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A study on relation of TSH (Thyroid stimulating hormone) levels in smokers and non-smokers-prospective study

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Abstract
Background: The effect of smoking on thyroid is believed to be due to the compound thiocyanate, a potent inhibitor of iodide transport, potentially capable of affecting the thyroid function. TSH is the major regulator of the morphologic and functional states of the thyroid. All steps in the formation and release of thyroid hormones are stimulated by TSH secreted by the pituitary thyrotrophs. These include both pro- (e.g. thyroid stimulating) and anti-thyroid actions and also actions that increase susceptibility to or exacerbation of the manifestations of Graves' disease. Thus, this study may be instrumental in early detection and management of thyroid disease in smokers.
The aim of this study is to evaluate and compare TSH thyroid stimulating hormones status in smokers and non smokers.
Methods: A prospective study is done with 60 patients divided into 2 groups. Group 1 (healthy male non-smokers) as control and Group 2 (healthy male smokers). Fasting blood samples were collected and serum TSH levels were estimated. The data was analyzed using unpaired t test.
Results: In the present study significant decrease in TSH observed in the cases compared to the controls. Mean±S.D of TSH in Group 1 is 2.80±0.83 and Group 2 is 1.00±1.02, there is a significant decrease in serum TSH levels in Group 2.
Interpretations & Conclusion: The finding in this study indicates that smoking is associated with biochemical hyperthyroidism. Hence evaluating thyroid hormone status in smokers might help in identifying occurrence of thyroid disorders and appropriate measures could be taken to prevent severity of morbidity and mortality associated with smoking.

Keywords: Smokers, thyrotrophs, TSH

Introduction
Tobacco smoking has complex effects on endocrine organs some of which may be associated with important clinical implications (Kapoor and Jones, 2005) [4]. Tobacco smoking increases the risk and severity of Graves' disease, Graves' ophtalmopathy and thyroid multi modularity (Vestergaard *et al*., 2002) [12]. However the relationships between tobacco smoking and thyroid function and thyroid autoimmunity are still controversial. Smoking has shown to have variable effects on thyroid function reflected by increase or decrease in serum thyrotropine (TSH) or thyroxine (T4) and triiodotironin (T3) levels. Several population based studies, especially in iodine deficient areas, have reported lower levels of serum TSH and higher levels of thyroid hormones in smokers than non smokers suggesting a stimulating effect of tobacco smoke on thyroid gland (Asvold *et al*., 2007) [1].
TSH is the major regulator of the morphologic and functional states of the thyroid. All steps in the formation and release of thyroid hormones are stimulated by TSH secreted by the pituitary thyrotrophs. Thyroid cells express the TSH receptor (TSHR), a member of the glycoprotein G protein–coupled receptor family. It is a glycoprotein secreted by the thyrotrophs in the anteromedial portion of the adenohypophysis. In normal serum, TSH is present at concentrations between 0.4 and 4.2mU/L. The level is increased in primary hypothyroidism and reduced in thyrotoxicosis. The plasma TSH half-life is about 30 minutes, and production rates in humans are 40 to 150mU/day.
Thiocyanate (SCN-), a major component of smoke, derived from hydrogen cyanide, a perchlorate like goitrogen is generated from cigarette smoke as a detoxifying product of cyanide.

The results of most of the studies have shown that smokers have higher prevalence of hyperthyroidism than hypothyroidism. This study will contribute to the existing knowledge of the detrimental effects of tobacco smoking on thyroid function. Thus, this study may be instrumental in early detection and management of thyroid disease in smokers.

This present study will contribute to the existing knowledge of the detrimental effects of tobacco smoking on thyroid function. Thus, the study was planned to analyse the alteration in thyroid profile of smokers in comparison with non-smokers and thus may be instrumental in early detection and management of thyroid disease in smokers.

Materials and Methods

Settings

A prospective study was conducted in the Department of Biochemistry, RIMS General Hospital, KADAPA.

Sources of samples and data

The cases and samples were collected from Department of General Medicine n& Department of Biochemistry, RIMS and general Hospital KADAPA.

Cases

Healthy male smokers, a brief history was taken and samples were collected.

Investigations were performed at the Department of Biochemistry, RIMS/ General Hospital, KADAPA.

Controls

Healthy male non-smokers, a brief history was taken and samples were collected and investigations were performed at the Department of Biochemistry, RIMS/ General Hospital, KADAPA.

In the present study the individuals were divided into two groups.

Table 1: In the present study the individuals were divided into two groups.

Group 1	Healthy male non - smokers	N = 30
Group 2	Healthy male smokers	N = 30

Informed oral and written consent was taken from all individuals who took part in the study.

Inclusion criteria

1) Smokers 2) No history of thyroid disease 3) Healthy males 4) No history of any steroids medication 5) No history of liver disease 6) Age between 25 to 50 years 7) No pituitary disorders.

Exclusion criteria

1) Non – smokers 2) History of thyroid disease 3) History of consumption of steroids medication 4) History of liver disease 6) Age less than 25 years and above 50 years 7) Patients with pituitary disorder

Specimen Collection

4ml of Fasting venous blood were collected in a vacutainer (RED CAP). Sample was centrifuged at 3000 r.p.m for 10 minutes and serum was separated for analysis within two hours of collection of blood. Grossly haemolysed and lipemic samples were excluded.

Ethical Clearence

The ethical issues involved in this study were reviewed and approved by the ethics scientific committee of Rajiv Gandhi Institute of Medical Sciences, KADAPA.

Statistical analysis

The data was analysed using SPSS

Methodology

TSH (Thyroid stimulating hormone) ELISA (CALBIOTECH KIT) – TS227T [145]

Results

The present study was undertaken in the department of Biochemistry RIMS/ General Hospital. A total of 60 male subjects of 30 subjects were smokers and 30 were non-smokers. The results were expressed in µIU/ml for serum TSH. The Mean ± SD of all the parameters studied in the total cases were significantly different from those of controls. The significance of different mean values of different groups is represented by P values and P values < 0.05 is considered as significant.

Table 2: Mean ± SD of TSH levels in smokers and Non smokers

Parameter	Group 1 (Control)			Group 2 (Cases)		
	Mean	±S.D		Mean	±S.D	
TSH*	2.80	0.83		1.00	1.02	

Table 3: Age of total study population (N = 60) i.e Group 1 and 2

Minimum Age	Median Age	Maximum Age
26.00	32.50	46.00

Thus the age range of the total study population is between 25 – 48 years

Table 4: Age of smokers in the smoking population study i.e Group 2

Minimum Age	Median Age	Maximum Age
26.00	33.00	46.00

Table 5: Age of non-smokers in the non-smoking population study i.e Group 1

Minimum Age	Median Age	Maximum Age
26.00	31.00	44.00

Table 6: Number of cigarettes smoked per day in a study population

Minimum	Maximum	Mean ± SD
4.00	42.00	10.12 ± 5.82

Table 7: Duration in years of cigarette smoking in a study population

Minimum	Maximum	Mean ± SD
4.00	24.00	13.02 ± 4.36

Table 8: Abnormal thyroid Results among smoker population

	No. of Positive cases	Percentage %
TSH Abnormal	20	66.66

Results

Non-smokers had TSH within normal limits. In this study we analyzed data on 82 men, of whom 41 were smokers and 41 were non-smokers.

In this study the age range for the study population was 26 to 46 years. The minimum age is 26.00 years, median age is 33.00 years and the maximum age is 46.00 years of the total study population respectively. The numbers of cigarettes smoked in a smoking population is minimum 4 per day and maximum 42 per day and the duration of smoking was minimum of 4 years and maximum of 24 years.

In this study the Mean ± SD of TSH was lower for smokers than non-smoker. The mean and median of TSH value in this study population is 2.712 (Group 2 Non smokers) and 1.003 (smokers) respectively.

Among 30 smokers (Group 2) 20 cases were positive for abnormally low TSH with a percentage of 66.66% is statistically significant.

Thus this study shows that is TSH is lower whereas T3 and T4 levels are higher among the smokers hence indicating hyperthyroidism.

Discussion

In this study we have found that tobacco smoking is associated with lower serum TSH values and lower risk of hypothyroidism.

In the present study, we compared the Mean ± SD of serum TSH levels between the two groups. Serum TSH in Group 1 was 2.80 ± 0.83 and Group 2 was 1.00 ± 1.02. The decrease in TSH levels in group 2 was significant ($p<0.0001$). This implies that the males who smoke have low levels of serum TSH as compared to non-smoking males. Due to multiple contrasting pathways in which tobacco smoke can effect in the functioning of thyroid gland. The decrease in serum TSH was probably due to thiocyanate, 2, 3-hydroxypyridine, 3, 4-benzpyrene and hydroxyquinones, present in tobacco smoke SCN− inhibits iodide transport into the thyroid gland. This may cause a relative ID state in the gland which may be responsible for the goitrogenic effect of cigarette smoking. However, nicotine-dependent stimulation of the sympathetic nervous system, which in turn stimulates the thyroid gland and enhances the secretion of thyroid hormones, might also be responsible for the lower TSH levels in smokers.

Slightly reduced serum TSH concentrations in smokers have been found in many previous (Asvold *et al*., 2007) [1] and a few recent studies such as, the 5 th Tromo Study conducted on 6 000 subjects in Norway (Jorde and Sundsfjord, 2006) [6], NHANES III study on 15 000 subjects in USA (Belin *et al*., 2004) [2] and another study by Knudsen *et al*. on over 4 000 subjects in Denmark (Knudsen *et al*., 2002) [5]. However, some studies have reported no association between serum TSH levels and smoking (Christensen *et al*., 1984) [3]. The mechanism behind lower serum TSH levels in smokers could be due to nicotine induced sympathetic stimulation, or increased evolution of thyroid autonomy, caused by the iodine depletion effect of thiocyanate in the thyroid (Pontikides and Krassas, 2002) [9]. Nicotine induces sympathetic activation which can increase thyroid hormone secretion and it may also have a direct thyroid stimulatory action along with other components of tobacco smoke like benzpyrene (Utiger, 1995) [7]. Serum thiocyanate concentration is higher in smokers than non-smokers (Foss and Lund-Larsen, 1986 [10]. However it inhibits iodine uptake and thyroid hormone synthesis and also increases the efflux of iodine from thyroid gland, the effect of which is not compatible with the stimulatory effect of tobacco smoke on thyroid gland, reported by a few studies (Tonacchera *et al*., 2004; Fukayama *et al*., 1992) [11, 8].

In addition to TSH, T3 and T4 for predicting thyroid disorders in smokers, other biomarker such as thyroid peroxidase antibody (TPO-Ab) thyroglobulin antibody (Tg-Ab) and thiocyanate (SCN^-) can be used for predicting thyroid disorder in smokers.

Conclusion

In conclusion, smoking is associated with decreased serum TSH levels, lower risk of hypothyroidism and possibly lower frequency of thyroid auto immunity, a finding which helps the physician in important decision making in the treatment of subjects with subclinical hypothyroidism. Therefore, it seems that more care should be taken to avoid hypothyroidism in individuals already at cardiac risk from smoking by adjusting normal TSH ranges in smokers.

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Conflict of interest: The author have no any conflict of interest in this study.

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