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STUDY OF LIPID PROFILE IN CHRONIC SMOKERS ATTENDING SHADAN HOSPITAL-"TRASH THE ASH"

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ABSTRACT

Smoking is one of the most potent and prevalent addictive habits, influencing behavior of human beings. There is a dose response relationship between number of cigarettes smoked per day and cardiovascular morbidity and mortality. The study is designed to observe the effects of chronic smoking on plasma lipoprotein levels. A total of 100[50 non-smokers (group A) and 50 smokers (group B)] at Shadan hospital aged between 25-45 years were selected for this study. The tests are done by GPO-PAP, End point assay; CHOD-PAP, Enzymatic end point assay; Friedewald equation. Results Plasma total cholesterol in smokers is 178.68 ±34.59 and in non-smokers it is 165 ±25.84 and p ≤0.02 which is statistically significant.

KEYWORDS: Lipid Profile**,** Chronic Smoking, Plasma Cholestrol.

INTRODUCTION

Cigarette smoking is a major risk factor for cardiovascular diseases, obstructive pulmonary diseases, cancer. Smoking is one of the most potent and prevalent addictive habits, influencing behavior of human beings. More over the prevalence of tobacco use has declined among men in some developed countries, it is still increasing in women and young people. There is a dose response relationship between number of cigarettes smoked per day and cardiovascular morbidity and mortality.

SUMMARY

Cigarette smoking is associated with adverse effects on lipid profile and homocysteine thus increasing risk for atherosclerosis and coronary heart disease. Smoking is a prominent risk factor for coronary artery disease, atherosclerosis and peripheral vascular disorders. This study was undertaken to evaluate serum lipid profile in chronic smokers and to compare it with healthy non-smokers, considered as controls. Serum lipid profile was measured in 100 subjects aged between 25-45 years. Out of which 50 were smokers and 50 non-smokers (controls). It was revealed that mean serum Total Cholesterol (178.68 ± 34.59 mg/dl), Triglyceride (144 ± 32.02 mg/dl), Low Density Lipoprotein Cholesterol (131.22 ± 86.38 mg/dl) were significantly higher in chronic smokers as compared to non-smokers with mean serum Total Cholesterol (165.1 ± 25.84 mg/dl), Triglyceride (159.34 ± 24.99mg/dl), Low Density Lipoprotein Cholesterol (86.38 ± 16.71 mg/ dl). On the other hand, value of mean serum High Density Lipoprotein Cholesterol was lower in chronic smokers (36.38 ± 3.1 mg/dl) than in non-smokers (38.82 ± 3.14 mg/dl). Thus this study concludes that cigarette smoking produced adverse effects on lipid profile, leading to increase cardiovascular disease risk among smokers.

Many teenagers and adults think that there are no effects of smoking on their bodies until they reach middle age.[1] Smoking-caused lung cancer, other cancers, heart disease, and stroke typically do not occur until years after a person's first cigarette.

In fact, the latest research shows that serious symptoms of addiction – such as having strong urges to smoke, feeling anxious or irritable, or having unsuccessfully tried to not smoke – can appear among youths within weeks or only days after occasional smoking first begins.[2] The average smoker tries their first cigarette at age 12 and may be a regular smoker by age 14.[3] Everyday, individuals under 18 years of age become new regular, daily smokers.[4] Almost 90 percent of youths that smoke regularly report seriously strong cravings, and more than 70 percent of adolescent smokers have already tried and failed to quit smoking.[5]

Lipids play an important role in all aspects of biological life like serving as hormones or hormone precursor, helping in digestion, providing energy, acting as structural and functional compounds in bio-membranes and forming insulation to allow nerve conduction. There is a strong relationship between smoking and lipoprotein levels in plasma. Smoking leads to-Abnormalities in plasma lipoproteins levels. Lipids, a form of fat, are a source of energy for the body. Most people use this fat in its good form, called high-density lipoproteins, or HDLs. Some forms of fat, such as low-density lipoproteins (LDLs, triglycerides and cholesterol) can be harmful to the body. These harmful forms have their greatest effects on blood vessels. If produced in excess or accumulated over time, they can stick to blood vessel walls and cause narrowing. Such narrowing can impair blood flow to the heart, brain and other organs, causing them to fail. Most bodies have a balance of good and bad cholesterol. However, that is not the case for smokers. Nicotine increases the amount of bad fats (LDL, triglycerides, cholesterol) circulating in the blood vessels and decreases the amount of good fat (HDL) available.[6] These silent effects begin immediately and greatly increase the risk for heart disease and stroke.[7] In fact, smoking 1-5 cigarettes per day presents a significant risk for a heart attack.[8]

The study is designed to observe the effects of chronic smoking on plasma lipoprotein levels. A total of 100[50 non-smokers (group A) and 50 smokers (group B)] at Shadan hospital aged between 25-45 years were selected for this study. The test are done by GPO-PAP, End point assay; CHOD-PAP, Enzymatic end point assay; Friedewald equation.

REVIEW OF LITERATURE

Diseases related to tobacco smoking have been shown to kill approximately half of long term smokers when compared to average mortality rates faced by non-smokers. A 2007 report states that, each year, about 4.9 million people worldwide die as a result of smoking.[10] Tobacco smoking is the most popular form, being practiced by over one billion people globally, of whom the majority are in the developing world.[11]

Smoking is of two types

Active smoking

Passive smoking is the inhalation of smoke, called second-hand smoke (SHS), or environmental tobacco smoke (ETS), by persons other than the intended "active" smoker. It occurs when tobacco smoke permeates any environment, causing its inhalation by people within that environment

Smoking Mainly Affects Cardiovascular System

The cardiovascular system includes the heart and all of the blood vessels that carry blood to and from the organs. Smoking mainly and to a greater extent affects the cardiovascular system leading to greater cardiovascular risks.

Smoking decreases the nitric oxide which dilates blood vessels, and increases the endothelin-1 which leads to **vasoconstriction** causing immense reduction in blood supply. Vasoconstriction may produce complications for individuals whose blood vessels are already narrowed by plaques (atherosclerosis), or partial blood clots, or individuals who have sickle cell disease. Such individuals are at higher risk of stroke or heart attack.

Atherosclerosis: Atheroma means soft lipid center and sclerosis means scarring. It is a process in which cholesterol esters form “plaques” and stick to the walls of an artery reducing blood flow. This process starts at a younger age but is in the form of fatty streaks and dots then forms gelatinous lesion and finally at the age of 40 years forms atheromatous plaque leading to various complications like scarring, calcification, ulceration, hemorrhage and thrombosis. Nicotine and other toxic substances from tobacco smoke are absorbed into the blood stream and are circulated throughout the body. These substances damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a non-smoker. In this way, smoking increases the risk of heart disease by hastening atherosclerosis.

Thrombosis: Thrombus means blood clot. Components of tobacco smoke result in dangerously increased rates of clot formation. Smokers have elevated levels of thrombin, which is an important factor in blood clotting. This increases formation of blood clots which may get detached and further lead to more severe complication that is embolism.

Another effect of smoking on heart is to **increase heart rate.** Nicotine consumption increases heart rate, within 30 minutes after puffing; and the higher the nicotine consumption (like in chronic smokers) the higher the heart rate.

Blood pressure is defined as product of heart rate and cardiac output. It is expressed as a fraction, systolic over diastolic pressure. Systolic blood pressure is the highest arterial pressure during contraction of the heart while diastolic blood pressure is the lowest pressure during the heart’s relaxation phase. Nicotine consumption increases blood pressure. Higher blood pressure requires that the heart pump harder in order to overcome the opposing pressure in the arteries. The higher pressure can also cause organ damage where blood is filtered, such as in the kidneys.

Relationship Between Smoking And Abnormal Lipid Profile

The cardiovascular risk is significant for smokers who don’t have high cholesterol, and it increases significantly if they have a high lipid [cholesterol and triglycerides] profile, they smoke, are male, or have diabetes. When these factors combine, the risk increases exponentially.

Some suggested that the effect of smoking on LDL is mediated through reduction in lipoprotein lipase,[12] which was contradicted by Moriguchi and Eliasson et al. that no difference in lipoprotein lipase activity observed between smokers and non-smokers[13] Plasma lipase is an important regulator of plasma lipoprotein concentration. TGL rich lipoprotein is hydrolyzed by the catalyst lipoprotein lipase and thus, enables clearance of TGL from blood. Among smokers, hepatic lipase has been activated, which converts VLDL to LDL,[14] Nicotine also exerts hyperlipidemic effects by increasing the synthesis of TGL rich lipoprotein.[15]

In another study conducted by Majos O. D. et al.[16] reported that there is significant decrease in HDL-C, but there is no change in total cholesterol and triglycerides. Another report shows lower but no significant HDL levels in smokers.[17]

But a study conducted by Siekmeier et al.[18] reported the HDL levels are same for smokers and non-smokers. These variations might be due to different demographics of the subjects, different laboratory methods used and geographical distribution of the subjects.

Venkatesan et al found that total cholesterol and LDL-C were significant increase in smokers while HDL, VLDL, and triglyceride were not vary between two groups[18] Contrary report to this has been documented by Sirisali et al who found that total and LDL cholesterol did not vary between smokers and non- smokers,[19] Mammas et al[20] showed significant increasing in serum triglyceride and fall in LDL-C, while others were not significant. This difference in observation can be due to ethnic differences in the study population.[21]

The mean serum triglycerides levels in smokers were much higher when compared to non-smokers. These findings are similar to those observed by Wynder et al[22] and Rustogi et al.[23] The mean values of s. triglycerides were significantly higher in those subjects smoking 10-15 cigarettes/bidis per day as compared to those smoking 5-10 cigarettes/ bidis per day and even higher in case of those who smoked >15 cigarettes/bidis per day. These findings are similar to those of Rustogi et al. Recent studies have suggested that triglyceride levels are the most important factor leading to CHD.[24]

The mean serum total cholesterol was significantly higher in smokers. The total cholesterol values in subjects smoking 5-10 cigarettes/bidis per day were less and those smoking 10-15 cigarettes/bidis per day. These findings are in accordance with those of Muscat JE et al,[25] NS Neeki et al.[26] Increased cholesterol levels and CHD are observed in cigarette smokers.[27]

It was observed that HDL-cholesterol level was decreased in smokers when compared with non-smokers. This finding is similar to that of Rosenson[28] who reported that there is fall in HDL-C level by 3-5 mg/dl in smokers.

On the other hand, LDL & VLDL levels were also significantly increased in smokers than non-smokers and are in agreement with results of Kesaneimi and Grundy.[29] These values were significantly higher in subjects with severe smoking than that of mild and moderate smokers. These observations are also similar to those of Rustogi et al. The values of serum triglycerides, cholesterol and VLDL were higher in those subjects smoking cigarettes/ biddies for more than 15 years as compared to those who smoked for 10-15 years, 5-10 years and 1-5 years. A rising trend of mean values was seen with increase in duration of smoking but the differences were not statistically significant.

The present study is concerned with lipid profile in healthy non-smokers serving as controls (group B) and smokers (group A). The study was done using End point assay methods and Friedewald's equation. The finding of the study conducted differ from above findings due to different demographic methods and different laboratory methods used.

AIMS AND OBJECTIVES

Aim of this research is to find out the effect of smoking on serum lipoprotein levels.
The main objectives are

- Comparative study of lipid profile in non- smokers (group B) and smokers divided into (group A).
- To study variable patterns of lipid profile.
- To study the different effect of lipid profile in smokers.
- Increase knowledge of common programs and policies for protection and prevention.
- To identify information on health promotion interventions for smoking cessation, physical activity and healthy eating that are of proven effectiveness for use in populations at large.
- Development of specific warning messages.

MATERIALS AND METHODS

This is a prospective study conducted at Shadan Institute of Medical Sciences; Hyderabad after an approval from Institutional ethical committee and obtaining an informed consent from the study participants.

Inclusion Critezria

1) Subjects aged between 25-45 years, non- smokers and smokers.

Exclusion Criteria

1. Subjects with history of medical disorders such as hepatic, renal and cardiac disorders.
2. Alcoholics, family history of dyslipidemia.
3. Subjects on medications such as beta blockers, steroids, vitamin supplementation and lipid lowering drugs.

Collection of Blood Sample and Estimation of Lipid Profile

9-12 hours fasting samples are collected. Patient should sit comfortably for at least 5 minutes before sample collection. Torniquet should be released within 1 minute of application. The test are done by GPO-PAP, End point assay; CHOD-PAP, Enzymatic end point assay; Friedewald equation.

Procedure

For Total Cholesterol-Take serum/standard then add reagent 1 (Cholesterol mono reagent) to it. Mix well and incubate at 37^0C for 10 minutes and measure absorbance at 505nm.

For HDL-Cholesterol

Step 1: HDL-Cholesterol Separation

Take serum then add reagent 3 (Precipitating reagent) to it. Keep at room temperature for 10 minutes and then centrifuge for 15 minutes at 2000rpm.

Step 2: HDL-Cholesterol Estimation

Add supernatant from step 1/reagent 4 (HDL Cholesterol standard) then add reagent 1 (Cholesterol mono reagent). Mix well and incubate at 37^0C for 10 minutes and measure absorbance at 505nm.

For Trigycerides

Add serum/standard then add reagent 1 (Triglyceride mono reagent). Mix well and incubate at 37^0C for 10 minutes and measure absorbance at 505nm.

For LDL-Cholesterol

By Freidewalds equation

LDL Cholesterol = Total cholesterol-triglycerides/5-HDL cholesterol.

For VLDL-Cholesterol-([TG]/5) is used as an estimate of VLDL-cholesterol concentration.

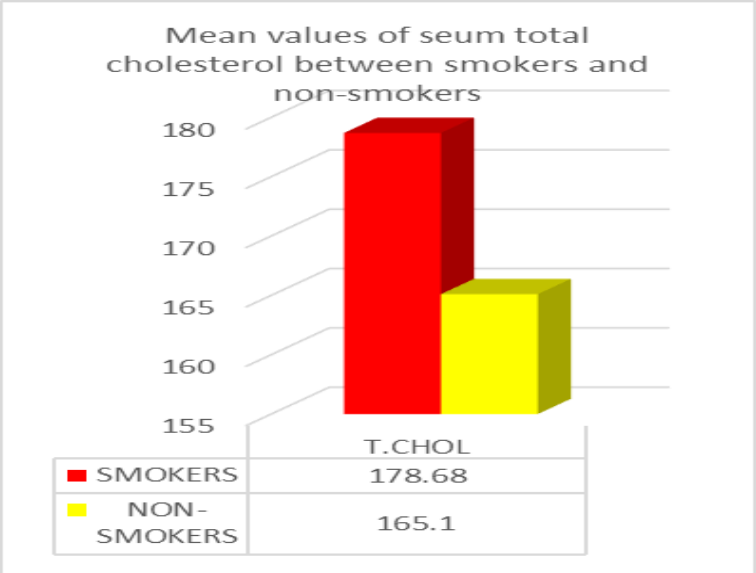
Statistical Analysis: Data were described using mean and standard deviation for the significant differences between groups. T-test was used to compare the differences of lipid profiles between smokers and non-smokers. All statistical tests were considered significant in p-value of <0.05 with a confidence level of 95%.

RESULTS

In table 1, plasma total cholesterol in smokers is 178.68 ±34.59 and in non-smokers it is 165 ±25.84 and p ≤0.02 which is statistically significant.

Table 1: Total-Cholesterol.

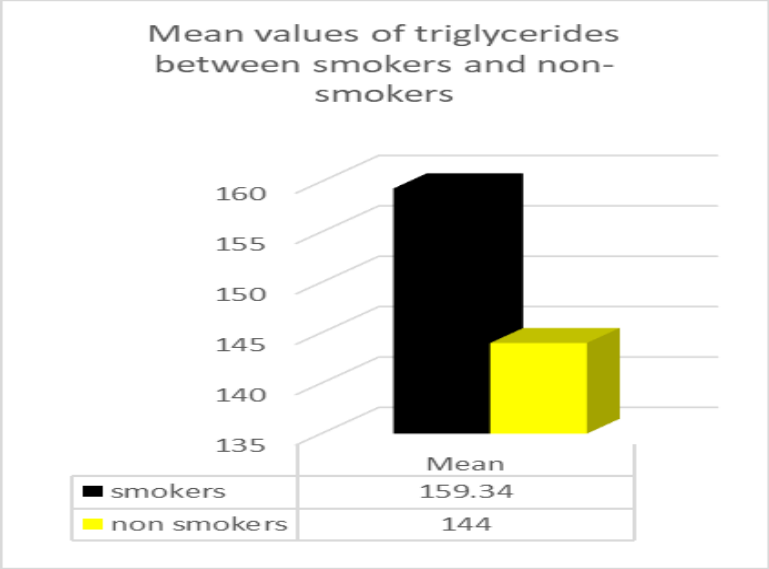
Parameter	Smokers	Non-smokers	P value
Mean Values	178.68 ±34.59	165 ±25.84	p ≤0.02 Significant



In table 2, triglycerides in smokers is 153.34 ±32.02 and in non- smokers it is 144 ±24.99 and p ≤0.008 which is very statistically significant.

Table 2: Triglycerides.

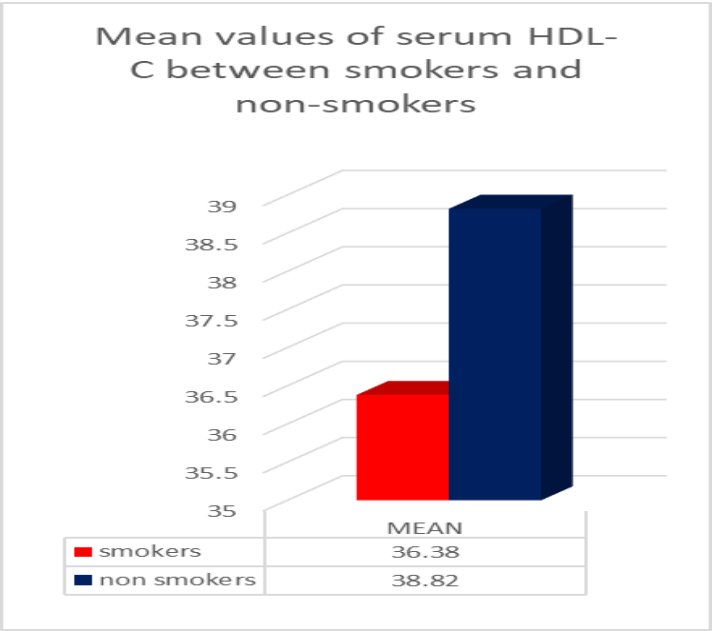
Parameter	Smokers	Non-smokers	P value
Mean Values	153.34 ±32.02	144 ±24.99	p ≤0.008 Significant



In table 3, HDL levels in smokers is 36.38 ±3.10 and in non-smokers it is 38.82 ±3.14 and p ≤0.0002 which is statistically significant.

Table 3: HDL-cholesterol.

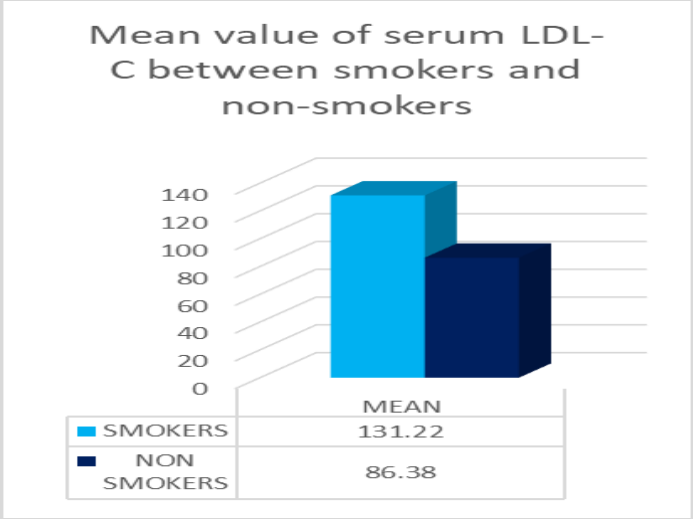
Parameter	Smokers	Non-smokers	P value
Mean Values	36.38 ±3.10	38.82 ±3.14	p ≤0.0002 Significant



In table 4, LDL levels in smokers is 131.22 ±52.4 and in non-smokers it is 86.38 ±16.71 and p ≤0.0001 which is extremely statistically significant.

Table 4: LDL-cholesterol.

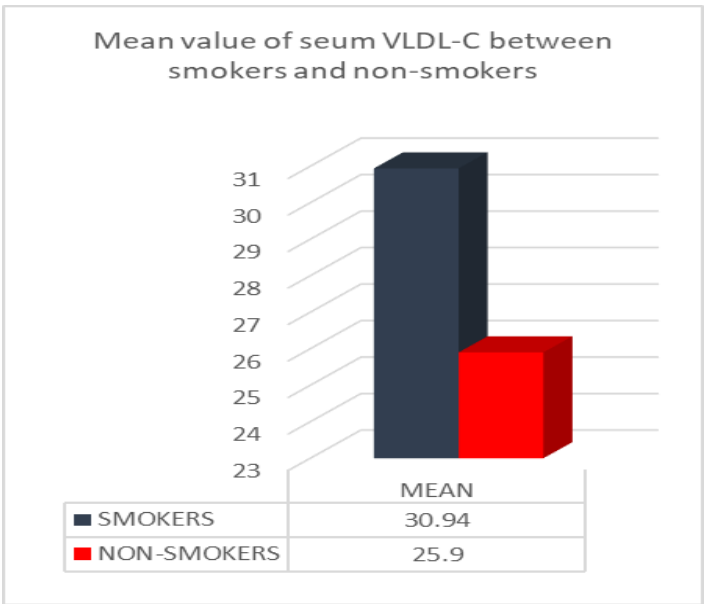
Parameter	Smokers	Non-smokers	P value
Mean Values	131.22 ±52.4	86.38 ±16.71	p ≤0.0001 Extremely significant



In table 5, VLDL levels in smokers is 30.94±9.7 and in non-smokers it is 25.9±3.38 and p≤0.0002 which is statistically significant.

Table 5: VLDL-cholesterol.

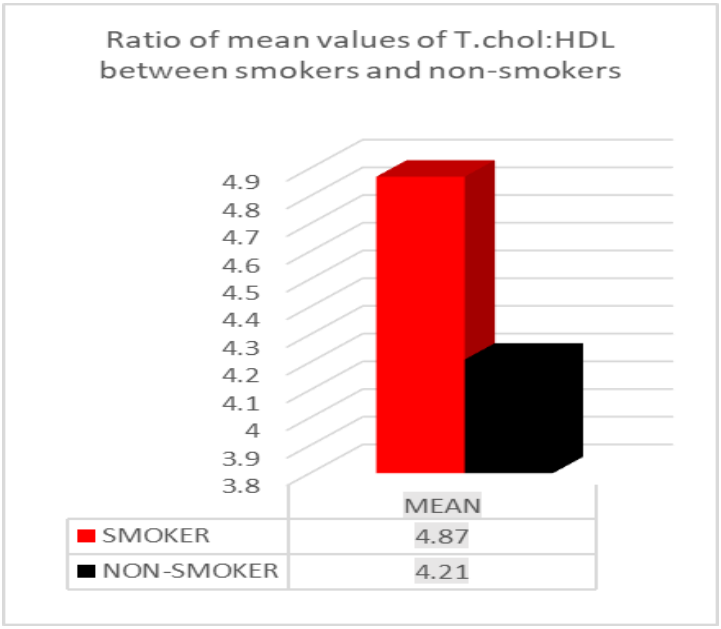
Parameter	Smokers	Non-smokers	P value
Mean Values	30.94±9.7	25.9±3.38	p≤0.0002 Significant



In table 6, T. Cholesterol: HDL ratio in smokers is 4.87 ±0.97 and in non-smokers it is 4.21±0.63 and p≤0.0001 which is statistically significant.

Table 6: T.chol: HDL.

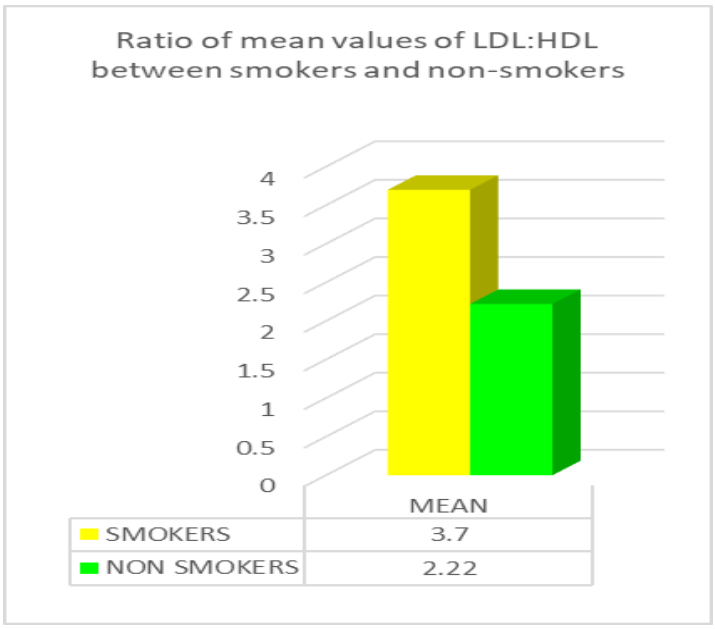
Parameter	Smokers	Non-smokers	P value
Mean Values	4.87 ±0.97	4.21±0.63	p≤0.0001 Significant



In table 6, LDL: HDL ratio in smokers is 3.7 ±1.26 and in non-smokers it is 2.22 ±0.50 and p≤0.0001 which is extremely statistically significant.

Table 7: LDL: HDL.

Parameter	Smokers	Non-smokers	P value
Mean Values	3.7 ±1.26	2.22 ±0.50	p≤0.0001 Significant



DISCUSSION

In the present study the parameters of lipid profile that is total cholesterol, triglycerides, LDL, VLDL, T. Cholesterol:

HDL and LDL: HDL were significantly elevated in smokers when compared to non-smokers.

Total cholesterol-178.68 ±34.59 mg/dl and in non-smokers it is 165 ±25.84 mg/dl, p ≤0.02.

Triglycerides-153.34 ±32.02 mg/dl and in non- smokers it is 144 ±24.99 mg/dl, p ≤0.008.

The elevated levels of total cholesterol and triglycerides are in co-ordination with LDL, VLDL, T.cholesterol:hdl and ldl:hdl (atherogenic index) which are also elevated in smokers. While in non-smokers HDL levels are decreased in smokers when compared to non-smokers.

HDL cholesterol- HDL levels in smokers is 36.38 ±3.10 mg/dl and in non-smokers it is 38.82 ±3.14 mg/dl, p ≤0.0002.

LDL cholesterol- LDL levels in smokers is 131.22 ±52.4 mg/dl and in non-smokers it is 86.38 ±16.71 mg/dl, p ≤0.0001.

VLDL cholesterol-VLDL levels in smokers is 30.94±9.7 mg/dl and in non-smokers it is 25.9±3.38 mg/dl, p≤0.0002.

Ratio of T.Chol: HDL was significantly higher in smokers 4.87±0.97mg/dl than in non-smokers 4.21±0.63mg/dl, p≤0.0001.

The atherogenic index (LDL: HDL) was significantly higher in smokers 3.7±1.26 mg/dl than in non-smokers 2.220±0.50 mg/dl, p≤0.0001.
Epidemiological studies have shown that long-term morbidity and mortality in coronary artery disease manifest over years and is directly proportional to circulating levels of lipoproteins, in particular LDL, so also known as "Bad cholesterol".

The most well-documented impact that smoking has on cholesterol is

1. Nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol.[30] as well as hepatic secretion of very low density lipoprotein (VLDL) and hence increased TGL.[31]
2. Smoking decreases estrogen levels and further leads to decreased HDL cholesterol concentration.[32] Also, HDL concentration was inversely related to VLDL concentration in serum.
3. Smoking increases insulin resistance and thus, causes hyperinsulinemia. LDL, VLDL and TGL are elevated in hyper insulinemic conditions due to decreased activity of lipoprotein lipase.[33]

It has been suggested that smoking even of short duration and quite moderate consumption of cigarettes is associated with adverse lipoprotein profile and many other adverse effects. These findings suggest that smoking alters the lipid profile adversely causing dyslipidemia in smokers. Smoking plays the key role for atherosclerotic process and with coronary artery disease.

CONCLUSION

The total serum cholesterol, LDL, VLDL and Triglyceride values are higher in smokers as compared to Non-smokers. Serum levels of HDL are lower in smokers than the same in non-smokers. Increase in duration of smoking adversely affects lipid profile. It shows that serum anti-atherogenic HDL level is significantly low in chronic smokers.

So we would conclude that these indices or lipid ratios are used for identifying individuals at higher risk of cardiovascular disease in the clinical practices especially, when the absolute values of lipid profile seem normal or higher or deranged due to insufficient sources. Increased amount of smoking causes more of dyslipidemia. The rapid reduction in risks of cardiac events after cessation of smoking implies that policies that prevent and reduce smoking will have large benefits for reducing cardiovascular mortality and morbidity.

Some anti-smoking programmes can be taken up by the ministry of health through visible and audible communication aids for welfare of the public, to explain risks of smoking on the cardiovascular system and other systems.

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Competing interests: None.

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