Profile, Oral Squamous cell Carcinoma.

**Introduction**

Oral cancer is the sixth most common cancer in the world [1]. According to cancer registry data in India it is the no.1 cancer among men & no.3 among women. Early detection of cancer leads to improved treatment & better prognosis [2]. Blood based tests are easy to perform, economical & repeated samples can be tested [3]. Malignancy occurs due to uncontrolled proliferations of cells or decreased apoptosis [3]. Lipid is an essential component for the cell membrane, so the lipid level falls due to rapidly dividing cells [4, 5].

In the last few decades lipid levels & coronary disease association has been established. Researchers have also observed an inverse relationship between serum lipid level & cancer. Few studies have been done to find lipid level in carcinoma pharynx, carcinoma lungs, leukemia, breast cancer & oral cancer. Squamous cell carcinoma is the most common oral cancer & has been categorized by Broder as well differentiated, moderately differentiated & poorly differentiated. This study aims to see the lipid profile in the oral cancer mainly squamous cell carcinoma & its significant with different histological grade.

**Material and Method**

70 patients with of oral squamous cell carcinoma were studied in a Patna Medical College and Hospital. This was a prospective study. Known case of Hypertension, hypercholesterolemia and Diabetes mellites were excluded from the study. All the other patients who were histopathologically confirmed as oral Squamous cell carcinoma were included in the study. The patients were categorized into well differentiated, moderately differentiated and poorly differentiated squamous cell carcinoma. Written informed consent was taken from these patients. Blood samples of these patients were taken and their serum lipid profile was done by Merck semi autoanalyser using reagent from Merck. Cholesterol was calculated by enzymatic colorimetric method (end point reaction) by taking 1.0 mL R1 monoreagent to it 10 uL sample taken. In another test tube 1.0 mL R1 monoreagent and 10 uL standard samples mixed and incubated for 5 minutes at 37°C. Samples read at 500 nm in absorbance mode.

Triglyceride was also calculated just like cholesterol with triglyceride monoreagent and triglyceride standard. HDL was calculated by the following method.0.5ml serum added with 0.5ml HDL precipitating reagent and centrifused at 4000 r.p.m for 10 minutes. Then 0.05 ml
supernatant is taken and 1.0 ml cholesterol working enzyme added. In another test tube 0.05ml standard and 1.0 ml cholesterol working enzyme added. This was incubated for 10 minutes at 37°C and absorbance is measured at 510nm.

Calculation- HDL-Cholesterol in mg% = Absorbance of sample/absorbance of standard *100
VLDL and LDL were calculated using the following formula. VLDL=TG/5 LDL=TC-VLDL-LDL

Result

Out of the 70 cases 42 were male and 27 were female. Age range was from 24 years to 72 years with mean age of 52 years. All the cases had history of tobacco intake either in form of quid or smoking. Out of the 70 cases: 28 were well differentiated; 26 moderately differentiated and 16 poorly differentiated Squamous cell carcinoma.

Table 1: Showing lipid profile in different grades of cancer.

| Type       | No of cases | Total Cholesterol(TC) | High density lipoprotein(HDL) | Low density lipoprotein(LDL) | Very low density lipoprotein(VLDL) | Triglyceride (TG) |
|------------|-------------|------------------------|------------------------------|-------------------------------|-----------------------------------|-------------------|
| W.D.       | 28          | 138.2±10.28            | 26.01±4.16                   | 85.35±14.16                   | 15.21±7.95                       | 79.16±12.12       |
| M.D.       | 26          | 129.3±6.25             | 27.31±2.71                   | 78.42±12.24                   | 16.16±3.31                       | 78.15±13.17       |
| P.D.       | 16          | 122.0±8.29             | 24.23±3.95                   | 80.36±8.71                    | 18.28±5.12                       | 80.34±14.21       |

W.D.=Well Differentiated, M.D.=Moderately Differentiated, P.D.=Poorly Differentiated.

Discussion

Oral cancer is the commonest cancer among males in India due to the habit of taking tobacco in the form of quid and smoke. Most common among oral cancer is squamous cell carcinoma. Our study also had a male preponderance similar to study by Singh S et al[9] and Kumar P et al[3]. Oral cancer occurs mainly in older age group. Our study had a mean age of 52 years similar to Singh S (51.07 years), Kumar P (4th to 5th decade)[3,9]. Our study showed lowering of all the lipid parameters. This lowering was not according to the grade of cancer.

Table 2: Showing comparative analysis of lipid profile by different writers.

| Lipid profile | Singh et al [9] | Kumar et al [3] | Present |
|---------------|-----------------|-----------------|---------|
|               | W.D.            | M.D.            | P.D.    | W.D. | M.D. | P.D. |
| Total cholesterol | 175.64          | 179.93          | 179.00  | 131.43 | 116.5 | 114.00 | 138.2 | 129.3 | 122.0 |
| HDL           | 40.43           | 41.63           | 38.78   | 31.12 | 31.54 | 25.35 | 26.01 | 27.31 | 24.23 |
| LDLC          | 127.21          | 113.37          | 112.11  | 109.36 | 105.57 | 111.00 | 85.55 | 78.42 | 80.36 |
| VLDL          | 23.86           | 23.41           | 22.11   | 27.02 | 17.91 | 24.1  | 15.21 | 16.16 | 18.28 |
| TG            | 102.50          | 101.96          | 94.56   | 116.67 | 99.14  | 121.5  | 79.16 | 78.15 | 80.34 |

W.D.=Well Differentiated, M.D.=Moderately Differentiated, P.D.=Poorly Differentiated. HDL-High Density Lipoprotein, LDLC-Low density lipoprotein, VLDL-Very low Density Lipoprotein, TG-Triglyceride.

Singh S et al also showed lower lipid profile levels [9]. Kumar P also showed inverse relationship of lipid profile with carcinoma [3]. But Bailward showed triglyceride level to be variable, HDL lowered, LDL and VLDL levels were non significant [10]. Kumar P et al and Singh et al showed lowered lipid level which had no correlation with grading[3][9]. This was similar to the present study. Rose et al showed 66% high mortality rate in cancer with low cholesterol [7]. This fall in lipid profile may be due to the rapidly proliferating cancer cells which need lipid for its cell membrane. Another theory is that tobacco, which contains nicotine is a proven carcinogenic agent in animal studies, causes production of free radical and reactive oxygen species that cause peroxidation of polyunsaturated fatty acid(PUFA) leading to carcinogenesis, thus fall in lipid level[11, 12]. Cholesterol and triglyceride are important constituents of cell and carries out many important physiological functions. Cholesterol maintains the structure and function of cell membrane. It also helps in activity of membrane bound enzyme and also stabilizes DNA helix [13]. Cholesterol is in our tissue and plasma.
as free cholesterol or in combination with long chain fatty acid, as cholesterol ester. Cholesterol is the precursor of mainly steroids in our body example corticosteroid, sex hormone, bile acid and vitamin D. Cholesterol is an constituent of lipoprotein fraction like LDL, HDL and VLDL.

Cholesterol is amphipathic lipid and an essential structure component of membrane and plasma lipoprotein. Lipoprotein transports free cholesterol and readily equilibrates with cholesterol in membranes. Storage form of cholesterol is cholesterol ester. LDL is the mediator of cholesterol and cholesterol ester uptake in many tissue [14].

In cancer, blood cholesterol undergoes early and significant change. This change is due to cancer or its effect is not known. Some studies have shown that tumor cells have direct lipid lowering effect or there is some secondary dysfunction of lipid metabolism or secondary to antioxidant vitamin.[15,16,17]. Previous reports have shown that low HDL is a predictor of cancer [18,19,20] and this may be due to increase utilization of cholesterol for membrane biosynthesis.

Double bond fatty acid are susceptible to oxidation. Fatty oxidation converts fatty acid to hydroperoxides which decompose to form ketoacid and hydroxyketoacid. This causes destruction of fat soluble vitamins in food. Oxidative damage also occurs in membrane phospholipid damaging cell membrane and also membrane protein. Lipid peroxidation occurs by hydrogen peroxide which is formed by Haber Weiss reaction or Fenton reaction [21].

Tobacco carcinogens produces reactive oxygen species which causes lipid damage and generates lipid peroxides [9]. This process affect essential constituents of cell membranes and may result in tumorigenesis.[22][23].

Vitamin E is co-transported with cholesterol and prevent lipid peroxidation.[24]. It has been observed that fat soluble antioxidant vitamin such as vitamin E and beta carotene decreases in tobacco users[25] as fatty acid oxidation destroys fat soluble vitamin. So there is a decrease in plasma level of total cholesterol, HDL, LDL, VLDL and Triglyceride.

So the fall in lipid can be a cause or a result of cancer is still debatable.

**Conclusion**

In our condition where poverty is high and oral cancer continues to rise, lipid profile estimation can be easy, affordable method to detect cancer early. This avenue needs to researched further so as to find the cause of fall in serum lipid level which can be a cause leading to carcinogenesis or a result of cancer.

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