Original Article

International Journal of Dental and Health Sciences
Volume 02,Issue 01

RELATIONSHIP BETWEEN SERUM LIPID PROFILE AND ORAL SQUAMOUS CELL CARCINOMA

Kumar Ashutosh[1]
1.Postgraduate student,Department of E.N.T,G.S.V.M Medical College, Kanpur

ABSTRACT:

Background: Alteration in the plasma lipid profile has been associated with a variety of cancers and precancerous conditions of oral cavity. Low levels of lipids serves as a marker and prognostic indicator in the early detection of oral precancerous and cancerous conditions.

Aim: The present study aimed to evaluate the alteration in plasma lipid profile in oral squamous cell carcinoma (SCC) patients.

Materials And Methods: A total of 120 patients were included in the study, 60 with Oral SCC and 60 healthy controls. Fasting plasma lipid profile including Total Cholesterol (TC), Very Low Density Lipoproteins (VLDL), Low Density Lipoproteins (LDL), High Density Lipoproteins (HDL) and Tri-Glycerides (TG) were measured using automatic analyser. The data obtained were analysed using independent sample 't' test.

Results: There was statistically significant decrease in plasma total cholesterol, TG, LDL, VLDL and HDL observed in patients with Oral SCC as compared to the control group.

Conclusion: The low lipid levels associated with Oral SCC indicates that there is inverse relationship between oral cancer and serum lipid profile. Decrease in the lipid levels may be considered as a useful biochemical marker in the early diagnosis of oral malignancy.

Key Words: oral squamous cell carcinoma, lipid, biochemical marker

INTRODUCTION:

Oral cancer most commonly occurs in middle-aged and older individuals, although a disturbing number of these malignancies is also being documented in younger adults in recent years.[1-3] Oral cancer is one of the most prevalent cancers and is the tenth most common causes of death.[4] Tobacco in the form of quid and smoking has been a main contributing cause for oral cancer. [5] Tobacco use and alcohol consumption act synergistically to cause cancer of the oral cavity. Cancer incidence and survival rates are clearly linked to socioeconomic factors.[6,7] Low-income and disadvantaged groups are generally more exposed to avoidable risk factors such as environmental carcinogens, alcohol, infectious agents, and tobacco use. These groups have less access to the health services and health education. The biological activity of oral Squamous Cell Carcinoma (SCC) is evaluated and descriptively categorized as highly, moderately, and poorly differentiated.[8] Lipids are major cell membrane components essential for various biological functions, including cell growth and division of normal and malignant tissues. The major alkaloid in areca nut arecoline undergoes nitrosation

*Corresponding Author Address: Dr.KumarAshutosh,Room no. 35,P.G Boys Hostel, Medical College campus, Swaroop Nagar, Kanpur, IndiaEmail:ashu.scorpion86@gmail.com

and gives rise to N-Nitrosamine, which might have cytotoxic effect on the cells.[9] These 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 / tumorigenesis.[10] Lipids play a key role in maintenance of cell integrity. Because of lipid peroxidation, there is a greater utilization of lipids including TC, lipoproteins, and triglycerides for new membrane biogenesis. Cells fulfil these requirements from circulation either by synthesis through the metabolism or from degradation of major lipoprotein fraction such as VLDL, LDL, or HDL.[11]

MATERIAL AND METHODS:

The study was conducted by the department of ENT, GSVM Medical College, Kanpur. Sixty patients with clinically and histopathologically proven oral squamous cell carcinoma (SCC) in the age group of 20-80 yrs coming to the ENT OPD at LLR Hospitals, Kanpur were included in the study.

The study subjects comprised 2 groups as follows:

1. Group 1: Oral SCC Group.
2. Group 2: Control Group.

Group 1 (Oral SCC group) comprised of sixty patients in the age group of 20-80 years who were diagnosed to have oral squamous cell carcinoma confirmed by histopathology.

Group 2 (Control group) comprised of equal number of healthy subjects in the same age group , sex matched with those of the Oral SCC group and with no deleterious oral habits and no associated oral lesions.

Exclusion criteria: Patients with systemic diseases/conditions that may be associated with alterations in the serum level of lipid profile like obesity, diabetes mellitus, hypertension, thyroid disorder, chronic liver disease, chronic heart disease, malabsorption syndrome were excluded.

The patients were explained in detail about the study and the procedure they were subjected to. A formal informed written consent was obtained. Systemic and detailed oral cavity examination of the patients was done. Histopathological examination was carried out in all the cases following incisional biopsy from the affected area of the oral cavity.

Statistical analysis - All the variables from the study were statistically analyzed using independent t- test by Statistical Package for the Social Sciences program [SPSS version 16.0]

RESULTS:

Mean age of patients with Oral SCC was 51.40 years. Maximum patients (22; 36.67 %) were in 40-50 yrs of age. Male to female ratio was 1.73:1.The mean serum Cholesterol, serum HDL, serum LDL, serum VLDL and serum TG levels in oral SCC group were 125.97 mg/dL, 38.86mg/dL, 64.45 mg/dL, 23.20 mg/dL and 103.23 mg/dL respectively. However,

in the control group the corresponding values were 184.38 mg/dL, 57.44 mg/dL, 86.50 mg/dL, 36.17 mg/dL and 116.25 mg/dL respectively. A statistically significant reduction [P<0.001] was noted between the control group and Oral SCC cases for all lipid parameters.

DISCUSSION:

The habit of tobacco consumption is a known etiologic factor for development of oral precancerous diseases and head/neck cancer.[12-13] Cholesterol which is an amphipathic lipid is an essential structural component of all cell membranes and of the outer layer of plasma lipoproteins. It is present either as free cholesterol or combined with a long-chain fatty acid, as cholesterylesterin tissues and in plasma lipoprotein. It is synthesized from acetyl-CoA in many tissues and is ultimately eliminated as cholesterol or bile salts from the body. In the circulation, lipoprotein transports free cholesterol and it readily equilibrates cholesterol in other lipoproteins and in membranes.[14-17] Free radicals and reactive oxygen species are generated from tobacco carcinogens which are responsible for high rate of oxidation / per oxidation of polyunsaturated fatty acids. It results in greater utilization of lipids including total cholesterol, lipoproteins and triglycerides for new membrane biogenesis. Cells fulfil these requirements either from circulation, or by synthesis through metabolism or from degradation of major lipoprotein fractions like VLDL, LDL, and HDL.[18-20] Earlier studies have shown alteration of plasma lipid profiles in head and neck and other cancers.[11] In the present study, a significant decrease in plasma total cholesterol, HDL, LDL, VLDL and TG was observed in oral SCC patients as compared to the controls.

CONCLUSION:

Our study shows that there is an inverse relationship between serum lipid profile and oral SCC. The change in plasma lipid levels may be used as a diagnostic or prognostic biochemical indicator for early diagnosis of oral premalignant and malignant conditions. However, a detailed study on large sample size and on role of cholesterol in neoplasia should be carried out for better understanding of this inverse relationship of serum lipid profiles and oral pre malignant and malignant conditions.

REFERENCES:

1. Chen JK, Katz RV, Krutchkoff DJ. Intraoral squamous cell carcinoma. Epidemiologic patterns in Connecticut from 1935 to 1985. Cancer 1990;66:1288-1296.
2. Llewellyn CD, Johnson NW, Warnakulasuriya KA. Risk factors for squamous cell carcinoma of the oral cavity in youn people—a comprehensive literature review. Oral Oncol 2001;37:401-418.
3. SchantzSP,Yu GP. Head and neck cancer incidence trends in young Americans, 1973-1997, with a special analysis for tongue cancer. Arch Otolaryngol Head Neck Surg 2002;128:268- 274.
4. Lohe VK, Degwekar SS, Bhowate RR, Kadu RP, Dangore SB. Evaluation of correlation of serum lipid profile in

patients with oral cancer and precancer and its association with tobacco abuse. J Oral Pathol Med.2010;39:141–8.
5. Sankaranarayanan R. Oral cancer in India: An epidemiologic and clinical review. Oral Surg Oral Med Oral Pathol 1990;69:325-30.
6. Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJL, editors. Global burden of disease and risk factors. Washington: The Word Bank/Oxford University Press; 2006.
7. Jamison DT, Breman JG, Measham AR, Alleyne G, Claeson M, Evans DB, et al.,editors. Disease control priorities in developing countries. 2nd ed. Washington: The World Bank/Oxford University Press; 2006.
8. Anneroth G, Batsakis J, Luna M. Review of the literature and a recommended system of malignancy grading in oral squamous cell carcinomas. Scand J Dent Res 1987;95:229-49.
9. Hoffmann D, BrunnemannKD,Prokopczyk B, DjordjevicMV.Tobacco specific N-nitrosamines and areca derived N-nitrosamines: chemistry, biochemistry, carcinogenecity, and relevance to humans. J ToxicolEnv Health 1994; 41:1-52.
10. Ames BN. Dietary carcinogens and anticarcinogens: Oxygen radicals and degenerative diseases. Science.1983;221:1256–64.
11. . Patel PS, Shah MH, Jha FP, Raval GN, Rawal RM, Patel MM. Alterations in plasma lipid profile patterns in head and neck cancer and oral precancerous conditions. Indian J Cancer. 2004;41:25–31.
12. Gurudath S, Ganapathy K, D S, Pai A, Ballal S, Ml A. Estimation of superoxide dismutase and glutathione peroxidase in oral submucous fibrosis, oral leukoplakia and oral cancer — A comparative study. Asian Pac J Cancer Prev. 2012;13:4409–12.
13. Gupta PC, Ray CS. Smokeless tobacco and health in India and South Asia. Respirology. 2003;8:419–31.
14. JG Chawda, SS Jain, HR Patel, N Chaduvula, K Patel. The relationship between serum lipid levels and the risk of oral cancer. Indian J Med PaediatrOnc. 2011;32(1):34–37.
15. JE Bouquot, BS Whitaker. Oral leukoplakia- rationale for diagnosis and prognosis of its clinical subtypes or phases. Quintessence Int. 1994;25:133–140.
16. T Axell, P Holmstrup, IR Kramer, JJ Pindborg, M Shear. International seminar on oral leukoplakia and lesions related to tobacco habits. Community Dent Oral Epidemiol. 1984;12:145–154.
17. WK Hong, J Endicott, LM Itri, W Doos, JG Batsakis, R Bell, al et. 13-cis-retinoic acid in the treatment of oral leukoplakia. N Engl J Med. 1986;315 (24):1501–1505.
18. WT Friedawald, RI Levy, DS Fredrickson. Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, without use of the Preparative Ultracentrifuge" Clin Chem. 1972;18(6):492–502.
19. P McCarthy, G Shklar. Keratinizing lesions of the oral mucosa in Diseases of the oral mucosa, 2nded.Philadelphia, USA:Lea and Febiger. 1980:182–202.

20. World health organization collaborating centre for oral precancerous lesions. Definition of oral leukoplakia and related lesions: an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol. 1978;46:518–539.