

Serum Lipid Profile in Chronic Smokers with Head and Neck Malignancy vs Chronic Smokers without Head and Neck Malignancy: A Comparative Study

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ABSTRACT

Aim: Lipids play an important role in cell division and growth and have long been associated with cancers. Smoking is also known to alter serum lipid level. In this study, serum lipid profile in chronic smokers with head and neck malignancy was compared with chronic smokers without head and neck malignancy.

Materials and methods: In this study, 50 chronic smokers with histopathologically proven head and neck malignancy and 50 chronic smokers without malignancy were studied. Fasting blood samples were collected from the subjects and plasma lipid profile estimated.

Results: There is no significant difference in total cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein, very low-density lipoprotein values between the groups.

Conclusion: There is no significant alteration of serum lipid profile in patients with head and neck malignancies and therefore it cannot be used as a serum marker as a diagnostic tool for early detection.

Keywords: Chronic smoker, Fasting lipid profile, Head and neck malignancy.

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INTRODUCTION

Squamous cell cancer constitutes the most common head and neck malignancy and is related to tobacco and alcohol. Tobacco is the most important factor, and over 90% of patients have a history of smoking.¹

Lipids are the major components in cell membrane. They take part in various biological functions like cell growth and cell division of both normal and malignant

tissue.² Triglycerides (TGs) – cholesterol, low-density lipoprotein (LDL) cholesterol, and high-density lipoprotein (HDL) cholesterol – constitute plasma lipid profile. Lipids are carried in body fluids with the help of lipoproteins. The LDLs are responsible for the transport of cholesterol from liver to the cells and HDLs are involved in the transport of cholesterol from cells to the liver. Chylomicrons transport TGs from intestine to all cells. Very low-density lipoproteins (VLDLs) are involved in the transportation of TG from liver to other cells.³

Lower blood cholesterol levels are seen with various cancers.^{4,5} Because of rapidly proliferating tumor cells, there is increase in demand, and due to this there is reduction in blood cholesterol levels.^{2,3} Tobacco induces generation of free radicals and reactive oxygen species responsible for increase in peroxidation of polyunsaturated fatty acids (PUFAs), which affect cell membrane and in turn lead to increased utilization of lipids.^{2,6} Furthermore, some investigators have also documented low serum cholesterol levels with increased risk of cancer occurrence and mortality.^{7,8}

Only a few reports are available on plasma lipid profile in head and neck cancer.⁹ Considering these curiosities, the present study is aimed to compare serum lipid profile in chronic smokers with head and neck malignancy with chronic smokers without head and neck malignancy as smoking is also known to alter the lipid profile.

MATERIALS AND METHODS

This was a cross-sectional study conducted in a tertiary care hospital between May 2015 and April 2016. The participants were divided into two groups. In group I chronic smokers with histopathologically proven head and neck malignancy were included. In group II chronic smokers without malignancy who demographically matched with group I were included. Those who smoke at least one pack daily for at least 10 years were considered chronic smokers. Individuals suffering from medical problems which can alter lipid profiles like diabetes mellitus, uremia, nephritic syndrome, patients on lipid-lowering drugs, and women on oral contraceptives were excluded from this study. The patients were explained about the study and the procedure involved, and written and

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informed consent was taken. Ethical clearance for the study was obtained from the hospital ethical committee. Relevant clinical and demographic data were obtained from the patient. They also underwent a detailed ear, nose, and throat examination. Fasting blood samples (4 mL) were collected from the subjects in green heparin vacutainer and plasma lipid profile estimated. The total cholesterol (TC), TG, and HDL were estimated by microslide method in vitrous 5600 machine using colorimeter principle. The LDL and VLDL were calculated: $VLDL = TG/5$, $LDL = TC - HDL - VLDL$.

RESULTS

Each group comprised 50 males. The mean age and standard deviation (SD) of malignant group was 54.58 ± 12.585 and of nonmalignant group was 54.84 ± 13.730 .

The obtained values of TC, TG, HDL, LDL, and VLDL were tabulated and compared. Statistical analysis was done using unpaired t-test.

The nonmalignant group lipid profile parameters (mean and SD) were TC 160.68 ± 50.393 , TG 116.58 ± 79.672 , HDL 37.26 ± 16.505 , LDL 100.10 ± 39.494 , and VLDL 23.30 ± 15.931 .

The malignant group lipid profile parameters (mean and SD) were TC 157 ± 36.309 , TG 113.52 ± 40.619 , HDL 37.08 ± 12.200 , LDL 96.80 ± 30.279 , and VLDL 23.10 ± 9.237 (Table 1).

As seen in the table, there is no significant difference in TC, TG, HDL, LDL, VLDL values between both groups, including statistically (p-value > 0.05).

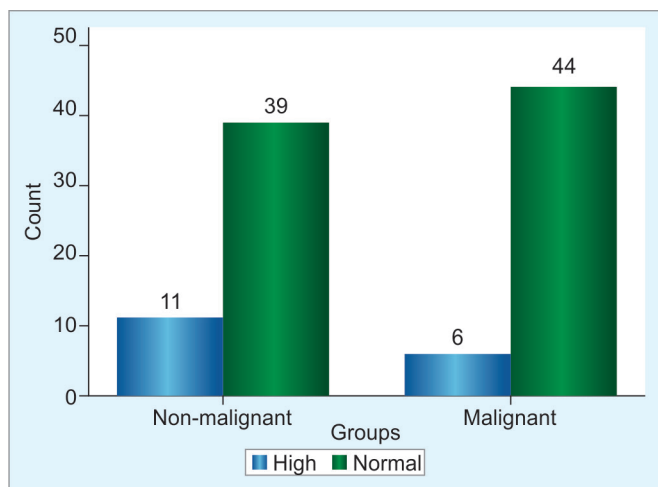
Also, in both the groups, majority of lipid profile values were within the normal range. In group I, 6 patients had elevated TC, 8 had elevated TG, 16 had elevated HDL, 22 had elevated LDL, and 7 had elevated VLDL. In group II, 11 had elevated TC, 12 had elevated TG, 16 elevated HDL, 23 elevated LDL, and 12 had elevated VLDL (Graphs 1 to 3). Hypolipidemia was not seen in any of the subjects.

DISCUSSION

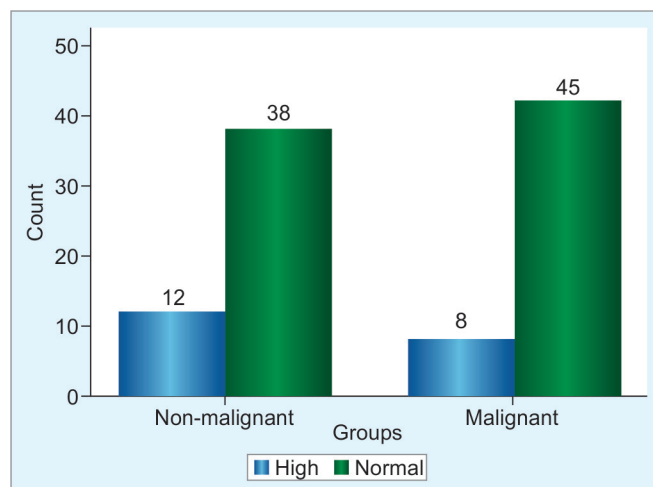
Head and neck cancer is one of the leading causes of morbidity and mortality, and habit of tobacco consumption is a known etiological factor. There are various studies on alteration in serum lipid profile pattern in malignancy.^{4,5} However, the reports on altered lipid levels in head and neck malignancy are few.⁹

Table 1: t-Test (group statistics)

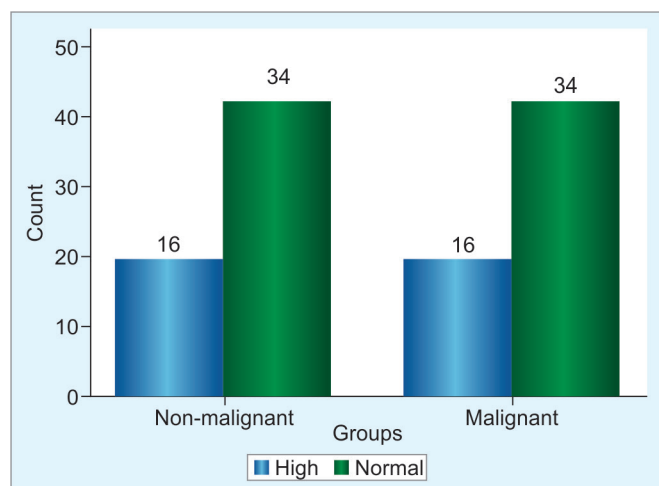
	Group	n	Mean	Std. deviation	Std. error mean	p-value
Total cholesterol	Nonmalignant	50	160.68	50.393	7.127	0.676
	Malignant	50	157.00	36.309	5.135	
Triglyceride	Nonmalignant	50	116.58	79.672	11.267	0.809
	Malignant	50	113.52	40.619	5.744	
High-density lipoprotein	Nonmalignant	50	37.26	16.505	2.334	0.951
	Malignant	50	37.08	12.200	1.725	
Low-density lipoprotein	Nonmalignant	50	100.10	39.494	5.585	0.640
	Malignant	50	96.80	30.279	4.282	
Very low-density lipoprotein	Nonmalignant	50	23.30	15.931	2.253	0.939
	Malignant	50	23.10	9.237	1.306	



Graph 1: Total cholesterol level



Graph 2: Triglyceride level



Graph 3: High-density lipoprotein level

In malignancies, the blood cholesterol undergoes significant and early changes, because of rapidly proliferating tumor cells and thereby increase in demand. There are many theories explaining this association: Newly forming and rapidly proliferating malignant cells need many basic components like lipids, leading to diminished lipid stores. Also tobacco induces generation of free radicals and reactive oxygen species responsible for high rate of oxidation/peroxidation of PUFAs, which affect cell membrane and in turn lead to increased utilization of lipids and low cholesterol levels may be causally associated.¹⁰

In a study conducted by Chao et al,¹¹ it was stated that hypolipidemia is a result of direct lipid-lowering effect of tumor cells and it should be recognized and early measure should be taken to restore cholesterol levels to avoid conditions that may hasten morbidity and mortality in cancer patients.

Chyou et al¹² and Schatzkin et al⁹ observed an inverse trend between lower serum cholesterol level and head and neck as well as esophageal cancer.

Nayak et al⁶ studied the alterations in plasma lipid profile in patients with oral submucous fibrosis by comparing with tobacco consumers and healthy subjects. The mean plasma lipid profile level was lower in oral submucous fibrosis compared with controls and tobacco users.

Gupta et al¹³ found a direct relationship between lipid profile and cancer patients and an inverse relationship between lipid profile and oral submucous fibrosis patients.

Chawda et al¹⁴ found an inverse relationship between the lipid levels and the occurrence of oral cancer and stated that the lower plasma lipid status may be a useful indicator to detect the initial changes seen in neoplastic process. But the study had a smaller sample size of total 30 subjects.

Alexopoulos et al⁵ reported a nonsignificant difference in serum TG between control and patients, which is consistent with our study, while others have observed elevated TG levels in malignancy patients.¹⁵

In a study conducted by Goyal et al,¹⁶ they reported that there was no statistically significant change in values of serum lipid profile in either the oral tobacco habit group or the oral precancer group.

In our study, we found no significant change of serum lipid profile in chronic smokers with head and neck malignancy, in comparison with chronic smokers without malignancy. Another significant finding is that there is no significant alteration in lipid profile of head and neck malignancy patients as majority had a normal lipid profile. Though few had hyperlipidemia, none had hypolipidemia, unlike few other studies.

There are several studies that show significantly higher levels of TG, LDL, VLDL, and TC in smokers as compared with nonsmokers.^{17,18}

Khan et al¹⁹ observed that smoking does not alter the blood cholesterol and triglyceride levels significantly. Other reports conducted on various ethnic populations have also shown similar results that smoking does not alter serum lipid levels.²⁰

Similarly, in this study, there is no significant alteration of lipid profile in smokers, where only few showed hyperlipidemia, majority being normal.

CONCLUSION

There is no significant alteration of serum lipid profile in patients with head and neck malignancies and, therefore, it cannot be used as a serum marker as a diagnostic tool for early detection.

However, there is scope for further research with larger group as there are contradicting reports, but with smaller groups in few studies.

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