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SERUM LIPID PROFILE ANALYSIS IN PATIENTS WITH ORAL LEUKOPLAKIA

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ABSTRACT

Background: Oral cancer is preceded by potentially malignant disorders. Oral Leukoplakia is one of the best-known pre-malignant lesions in the oral cavity that has the highest rate of malignant transformation. Literature shows numerous studies have been conducted on an altered lipid profile in head and neck cancers. An inverse relationship between serum lipid profiles has been seen in oral cancer and precancer. The present study was done to evaluate the serum lipid profiles in oral leukoplakia cases. **Aim and objectives:** To evaluate the alteration in serum lipid profile in patients with oral leukoplakia. **Materials and method:** The study was conducted on 20 oral leukoplakia cases and 20 controls. Lipid profile included analysis of total cholesterol (TC), low density lipoprotein cholesterol (LDL), high cholesterol (HDL), very low density lipoprotein cholesterol (VLDL), triglycerides (TG) and ratio of high and low density lipoprotein cholesterol. Lipid profiles were measured using the standard reagents. **Results:** TG, LDL & VLDL were more in males in both groups. TC and HDL levels were highest in females, but were non significant. A significantly reduced serum level of HDL, VLDL, TGL, TC and LDL were also reduced in the oral leukoplakia group. TGL were highest in patients who had mild dysplasia and lowest TC, while, moderate dysplasia cases had highest TC and lowest TG, LDL, VLDL, HDL. **Conclusion:** The reduced levels of lipid profile in cases of leukoplakia could be due to tobacco habits that reduced the lipid fractions. The reduced levels of lipid in oral leukoplakia could be used to assess the malignant transformation that could help in early detection and prevent the progression to carcinoma.

Key words: carcinogenesis, lipid peroxidation, tobacco, dysplasia

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I. INTRODUCTION

Leukoplakia is a potentially malignant disorder (PMD) and plays a vital role in pathogenesis of oral squamous cell carcinoma (OSCC) in the oral cavity. [1] The risk of neoplastic transformation varies from 0.3 to 25%. The dysplastic changes in leukoplakia increase incidence of malignancy over 30%. Differentiation, proliferation and apoptosis are fundamental aspects of tumor biology. Constant growth of precancer and cancer need a positive balance between cell apoptosis and malignant cell proliferation. [2,3] Molecular markers are recently been used to detect cancer from body fluids like saliva to predict prognosis, and monitor disease progression. [4] The idea of screening cases with malignancy by blood-based tests is interesting due to its ease, non-invasiveness, cost-effectiveness, and possibility of repeated sampling. [5] Alterations in blood cholesterol levels in diagnosing and treating various diseases has been studied by several workers. Researchers have reported association of serum/plasma lipids and lipoproteins with different cancers, but only few studies are reported association with head and neck cancers. [5] Past studies have evaluated lipid profile mostly in cancer patients; whereas, present study has been conducted to assess the changes in lipid profile in cases with leukoplakia. Further, association of serum lipid profile with histological grading also has been assessed in the present study.

II. MATERIAL AND METHOD

The subjects were divided into 2 groups' healthy controls and leukoplakia. Inclusion criteria were histopathologically confirmed cases of leukoplakia and healthy individuals without systemic illness between age group of 18-55 years. After a thorough medical history 12 hour Fasting Venous blood was drawn from forearm of 30 controls and 20 histopathologically confirmed cases of leukoplakia. Patients were informed about the procedure and written consent was taken. Samples were allowed to clot for 1 hour at 37^0C & then stored at 4^0 c till analysed. Serum total cholesterol (TC), high density lipoprotein (HDL) & low density lipoprotein (LDL) were estimated by colorimetric methods using cholesterol kit obtained from span diagnostic Ltd.Serum lipid estimation was done using cholesterol kits obtained from span diagnostic Ltd. 0.01ml serum sample was mixed with 1ml of working reagent for assessing TC. TG was evaluated by mixing 0.01ml serum sample with 1ml of triglyceride assay reagent. 0.2ml serum sample with 0.2ml HDL precipitating reagent were mixed followed by 10 minute incubation at room temperature for HDL evaluation. Supernatant was formed by centrifugation at 2800 g for 10 minutes and was mixed with 1ml of cholesterol reagent. The above 3 Mixtures obtained were incubated at 37^{0c} C for 10 minutes & measured using a spectrophotometer at 505nm against blank using distilled water and were calculated.

Statistical analysis was done by One way ANOVA for multiple group comparison followed by students "t" test for two group comparison. Pearson correlation coefficient was used to measure the relationship between variables. For all the tests a "P" of 0.05 or less was considered for statistical significance.

III. RESULT

Comparison of serum lipid profile in both study groups:

TC was lowest in leukoplakia followed by control group. LDL was high in leukoplakia than controls and difference was significant (p<0.002). HDL level was lowest in leukoplakia than control with difference being significant (p<0.001). VLDL was lowest in controls compared to leukoplakia and was significant (p<0.001). TG was lowest in leukoplakia and controls and difference was significant (p<0.001). [Table 1]

Table 1 lipid profile in 2 groups

		N	Mean	td. Deviatio	Statistics/ lean Square	f2 (welch) F (Anova)	p value
Age	Control	30	40.5	14.227	15.066	61.567	<0.001
	Leukoplakia	20	44.15	11.878			
Triglyceride -100-150mg/dl	Control	30	95.04	12.6442	22.937	64.634	<0.001
	Leukoplakia	20	79.05	11.1849			
Total Cholesterol & lt; 250mg/dl	Control	30	154.04	16.9006	27.53	67.28	<0.001
	Leukoplakia	20	120.05	14.4312			
HDL N-40-50 mg/dl	Control	30	41.62	2.962	68.383	55.177	<0.001
	Leukoplakia	20	31.4	13.766			
LDL -N 100-130	Control	30	98.28	18.0329	2234.483	4.999	0.002
	Leukoplakia	30	104.55	15.3296			
VLDL-4-40mg/dl	Control	30	18.5	1.8871	11.96	49.434	<0.001
	Leukoplakia	20	20.45	12.0152			

Correlation of Histopathological changes with lipid profile

Dysplastic changes in leukoplakia

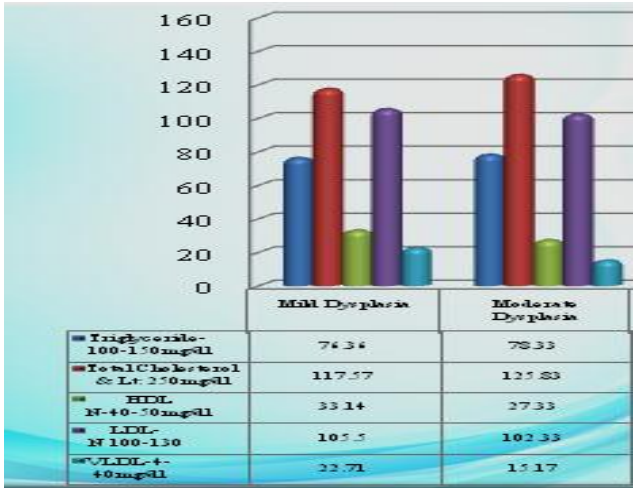
In mild dysplasia, the total cholesterol is 117.5±7.1, triglyceride is 79.3±9.5, VLDL is 22.71±13.5 and is significant (P 0.001, 0.001 and 0.002). The HDL is 33.1±14.5 and LDL is105.5±15.8 and are non significant (P 0.822 and 0.31).

In moderate dysplasia, total cholesterol, triglyceride, VLDL levels were 125.8±24.4, 78.3±15.4, 15.17±4.9 and the difference was statistically significant (P 0.001,0.001 and 0.002 resp.) and HDL, LDL levels were 27.3±15.01, 102.3±15.1 and were statistically nonsignificant (P 0.82,0.31 respectively). (Table 2, Graph 1)

Table 2 Correlation of lipid profile with Dysplasia (histological grading)

		N	Mean	Std. Deviation	Statistic	df1	df2	Sig.
Triglyceride -100-150mg/dl	Mild Dysplasia	14	79.36	9.532	12.159	5	26.154	<0.001
	Moderate Dysplasia	6	78.33	15.436				
Total Cholesterol & Lt;250mg/dl	Mild Dysplasia	14	117.57	7.187	20.495	5	25.036	<0.001
	Moderate Dysplasia	6	125.83	24.49				
HDL N-40-50 mg/dl	Mild Dysplasia	14	33.14	14.352	0.432	5	24.057	0.822
	Moderate Dysplasia	6	27.33	12.501				
LDL -N 100-130	Mild Dysplasia	14	105.5	15.878	1.259	5	25.379	0.312
	Moderate Dysplasia	6	102.33	15.135				
VLDL-4-40mg/dl	Mild Dysplasia	14	22.71	13.527	5.123	5	26.331	0.002
	Moderate Dysplasia	6	15.17	4.997				

Graph 1 correlation of lipid profile with histological grades



IV. DISCUSSION

Leukoplakia is the most common potentially malignant disorder of the oral mucosa. The term is derived from the Greek word Leuko meaning White and plax meaning plaque. Bazin first described it, but Schwimmer in 1877 differentiated it from psoriasis and termed it as Leukoplakia. Axell et al in 1984 defined leukoplakia as "whitish patch or plaque which cannot be characterized clinically or pathologically as any other disease and is not associated with any physical or chemical causative agent except the use of tobacco".[6,7]

Cholesterol and triglycerides are imperative lipid components of cell and are critical in carrying out necessary physiological functions. It is necessary for structural and functional cell integrity. [8] In cases of malignancy, significant changes in serum cholesterol occur. Because of carcinogenesis, low levels of serum cholesterol in blood and proliferating tissues are noted. Few explanations have been given for association of cholesterol and cancer: low cholesterol even before cancer detection could be result of cancer, cholesterol sets as a marker for cancer detection, may be associated with occurrence of few forms of cancers. [9] Reduced cholesterol may be due to increased lipid membrane biogenesis by cancer cells or direct lipid lowering effect of cancer cells or altered lipid metabolism or antioxidant activity. [10]

Lipid peroxidation is an essential biochemical process that involves the oxidation of polyunsaturated fatty acids, the important components of cell membranes. Tobacco carcinogens generate reactive oxygen species and lipid peroxides, leading to tissue injury due to elevated lipid peroxidation, further damaging the cellular structural blocks like lipids, proteins, DNA. Thus lipid peroxidation may have a role in endogenous formation of exocyclic DNA adducts.[4] It has been reported that smoking alters the serum lipid and lipoproteins by elevation of serum free fatty acids after smoking. It has been suggested that nicotine stimulates secretion of catecholamine's, leading to activation of adenyl cyclase of adipose tissue, resulting in increased lipolysis, increased concentration of plasma free fatty acids and increased secretion of hepatic triglycerides and VLDL cholesterol into the blood stream.[7] Exposure to tobacco carcinogens hampers antioxidant defense, leading to accelerated lipid peroxidation.

An alcoholic patient would be prone for developing nutritional deficiency and vitamin deficiency which in turn could affect the plasma lipid levels. Vitamin A levels have known to be monitoring cell differentiation and maturation process. However, the hypochoelsterolemia which prevails is due to the cancer effects or is it a cause for cancer occurrence is yet to be answered.[4] Low levels of cholesterol has been associated with increased incidence of cancer and the reverse also has been true where in cancers are associated with low levels of cholesterol.

We found a reduced TC, HDL, VLDL and TGL in leukoplakia when compared with the controls and findings were similar to that by Patel et al and also by Lohe VK et al and also Ghosh G et al. LDL levels were increased in the leukoplakia group and are similar to that reported by Manoharan.

Our study also found that as the grading increased from mild to moderate dysplasia the levels of lipid profile lowered further. 11-36% of cases have shown malignant transformation from dysplasia. [10] Hence a lowered lipid profile can help to assess the malignant transformation. It could have been a preamble finding in text.

V. CONCLUSION

We conclude from present study that biochemical changes take place in lipid levels in association with leukoplakia as compared to healthy individuals and these are attributed by increased membrane biogenesis of lipid by these tumor cells. Further research is still needed in this context.

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