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Role of Cardiac Biomarkers and Thyroid Profile in Smokers and Non –Smokers

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ABSTRACT

Smoking has a negative impact on all systems of the human body and is a known risk factor for a variety of deadly illnesses. A range of pathophysiological processes, including vasomotor effects, irritation, smooth muscle development, and platelet dysfunction, mitigate the effects of smoking on the cardiovascular system (CVS). Furthermore, smoking affects a person's oral microbiota and harms the periodontium via a variety of physical obstacles. The hypothalamic-pituitary-thyroid axis and thyroid function are both affected by smoking. Variations in thyroid hormone production, binding, transport, storage, and elimination, as well as changes in hormone concentration, are all negative mechanisms of smoking exposure. The goal of this study was to see how smoking affected the thyroid profile, serum TSH levels, T3 levels, and total T4 levels in smokers and non-smokers. Thyroid profile highly significant variation (t3 and t4 decreased in smokers patient 0.79±0.40, 4.00±0.70 as compared to non smokers patient 1.51±0.38, 6.85±1.20) and serum TSH increased in smokers patient (7.70±1.60) as compared to non-smokers patient ($P < 0.0001$). serum Trop-I, ALT(SGPT) and AST(SGOT) concentration increased in smokers patient (0.234±0.020, 75.30±5.43 and 34.40±2.20) as compared to non smokers patient (0.084±0.030, 40.90±8.90, 23.96±3.50) serum LDH and total CPK concentration also increased in smokers patient (185.90±4.20, 168.70±6.86) as compared to non smokers patient. To conclude, this research adds to our understanding of how smoking impacts thyroid functioning in cardiac biomarkers and adds to our understanding of its negative impacts. As a result, it might aid in the early detection and treatment of thyroid and cardiovascular illness among smokers.

KEY WORDS: THYROID PROFILE, CARDIAC BIOMARKERS, SMOKERS, NON SMOKERS.

INTRODUCTION

Tobacco use is the most prevalent way for people to be exposed to harmful compounds and chemical mediators.

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The tobacco pandemic is one of the world's most serious public health hazards, killing around eight million people per year. Furthermore, more than seven million people die as a consequence of direct tobacco use, while an estimated 1.2 million people die as a result of secondhand smoke(SHS) exposure. Smoke from a burning cigarette comes from both the main and side streams. Second Hand Smoke (SHS), which passive smokers are exposed to, is made up of both mainline (11%) and sidestream (85%) smoke, as well as other pollutants. Passive smokers, on the other hand, are exposed to a distinct set of toxicants than active smokers.

Smoking has a negative influence on all human bodily systems and is a prevalent risk factor for a variety of death-causing disorders. Smoking has various pathophysiological consequences on the cardiovascular (CV) system, including vasomotor effects, irritation, smooth muscle enlargement, and platelet dysfunction. Furthermore, smoking affects oral microbiota in humans and causes periodontal disease through a variety of immunological difficulties. The sluggish course of periodontal disease adds to the problem's complexity, necessitating a long-term follow-up to track the illness's course. The relationship between CP and coronary heart disease (CHD) is unclear. Other factors, however, confound this link. With the awareness that not only is the participating heritage included, but also natural elements and other threats. The link between CP and CHD has been the subject of inquiry for decades.

Smoke-induced thyroid homeostasis can occur in both active and passive smokers. The computer, thiocyanate, which is contained in a healthy half-day hydrogen cyanide for six days, is thought to be the main source of tobacco smoke's influence on the thyroid. It has been extensively researched as a possible goitrogen. It obstructs iodide transport and control, and if a deficit is discovered, it can lead to goitre. By distinguishing blood components linked with myocardial infarction, CV biomarkers can identify heart function. The biomarkers (ALT, AST, LDH, CK, and Tr-I) are listed below. The researchers determined that smoking can impair the cardiovascular system by raising the pressure on the ventricular wall, resulting in less damage and myocardial infarction. Furthermore, the more sensitive troponins (hstroponins) and CV have a substantial connection. The effect of excessive smoking on TBG, as well as the T3 and T4 binding interactions, is unknown. Oral contraceptives, on the other hand, have been demonstrated to enhance blood TBG concentration by 50% due to higher estradiol levels.

MATERIAL AND METHODS

The study conducted Department of Biochemistry this study included 60 healthy male and female subjects of age 30 -50 years Both smokers and Non-smoker subjects who were referred to Shalinitai Meghe hospital and Research center Consequently, Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine and Dept. of Respiratory, Datta Meghe Medical College and Shalinitai Meghe Hospital and Research Center, Nagpur in collaboration with JNMC & ABVRH.

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 cardiac biomarkers and Thyroid profile.

Biochemical Analysis:

- Thyroid profile and troponin I was estimate in Chemiluminescence immunoassay analyser.
- Serum ALT, AST, LDH AND Ck was estimated on AU480 Analyser.

Statistical Analysis: The SPSS (Statistical Package for the Social Sciences) application version-22 was used to conduct the statistical analysis. When P< 0.05, the significance was assessed. Microsoft Word version 2016 was used to chart and graph the statistical data.

Table 1. Thyroid profile and Cardiac Biomarker comparison between smokers and non- smoker's patient.

Parameters	Smokers (N-30)	Non- Smokers (N-30)	P- Value
T3	0.79±0.40	1.51±0.38	P < 0.0001
T4	4.00±0.70	6.85±1.20	P < 0.0001
TSH	7.70±1.60	4.20±0.49	P < 0.0001
TROP -I	0.234±0.020	0.084±0.030	P < 0.0001
ALT	75.30±5.43	40.90±8.90	P < 0.0001
AST	34.40±2.20	23.96±3.50	P < 0.0001
LDH	185.90±4.20	132.65±17.00	P < 0.0001
TOTAL CK	168.70±6.86	75.75±20.34	P < 0.0001

RESULTS

Table no 01 show the thyroid profile highly significant variation (t3 and t4 decreased in smokers patient 0.79±0.40, 4.00±0.70 as compared to non smokers patient 1.51±0.38, 6.85±1.20) and serum TSH increased in smokers patient (7.70±1.60) as compared to non smokers patient (P < 0.0001). serum Trop-I, ALT and AST concentration increased in smokers patient (0.234±0.020, 75.30±5.43 and 34.40±2.20) as compared to non smokers patient (0.084±0.030, 40.90±8.90, 23.96±3.50) serum LDH and total CPK concentration also increased in smokers patient (185.90±4.20, 168.70±6.86) as compared to non smokers patient.

DISCUSSION

Cigarette smoking is a leading cause of cardiovascular death and morbidity that may be prevented. Even when coronary artery disease is taken into consideration, epidemiological studies show that former and current smokers have a higher risk of heart failure. Tobacco

smoking has been shown in animal experiments to have direct harmful effects on the myocardium.8 Cigarette smoking, on the other hand, is an independent risk factor for periodontal disease start, progression, and severity. Furthermore, smoking has been demonstrated to impair the effectiveness of periodontal therapies. The purpose of this study was to compare the levels of blood cardiac biomarkers in smokers and non-smokers. The serum Trop-I, ALT, and AST concentrations were measured in this investigation. increased in smokers patient (0.234±0.020, 75.30±5.43 and 34.40±2.20) as compared to non smokers patient (0.084±0.030, 40.90±8.90, 23.96±3.50) serum LDH and total CPK concentration also increased in smokers patient (185.90±4.20, 168.70±6.86) as compared to non smokers patient.

Tobacco smoking affects the thyroid gland's practically all functions. When thyroid function is weakened, the harmful effects of smoking become apparent, 10 leading to hypothyroidism..9 It might have two modes of action on the thyroid gland: direct suppression by thiocyanate and indirect stimulation via the Hypothalamus–pituitary axis. Thiocyanate competes with iodide in the organification process, inhibiting iodine absorption and hormone production. 5 Other components of smoke, such as 2,3 hydroxypyridine, hinder deiodination by lowering iodothyronine deiodinase activity, which interferes with thyroid function. The present study low serum T3 and T4 levels and significantly high TSH values in both smokers and non smokers patient . Table no 01 show the thyroid profile highly significant variation (t3 and t4 decreased in smokers patient 0.79±0.40, 4.00±0.70 as compared to non smokers patient 1.51±0.38, 6.85±1.20) and serum TSH increased in smokers patient (7.70±1.60) as compared to non smokers patient ($P < 0.0001$).

TBG binds around 75% of serum T4; practically all of the rest is tied to transthyretin or albumin, leaving less than 0.1 percent free or unbound. It is unknown if cigarette smoking affects the capacity and affinity of T3 and T4 binding to TBG. Oral contraceptives, on the other hand, have been observed to raise blood TBG concentrations by up to 50% due to increased estradiol levels. Smoking-related alterations in maternal thyroid function during pregnancy may be linked to changes in thyroid hormone levels, which might have a negative influence on the baby' neurocognitive and neurobehavioral development. Smokers exhibited lower levels of serum TSH and greater levels of T3 than nonsmokers in a sample of pregnant women.12 Different studies on lipid profile in various groups of cases were reported 13-15. Studies related to effects of smoking16-17, thyroid disorders 18-20 were reviewed. Studies on usefulness of Alirocumab21-23 and Ghrelin in cardiac issues were reported24-25. Our findings shows that, as compared to nonsmokers, active and passive smoking was linked with a greater probability of having considerably lower TSH levels, suggesting that cigarette smoke exposure has an inhibitory impact on the thyroid. TSH and thyroid hormone levels were both within acceptable limits.

CONCLUSION

To conclude, this research adds to our understanding of how smoking impacts thyroid functioning in cardiac biomarkers and adds to our understanding of its negative impacts. As a result, it might aid in the early detection and treatment of thyroid and cardiovascular illness among smokers.

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