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Study of Lipid Profile and Thyroid Profile in Smokers and Non-Smokers

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ABSTRACT

In humans, cigarettes are the most common cause of toxic chemical poisoning and chemical-induced diseases. The problem of tobacco remains one of the world's most serious problems, killing more than eight million people a year. In addition, more than 7 million deaths are due to tobacco use, while 1.2 million are non-smokers (SMS). Tobacco is consumed by 337 million Indians over a decade. According to the World Health Organization, tobacco-related deaths in India could reach 1.5 million a year by 2020. When someone smokes regularly and consistently, carboxy Hb levels rise, causing progressive hypoxia, and when CO binds to Hb, active anemia increases. This causes a decrease in tissue volume and changes in hematological parameters. Study of Lipid Profile and Thyroid Profile In Smokers and Non-Smokers The study conducted Department of Biochemistry this study included 50 healthy male subjects of age 25 -50 years Both smokers and Non-smoker subjects who were referred to S MHRC Consequently, 25 subjects were smokers and 25 subjects were non-smokers. Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine and Dept. of Respiratory, DMMC and SMHRC, Nagpur in collaboration with JNMC & ABVRH (DMMC Deemed To Be University), Sawangi, Wardha, Maharashtra. Thyroid profile in the study group revealed a highly important difference (decrease in serum T3 and T4 and rise in serum TSH) of $P<0.0001$in smokers compared to non-smokers, as seen in table 1. In summary, this study will add to our understanding of how smoking affects thyroid function and will lead to our understanding of its negative effects. Because of this, it may be helpful in diagnosing and treating thyroid defects in smokers. Increased knowledge of participants about their thyroid status can serve as a smoking cessation and thus encourage them to improve their lifestyle habits. The smokers in this sample had dyslipidaemia the rate of smoking rises, as it does in heavy smokers, and the altered lipid profile worsens.

KEY WORDS: SMOKERS, LIPID PROFILE, THYROID PROFILE, WHO,CIGARETTE.

INTRODUCTION

Tobacco is consumed by 337 million Indians over a decade. According to the World Health Organization, tobacco-related deaths in India could reach 1.5 million a year by 2020. Cigarette use is an important and independent factor in the risk of atherosclerosis, heart disease, cardiovascular problems, and other cardiovascular diseases, and many findings indicate that nicotine is strongly associated with changes in lipid profile. Apart from all this information, there is still much debate about the elements of the lipid profile that are most affected by smoking, and if certain parts of the lipid profile influence other parts of the lipid profile explicitly or indirectly, and vice versa. Different investigators come to different conclusions. For example, Siekmeier et al. Concluded

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that HDL-C levels are the same for both smokers and non-smokers, while Ito et al.4 For smokers, lower levels of HDL-C were found. Smoking has many effects on the hypothalamic-pituitary-thyroid axis and thyroid gland function. Thyroid hormone synthesis, binding, delivery, storage and removal are all altered as a result of smoke exposure, which has led to the development of hormone concentrations.

In humans, cigarettes are the most common cause of toxic chemical poisoning and chemical-induced diseases. The problem of tobacco remains one of the world's most serious problems, killing more than eight million people a year. In addition, more than 7 million deaths are due to tobacco use, while 1.2 million are non-smokers (SMS). Polycythemia can be caused by too much carbon monoxide (CO) in humans and animals. CO has a half-life of 3-5 hours in the body. When someone smokes regularly and consistently, carboxy Hb levels rise, causing progressive hypoxia, and when CO binds to Hb, active anemia increases. This causes a decrease in tissue volume and changes in hematological parameters.

The effect of tobacco smoke on the thyroid is thought to be largely due to a compound called thiocyanate, which is a hydrogen cyanide that is half as healthy for more than 6 days. It has been well researched as goitrogen. It may prevent iodide transport and its regulation and can cause goiter in people who are deficient in iodine. Smoke from a burning cigarette emanates from both streams in the middle. Second Hand Smoke (SHS), which smokers have access to, is made up of both streams (11%) and side smoke (85%) of smoke, as well as other pollutants.

Aim: Study of Lipid Profile and Thyroid Profile In Smokers and Non-Smokers

MATERIAL AND METHODS

The study conducted Department of Biochemistry this study included 50 healthy male subjects of age 25 -50 years Both smokers and Non-smoker subjects who were referred to S MHRC Consequently, 25 subjects were smokers and 25 subjects were non-smokers. Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine and Dept. of Respiratory, DMMC and SMHRC, Nagpur in collaboration with JNMC & ABVRH (DMMC Deemed to be University), Sawangi, Wardha, Maharashtra.

Sample Collection: 5ml of each patient's blood sample was taken and separated in plain tube. The sample was used to estimate the levels of Lipid profile and Thyroid profile.

Biochemical Analysis: Lipid profile was estimated on AU480 Analyser and Thyroid profile were estimate in Chemiluminescence immunoassay analyser.

Table 1. Comparison between smokers and Non-smokers patients.

Parameters	Smokers (n=25)	Non-smokers (n=25)	P-value
Cholesterol (mg/dl)	210.5±16.4	164.2±20.9	P<0.0001
Triglyceride (mg/dl)	174.6±22.6	110.8±23.6	P<0.0001
HDL (mg/dl)	36.7±3.41	48.7±5.89	P<0.0001
LDL (mg/dl)	194.3±13.7	98.4±21.3	P<0.0001
VLDL (mg/dl)	34.8±9.87	21.4±6.11	P<0.0001
Total T3 (ng/ml)	0.80±0.41	1.67±0.39	P<0.0001
Total T4 (µg/dl)	4.10±0.73	6.94±1.24	P<0.0001
TSH (µIU/ml)	7.76±1.68	4.0±0.50	P<0.0001

RESULTS

Patients who were non-smokers and smokers were compared. Total cholesterol, triglycerides, LDL, and VLDL are statistically higher in smokers than non-smokers, but the same is true for HDL-cholesterol. Smokers had slightly lower HDL cholesterol than non-smokers. Thyroid profile in the study group revealed a highly important difference (decrease in serum T3 and T4 and rise in serum TSH) of P<0.0001in smokers compared to non-smokers, as seen in table 1.

DISCUSSION

In a recent study, researchers discovered that smokers had significantly higher levels of total cholesterol, triglycerides, LDL-C, VLDL-C, and a significantly lower level of HDL-C than non-smokers. These findings were similar to those reported by Devaranavadgi BB et al. The increase in catecholamine release caused by smoking, leading to an increase in VLDL-C and a decrease in HDL-C concentration, may also explain why HDL-C levels continue to fall in chronic smokers. As a result, smoking causes coronary artery disease and atherosclerosis by lowering theanti-atherogenic element HDL-C and actually increasing the atherogenic lipoproteins LDL-C, which severely weakens the vascular endothelium.

Low levels of serum T3 and T4, as well as high levels of TSH, were observed in both smokers and participants in the report, confirming the results of Nystrom et al. 12 and S S Fukata et al. 13, respectively. Nystrom found that smokers had lower serumT3 levels than

non-smokers, while Fukata found a link between smoking and hypothyroidism, caused by high serum thiocyanate, which is caused by smoking. smoking and hypothyroidism, caused by high serum thiocyanate, caused by smoking. Numerous studies have shown that smoking has a negative, positive, or negative effect on peripheral viral hormones. 14 T4 and retrospective T3 (rT3) levels were shown to increase in line with normal T3 levels in some studies, while others showed an increase in T3 without a corresponding increase in T4.15 Different studies on lipid profile in various groups of cases were reported 16-18. Studies related to effects of smoking19-20, thyroid disorders 21-23 were reviewed.

CONCLUSION

In summary, this study will add to our understanding of how smoking affects thyroid function and will lead to our understanding of its negative effects. Because of this, it may be helpful in diagnosing and treating thyroid defects in smokers. Increased knowledge of participants about their thyroid status can serve as a smoking cessation and thus encourage them to improve their lifestyle habits. The smokers in this sample had dyslipidaemia the rate of smoking rises, as it does in heavy smokers, and the altered lipid profile worsens. As a result, quitting smoking early can alter these processes, which may prevent any major health risks.

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