

Effect of Thyroid on Lipid Profile and Renal Function: An Observational Study from Tertiary Care Centre of Tribal Region of Bastar

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Abstract

Background: Thyroid hormone is a key substance in normal homeostasis, having variable influence on cell metabolism on different organs. Very little is known about the prevalence of thyroid disorders from our region. **Aim:** This study was conducted with the aim of finding prevalence of thyroid disorder and relation of thyroid hormone with renal function and cholesterol metabolism. **Subjects and Methods:** A total of 96 ambulatory patients were taken for study. Serum samples were collected and evaluated for triiodothyronine, thyroxine, thyroid-stimulating hormone, urea, creatinine, total cholesterol, triglyceride (TG), low density lipoprotein (LDL), very low density lipoprotein and high density lipoprotein (HDL). Analysis of variance and *t*-test were used to find a significant difference among the groups. **Results:** Prevalance of thyroid disorder among suspected patients was 64/96 (66%), of which 36/64 (56.3%) were hypothyroid and 28/64 (43.8%) were hyperthyroid. No relation was found with renal function, but cholesterol was found high (>250 mg/dl) among hypothyroid patients and significant increase in TG, LDL levels and significant decrease was in HDL. **Conclusion:** Thyroid disorder is high among subjects with hypercholesterolemia. This underscores the need to evaluate for thyroid disorder in hypercholesterolemic patients and vice-versa.

Keywords: Hypothyroidism, Hyperthyroidism, Creatinine, Serum cholesterol, Tribal, Urea

Introduction

Thyroid diseases are arguably, among the most common endocrine disorders world-wide. India too is no exception. According to a projection from various studies on thyroid disease, it has been estimated that about 42 million people in India suffer from thyroid diseases.^[1] Thyroid hormones (TH) regulate the renal hemodynamics and basal metabolic rate of most cells. The thyroid gland synthesizes and releases triiodothyronine (T3) and thyroxine (T4), which represent the only iodine containing hormones in the vertebrates. T3 is the biologically active thyroid hormone.^[2] These hormones are required for the normal growth, development and function of nearly all tissues, with major effects

on oxygen consumption and metabolic rate.^[3] TH synthesis and secretion is regulated by a negative feedback system that involves the hypothalamus, pituitary and the thyroid gland.^[4] THs regulate the basal metabolic rate of all cells including hepatocytes and hence, modulate hepatic function; the liver in turn metabolizes the thyroid hormones and regulates their systemic endocrine effects.^[5] Normal circulating levels of thyroid hormone are required for both normal hepatic circulation and normal bilirubin metabolism.^[6] Thyroid dysfunction may perturb liver function and vice-versa.^[5] In experimental animals, surgical or drug-induced hypothyroidism of a few weeks duration has been shown to result in a decrease in glomerular filtration rate.^[7,8] However, clinical studies on hypothyroid subjects are very few and not much data is available on how hypothyroidism influences renal function in human beings. Hence, we conducted this observational study to see the relation of the thyroid hormone with hepatic and renal functions.

Subjects and Methods

The study was conducted in Department of Biochemistry in a tertiary care center in Bastar area (tribal area) of Chhattisgarh,

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India. Study was carried out between April to September 2012 96 (75 female and 21 males) ambulatory patients between ages 15 and 55 presenting to various departments suspected of thyroid disorder visiting for the first time were screened. Blood pressure was not a desired parameter in our study. Brief clinical history and examination along with some epidemiological data were taken. After a written and informed consent samples were collected and processed. Approval from ethical committee was taken prior to study.

Collection and preparation of sample

We collected 5 ml of venous blood with full aseptic precautions without anticoagulant and allowed it to clot. Clotted blood was centrifuged and clear serum was collected. Fresh serum samples were taken. Serum was checked for hemolysis and if hemolyzed then that serum was discarded. Serum was analyzed for T3, T4 and thyroid-stimulating hormone (TSH) for thyroid profile. Serum having abnormal thyroid levels was also analyzed for urea, creatinine and cholesterol to find out any other disorder associated with abnormal thyroid status. Serum for analysis was stored at -20°C . Thawed samples were mixed prior to testing.

Analytical methods

A total volume of 50 μl of serum was taken to analyze T3, 25 μl for T4 and 100 μl for TSH hormone level by the enzyme-linked immunosorbent assay method (Omega diagnostics) at 450 nm filter using microplate reader model 680 (Biorad). Normal range for T3 was 0.5-1.9 ng/ml, for T4 was 4.8-11.6 pg/dl (females) and 4.4-10.8 pg/dl (males) and for TSH it was 0.3-6.3 $\mu\text{IU/ml}$.

Serum urea was measured using glutamate dehydrogenase-urease method in which urease hydrolyses urea to ammonia and ammonia formed further combines with alpha-ketoglutarate and NADH (reduced form of nicotinamide adenine dinucleotide [NAD]) to form glutamate and NAD, rate of oxidation of NADH to NAD is measured as a decrease in absorbance in a fixed time, which is proportional to urea concentration in the sample and optical density was taken at 340 nm. Serum creatinine was measured using Jaffe's method in which creatinine reacts with picric acid in alkaline medium to form an orange colored complex and rate of change of absorbance is measured at 505 nm using semi-auto analyzer. Serum cholesterol was measured using cholesterol oxidase (CHOD)- peroxidase (POD) method in which cholesterol ester in presence cholesterol esterase forms cholesterol, this free cholesterol in the presence of CHOD forms cholest-4-en-3-one and H_2O_2 , this H_2O_2 in the presence of 4 aminoantipyrine and phenol forms quinone imine and absorbance of quinone imine so formed is directly proportional to cholesterol concentration and optical density was taken at 505 nm. Low density lipoprotein (LDL) cholesterol was measured using direct reagent kit in which LDL is measured in serum without the need for any offline pre-treatment

or centrifugation, reagent consists of a reagent capable of solubilizing LDL specifically. Cholesterol esterase and chromogenic coupler react with this solubilize LDL to develop color, which is directly proportional to the concentration of LDL. High density lipoprotein (HDL) cholesterol was measured using Phosphotungstic method, in which chylomicrons, LDL and very low density lipoprotein (VLDL) are precipitated from serum by phosphotungstate in the presence of divalent cation such as magnesium, the HDL cholesterol remains unaffected, which is later on estimated CHOD-POD method. Results were expressed as mg/dl. Normal range for urea was 15-45 mg/dl, for creatinine was 0.5-1.5 mg/dl for cholesterol was 150-250 mg/dl, for TG was 35-165 mg/dl, for LDL was <150 mg/dl, for VLDL was 7-33 mg/dl and for HDL was 30-70 mg/dl. All these were measured using Semi-auto analyzer. Analysis of variance and *t*-test were used to find a significant difference among the groups. Statistical Package for the Social Sciences software, version 17.0 (Chicago IL, USA) was used for statistical analysis.

Results

A total of 96 patients were screened and 64 were having thyroid disorder. Patients of various age groups were selected with a range of 15-55; out of those 56.3% (36/64) were hypothyroid and 43.8% (28/64) were hyperthyroid [Tables 1 and 2]. Out of 36 hypothyroid patients 72.2% (26/36) were female and 27.7% (10/36) were male and out of 28 hyperthyroid 78.6% (22/28) were female and 21.4% (6/28) were male [Table 3]. Urea and creatinine were in normal range in all

Table 1: Thyroid status of study subjects

Thyroid status	No.	%
Euthyroid	32	33.3
Hyperthyroid	28	29.2
Hypothyroid	36	37.5
Total	96	100.0

Table 2: Levels of T3, T4 and TSH among study subjects

Thyroid profile	Euthyroid (n=32)	Hyperthyroid (n=28)	Hypothyroid (n=36)
T3	1.2 (0.3)	2.7 (0.5)	0.3 (0.1)
T4	7.7 (1)	18.9 (4.1)	2.6 (1)
TSH	2.7 (0.5)	0.2 (0.1)	18 (6.7)

TSH: Thyroid-stimulating hormone

Table 3: Characteristics of study subjects

Specification	No	%
Age		
<35	47	49.0
≥ 35	49	51.0
Sex		
Male	21	21.9
Female	75	78.1

thyroid disorder patients [Table 4]. Cholesterol was found high in 52.8% (19/36) individuals having hypothyroid disorder. Serum total cholesterol was found to be significantly high ($P < 0.001$) in hypothyroid individuals when compared with euthyroid individuals [Table 4]. Serum triglyceride (TG) and LDL were also found to be significantly raised in hypothyroid individuals ($P = 0.01$ and $P = 0.01$ respectively) [Table 4]. Serum HDL was also found to be lowered in hypothyroid individuals ($P < 0.001$) [Table 4]. Both TG and LDL are equally affected in hypothyroid individuals, but HDL is affected the most and least effect on VLDL component. In hyperthyroid individuals, TG and LDL was found to be significantly lower when compared with euthyroid individuals ($P < 0.01$ and $P = 0.02$ respectively) [Tables 4 and 5].

Discussion

The present study is the first description of thyroid disorder status in and around this region among tribal patients. Thyroid disorder level is high among native tribal of Bastar region involving more commonly female population. The reason for this is not well-known. This may be because Bastar region receives heavy rainfall and it is situated well above plains so

iodine of the superficial layer gets washed away with water and here consumption of seafood, which is rich in iodine, is also less as this area is far from the sea. Ground water of some regions in Bastar is rich in fluorine to the level that it may cause fluorosis too. It has long been suggested that dental fluorosis is associated with iodine deficiency disorder (IDD) and thyroid dysfunction.^[9-12] Fluoride itself has been effectively used as an anti-thyroid drug.^[13] The history of fluoride/iodine antagonism has already been established.^[12] TH deficiency and/or excess arising from fluoride toxicity leading to IDD such as low intelligence quotient, deaf-mutism and cretinism in children have been reported from elsewhere.^[14,15] Hypothyroid state is having more impact on cholesterol metabolism as indicated in our study, which may result in high cholesterol state. This result matches with the study of Rizos *et al.*,^[16] but different from the study of Langer *et al.*^[17] We found no role of thyroid hormone on renal function. Some studies Iglesias *et al.*, have shown thyroid role in renal dysfunction.^[18]

Conclusion

Hypothyroid state is having a role in increased cholesterol, i.e., hepatic metabolism which in turn is responsible for

Table 4: Association of serum cholesterol, TG, LDL, VLDL, HDL, creatinine and urea with thyroid status

Specification	Mean	SD	SE	95% CI for Mean		Test of significance
				Lower bound	Upper bound	
S. Cholesterol						
Euthyroid	200.1	24.5	4.3	191.3	209	($t=2.2$, $df=58$, $P=0.03$)*
Hyperthyroid	185.3	26.7	5	175	195.7	($t=9.4$, $df=66$, $P<0.001$)**
Hypothyroid	254.1	22.7	3.8	246.4	261.8	
S. TG						
Euthyroid	104.4	31.6	5.6	93.1	115.8	($t=3.1$, $df=58$, $P<0.01$)*
Hyperthyroid	80.8	26.2	5	70.6	90.9	($t=2.5$, $df=66$, $P=0.01$)**
Hypothyroid	123.1	29.7	5	113	133.1	
S. LDL						
Euthyroid	103.1	22	3.9	95.1	111	($t=2.3$, $df=58$, $P=0.02$)*
Hyperthyroid	90.7	18.8	3.6	83.4	98	($t=2.4$, $df=66$, $P=0.01$)**
Hypothyroid	117.5	26.9	4.5	108.4	126.6	
S. VLDL						
Euthyroid	22.4	5.6	1	20.4	24.4	($t=1$, $df=58$, $P=0.31$)*
Hyperthyroid	20.9	5.8	1.1	18.7	23.2	($t=0.8$, $df=66$, $P=0.41$)**
Hypothyroid	23.6	6.2	1	21.5	25.7	
S. HDL						
Euthyroid	52.7	10.8	1.9	48.8	56.6	($t=1.6$, $df=58$, $P=0.10$)*
Hyperthyroid	47.8	12.6	2.4	42.9	52.6	($t=3.9$, $df=66$, $P<0.001$)**
Hypothyroid	40	15.2	2.5	34.9	45.1	
S. Urea						
Euthyroid	27.4	7.2	1.3	24.8	30	($t=1.4$, $df=58$, $P=0.17$)*
Hyperthyroid	30.4	9.4	1.8	26.7	34	($t=0.7$, $df=66$, $P=0.47$)**
Hypothyroid	28.8	9	1.5	25.8	31.9	
S. Creatinine						
Euthyroid	1	0.3	0.05	0.9	1.1	($t=0.1$, $df=58$, $P=0.91$)*
Hyperthyroid	1	0.3	0.05	0.9	1.1	($t=0.3$, $df=66$, $P=0.79$)**
Hypothyroid	1	0.4	0.1	0.9	1.2	

*Eu and Hyper, **Eu and Hypo, S. TG: Serum triglyceride, S. LDL: Serum low density lipoprotein, S. VLDL: Serum very low density lipoprotein, S. HDL: Serum high density lipoprotein, S. Cholesterol: Serum cholesterol, S. Urea: Serum urea, S. Creatinine: Serum creatinine, SD: Standard deviation, SE: Standard error, CI: Confidence interval

Table 5: ANOVA					
Group accessed	Sum of squares	Df	Mean square	F	P value
S. Cholesterol					
Between groups	86629.5	2	43314.7	72.0	<0.001
Within groups	55944.4	93	601.6		
S. TG					
Between groups	28189	2	14094.5	16.3	<0.001
Within groups	80427	93	864.8		
S. LDL					
Between groups	11432.7	2	5716.3	10.6	<0.001
Within groups	49984.6	93	537.5		
S. VLDL					
Between groups	111	2	55.5	1.6	0.20
Within groups	3224.3	93	34.7		
S. HDL					
Between groups	2802.3	2	1401.1	8.2	<0.001
Within groups	15945.7	93	171.5		
S. Urea					
Between groups	130.1	2	65.1	0.9	0.40
Within groups	6823.1	93	73.4		
S. Creatinine					
Between groups	0.01	2	0.0	0.03	0.96
Within groups	9.5	93	0.1		

S. TG: Serum triglyceride, S. LDL: Serum low density lipoprotein, S. VLDL: Serum very low density lipoprotein, S. HDL: Serum high density lipoprotein, S. Cholesterol: Serum cholesterol, ANOVA: Analysis of variance

complications of high blood cholesterol viz. hypertension, Cardiovascular disease etc., So every hypercholesterolemic patient should be evaluated for thyroid disorder and vice-versa.

References

1. Available from: <http://www.ias.ac.in/currsci/oct252000/n%20kochupillai.PDF>. [Last accessed on 2011 Apr 2].
2. Boelaert K, Franklyn JA. Thyroid hormone in health and disease. *J Endocrinol* 2005;187:1-15.
3. Yen PM. Physiological and molecular basis of thyroid hormone action. *Physiol Rev* 2001;81:1097-142.
4. Shupnik MA, Ridgway EC, Chin WW. Molecular biology of thyrotropin. *Endocr Rev* 1989;10:459-75.

5. Malik R, Hodgson H. The relationship between the thyroid gland and the liver. *QJM* 2002;95:559-69.
6. Youssef WI, Mullen KD. The liver in other (nondiabetic) endocrine disorders. *Clin Liver Dis* 2002;6:879-89.
7. Davis RG, Madsen KM, Fregly MJ, Tisher CC. Kidney structure in hypothyroidism. *Am J Pathol* 1983;113:41-9.
8. Zimmerman RS, Ryan J, Edwards BS, Klee G, Zimmerman D, Scott N, *et al.* Cardiorenal endocrine dynamics during volume expansion in hypothyroid dogs. *Am J Physiol* 1988;255:R61-6.
9. Stocks P. Goitre in the English school child. *Q J Med* 1928;21:223.
10. Wilson DC. Distribution of fluorosis in India and in England. *Nature* 1939;144:155.
11. Wilson DC. Fluorine in the etiology of endemic goiter. *Lancet* 1941;i: 211-2.
12. Parents of Fluoride Poisoned Children (PFPC) [Homepage on the Internet]. History of the fluoride iodine antagonism. ©1996-2005. Available from: http://64.177.90.157/pfpc/html/thyroid_history.html. [Updated frequently; Last cited on 2005 Mar 30].
13. Gallerti P. On the use of fluoride to treat overactive thyroid. *Fluoride* 1976;9:105-15.
14. He H, Chen ZS, Liu XM. The influence of fluoride on human embryo. *Chin J Control Endem Dis* 1989;4:136-7.
15. Du L, Wan CW, Cao XM. The influence of chronic fluorosis on the development of the brain of the embryo. *J Fluorosis Res Commun* 1991;138.
16. Rizos CV, Elisaf MS, Liberopoulos EN. Effects of thyroid dysfunction on lipid profile. *Open Cardiovasc Med J* 2011;5:76-84.
17. Langer P, Kocan A, Tajtakova M, Petrik J, Koska J, Hucková M, *et al.* Thyroid function and cholesterol level: Paradoxical findings in large groups of population with high cholesterol food intake. *Endocr Regul* 2003;37:175-80.
18. Iglesias P, Díez JJ. Thyroid dysfunction and kidney disease. *Eur J Endocrinol* 2009;160:503-15.

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