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Original Research Article

To study the thyroid status in type 2 DM and its possible relation with hyperglycaemia, obesity and dyslipidemia

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Abstract: The coexistence of diabetes mellitus (DM) with hypothyroidism is a known clinical observation. Patients with diabetes mellitus may be at an increased risk of thyroid disease and further thyroid abnormalities can have adverse effect of metabolic control, dyslipidemia and cardiovascular risk. Poor glycemic control can produce features similar to hyperthyroidism, such as weight loss despite increased appetite and fatigue and clinical features of hypothyroidism also overlap with diabetes. In view of the frequency both of type 2 diabetes mellitus and thyroid disease and their significant interrelationship and clinical implications, this study proposed to evaluate the prevalence of thyroid abnormalities in T2DM patients and possible relations between thyroid hormones, obesity, glycaemic control and lipid profile parameters. The study was conducted in the Department of Biochemistry and Medicine, BPS Govt Medical College. Hundred confirmed cases of type-2 DM were taken into study. Hundred healthy age/sex matched individuals were taken as control. BMI, waist hip ratio, lipid profile parameters and HbA1c were significantly higher in study group reflecting the obesity, dyslipidemia and poor glycemic control in the study group patients. Prevalence of abnormal thyroid profile was 19% (28.3% in females and 8.5% in males). Subclinical hypothyroidism was observed to be the most common thyroid abnormality (47.4%), followed by subclinical hyperthyroidism (31.6%) and hypothyroidism (21.1%). Thyroid hormone levels (TSH, FT3, FT4, T3, T4) were significantly lower than in control though mean±SD were in the euthyroid range. Thyroid hormones were significantly correlated with BMI, HbA1c, Triglycerides and HDL reflecting their effect on glycemic control and lipid profile. Given the significant prevalence of thyroid abnormalities in type 2 diabetics and their significant correlation with HbA1c and lipid parameters observed, it will be beneficial clinically to assess thyroid hormones in type 2 diabetics as failure to recognize the presence of abnormal thyroid hormone levels in diabetes may be a primary cause of poor management often encountered in some treated diabetics. Further investigations in larger number of subjects with longitudinal serial testing are needed to understand the intimate mechanisms of lipid and glucose metabolism in type 2 diabetes with respect to thyroid function.
Keywords: diabetes mellitus (DM), hypothyroidism.

INTRODUCTION

Diabetes mellitus and thyroid diseases are the two common endocrinopathies seen in the adult population. Al-Attas *et al.* found significantly lower concentration of urine iodine in T2DM along with negatively correlation of urinary iodine with obesity, glucose, insulin, triglyceride and resist in while it was positively associated with TSH [1]. Clinical evidences have also reported subclinical alterations of thyroid function in obesity, although the relationship between thyroid status and obesity remains unclear. Central obesity was found to be the most powerful predictor of TSH [2]. The prevalence of subclinical hypothyroidism (a raised serum TSH and normal serum free T4) was 8.6% and subclinical hypothyroidism was noted to be a common but incidental finding in the study by Chubb *et al.* [3]. In metabolically stable diabetics, basal TSH levels and TSH response to thyrotropin-releasing hormone (TRH) are essentially normal. However, poorly controlled diabetic patients may show impaired TSH response to TRH or loss of the normal nocturnal TSH peak [4]. In an animal study on feline cats before and after the development of obesity, obesity was associated with a significant increase in FT4 within the normal range. Fatty acids were proposed to inhibit the cellular uptake of TH and/or pituitary TH receptor binding, leading to TH resistance. Increased leptin may also alter sensitivity to negative feedback of TH [5]. Resistin has been suggested to be the link between obesity and type 2 diabetes. Resistin is also regulated by thyroid hormones; it is severely decreased in hyperthyroid rats [6].

Type 2 diabetic patients with subclinical hypothyroidism are associated with an increased risk of nephropathy and cardiovascular events, but not with retinopathy [7]. Further depression a feature shown to be associated with type 2 diabetes is also a clinical feature of hypothyroidism [8]. In the context of chronic hyperglycaemia, low free T4 within the euthyroid range confers diminished HDL antioxidative capacity, a pathophysiologically relevant metric of HDL functionality [9]. Patients with diabetes mellitus may be at an increased risk of thyroid disease and further thyroid abnormalities can have adverse effect of metabolic control, dyslipidemia and cardiovascular risk. In the cross-sectional analysis by Chen *et al*., subclinical hypothyroidism was associated with a greater prevalence of diabetic nephropathy compare to euthyroid diabetics [7]. Poor glycemic control can produce features similar to hyperthyroidism, such as weight loss despite increased appetite and fatigue. On the other hand, severe diabetic nephropathy can be mistaken for hypothyroidism because patients with this condition may have edema, fatigue, pallor, and weight gain. Subclinical hypothyroidism can elevate serum LDL cholesterol and worsen pre-existing dyslipidemia [10,11].

Udiong *et al*. in a study conducted in Nigeria described that TSH levels in diabetics were significantly lower than the levels of non-diabetic controls. Male diabetics had lower levels of TSH than diabetic females. The level of T_3 in diabetic males was higher than the level in females. 26.6% had low plasma thyroid hormone levels, 19.8% had raised plasma thyroid levels and approximately 54% were euthyroid. A high incidence of 46.5% of abnormal thyroid hormone levels among the diabetics (hypothyroidism 26.6%, hyperthyroidism 19.9%) was seen. The prevalence of hypothyroidism was higher in women (16.8%) than in men (9.9%), while hyperthyroidism was higher in males (11%) than in females (8%) [12]. Celani *et al* described abnormal TSH concentrations were detected in 31.4% of the patients of type 2 diabetes screened. Subclinical hypothyroidism (high TSH, normal FT_4) was most (48.3%), followed by subclinical hyperthyroidism (low TSH, normal FT_4) (24.4%) and by definite hypothyroidism (high TSH, low FT_4) in 23.15% of patients [13]. Kabadi *et al*. described that serum T_3 levels declined and rT_3 levels rose in diabetic patients with worsening of metabolic control [11]. After two year Kabadi did another study and concluded that serum T_3 levels were significantly lower and rT_3 (reverse T_3) significantly higher in subjects prior to the treatment as compared to normal subjects. Both T_3 and rT_3 were normalized when adequate metabolic control was achieved after the treatment, (as reflected by normalization of HbA1c) [14]. Schlienger *et al*. described in type 2 diabetes subjects, a significant decrease in T_3 and rise in reverse T_3 whereas T_4 was normal. In the poorly controlled diabetics (HbA1c ≥ 12%), T_3 was 90±5 ng/dl, which differed significantly from the level found in the better controlled patients (106±5 ng/dl). Negative linear correlation was found between T_3 and HbA1c and a positive correlation was found between reverse T_3 and HbA1c [15]. Maskey *et al*. in a study of 271 subjects in Nepal described the prevalence of hypothyroidism (clinical and subclinical) in diabetics as 4.05% with female preponderance. Of the patients with abnormal thyroid profile 30.4% were clinically hypothyroid and 17.4% were subclinical hypothyroid. 4.3% patients had subclinical hyperthyroidism. High-density lipoprotein among different thyroid status was statistically significant [16]. In the study of 120 T2DM patients at Hyderabad, hypothyroidism was seen in 32 (27%, 10% being subclinical), of which 80% were females. 70% of patients with hypothyroidism were between 40 and 60 years age [17].

Islam *et al*., Ardekani *et al*., Alagia *et al*. in separates studies showed that the levels of thyroid hormone were not significantly different from levels in non-diabetic controls [18-20]. Duran *et al*. described that the median total thyroid volume for patients with DM was significantly higher. Thyroid volume was significantly correlated with age ($r=0.92$, $p<0.001$) and TSH ($r=0.435$, $p<0.001$) [21]. Zhang *et al* concluded that TSH was positively associated with serum TC and LDL-C in euthyroid diabetic women [22].

In view of the frequency both of type 2 diabetes mellitus and thyroid disease and their interrelationship this study was proposed to evaluate the prevalence of thyroid abnormalities in T2DM patients and possible relations between thyroid hormones, obesity, glycaemic control and lipid profile parameters.

METHODOLOGY

Hundred confirmed cases of type2 diabetes mellitus in the age group of 30 to 75 years attending or admitted in the Department of Medicine were enrolled into the study. Hundred healthy age/sex matched individuals were taken as control. Ethical clearance was taken from the Institutional Ethical Committee. Informed consent was taken from all the patients and controls after explaining the details of the study in the local language. Criteria for the diagnosis of diabetes as per the American Diabetic Association are [23]:

1. $HbA1_C$ ≥ 6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.
   OR
2. FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hrs.

OR

3. 2-hrs plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

OR

4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥ 200 mg/dL (11.1 mmol/L). Random is defined as without regard to time since the last meal.

In the absence of unequivocal hyperglycemia, criteria 1–3 should be confirmed by repeat testing. (NGSP- National Glycohemoglobin Standardization Program, DCCT - Diabetes control and complications trial)

Exclusion Criteria used were:

1. History of total/ subtotal thyroidectomy, patients on I^{131} treatment, lithium, antithyroid drugs, diagnosed cases of Grave's disease, toxic multinodular goiter, toxic adenoma, carcinoma patients, gestational hyperthyroidism patients
2. Patients with history of chronic renal failure or radiation exposure
3. Patients with known liver, kidney or other acute and chronic diseases like tuberculosis etc.
4. Patients with acute or chronic complications of DM
5. Patients taking medications known to influence thyroid status

History was taken from all diabetic patients and control subjects and complete general and systemic physical examination was performed. All patients and controls were subjected to anthropometric measurements, routine and special investigations. Anthropometry included measurement of weight, height, waist circumference, hip circumference, BMI and waist hip ratio. Routine investigations included haemoglobin, total leukocyte count, blood urea, serum creatinine, fasting and postprandial blood glucose levels, uric acid and liver function tests. Special investigation included glycosylated haemoglobin, T3, T4, FT3, FT4, TSH and lipid profile [TG, TC, HDLC, LDLC, VLDLC].

Specimen Collection and testing

5ml overnight fasting blood sample was collected from the antecubital vein aseptically without anticoagulant and allowed to clot. Serum was separated by centrifugation at (3000 rpm for 15mins) of the sample and used for the assays (sample was stored at 2 to 8^0 C for 1day, and at 20^0C if storage if required for more than 1 day). 1ml blood sample was collected in EDTA (purple vacutainer) vial separately irrespective of time and meal for estimation of glycosylated haemoglobin. Efforts were made to carry out all investigations on same day of sample collection minimizing the need for sample storage. Appropriate quality controls were carried out for all investigation which was perfomed using commercial kits on autoanalyzer and by ELISA technique. Reference Ranges of thyroid hormones and lipids used were:

THYROID HORMONES

Thyroid-stimulating hormone	0.34–5.0 mIU/L
Thyroxine, free (fT4)	9.0–24.5. pmol/L
Triiodothyronine, free (fT3)	2.4–4.2 pg/mL
Thyroxine, total (T4)	5.4–11.7 µg/dL
Triiodothyronine, total (T3)	77–135 ng/dL

LIPIDS - NCEP - ATP III guidelines

Total cholesterol	mg/dL
Desirable	**<200**
Borderline High	200–239
High	240
LDL cholesterol	mg/dL
Therapeutic option for very high risk pats.	<70
Optimal	<100
Near Optimal/ above optimal	**100–129**
Borderline high	130–159
High	160–189

Very High	≥ 190
HDL cholesterol	mg/dL
Low	<40
High	≥ 60
Triglycerides	
Desirable	**<150 mg/dL**
Borderline high	150-199
High	200-499
Very High	≥ 500

Statistical Analysis

SPSS ver. 20 was be used. Comparison of data between groups was done using Mann Whitney / 't' test for quantitative data and Chisquare test for qualitative data. Correlations between groups will be analyzed using Pearson correlation coefficient (r) formula.

OBSERVATIONS AND RESULTS

The mean age of study and control group was 51.7±9.9 and 50.5±9.7 respectively. Both study and control group had 47 males and 53 females. The findings are summarized in tables 1-4 and figures 1-4. BMI, waist hip ratio and values of triglycerides, total cholesterol, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol, fasting blood sugar and HbA1c were significantly higher in study than in control group subjects (Table 1). The prevalence of dyslipidemia was significantly higher in study group than control group for all the lipid parameters (table 2, figure 1). All the parameters of thyroid profile were significantly lower in study group (table 1, figure 2). The prevalence of thyroid disorder in diabetic patients was 19%. Primary hypothyroidism was observed in 4 patients (31.6%), subclinical hypothyroidism in 9 (47.4%) patients, and subclinical hyperthyroidism in 6 (21.1%) patients (table 3, figure 3). Significant correlation of thyroid hormones with BMI, HbA1C and lipid profile parameters in the study group (r and p values, Pearson Correaltion) are summarized in table 4 and figure 4.

Table-1: BMI, waist hip ratio (W/H ratio), routine investigations, lipid profile, blood sugar, HbA1c and thyroid hormones in study and control group (values in mean±SD, Mann-Whitney test)

	Study group	Control group	p value
BMI	27.3±2.3	23.5±2.2	<0.001
W/H ratio	0.970±0.13	0.897±0.14	<0.001
Haemoglobin (gm/dL)	11.65±1.7	12.11±1.78	0.544
TLC (/mm3)	7765±1230	7154±1342	0.144
Blood urea (mg/dL)	35.66±7.68	32.68±8.56	0.145
Serum creatinine (mg/dL)	1.09±0.28	1.03±0.24	0.543
TG (mg/dL)	199.0±67.4	152.2±33.9	<0.001
TC (mg/dL)	252.5±45.3	204.2±25.8	<0.001
HDL-C (mg/dL)	40.3±6.01	46.2±7.4	<0.001
LDL-C (mg/dL)	172.4±42.6	127.5±25.7	<0.001
VLDL-C (mg/dL)	39.8±13.5	30.4±6.78	<0.001
Fasting Blood sugar (mg/dL)	143.6±18.5	76.0±7.1	<0.001
HbA1c (%)	8.7±2.0	5.6±0.70	<0.001
Serum TSH (mIU/L)	2.52±2.45	2.82±1.01	<0.001
Serum T3 (ng/dL)	90.47±32.79	115.07±30.62	<0.001
Serum FT3 (pg/mL)	2.98±1.08	3.80±1.03	<0.001
Serum T4 (µg/dL)	7.54±1.68	8.40±1.32	<0.001
Serum FT4 (pmol/L)	15.20±3.48	16.54±2.77	0.006

Table-2: Comparison of prevalence of dyslipidemia in study and control groups

Lipid Parameter	Study Group	Control Group	p value
TG > 150 mg/dL	72%	47%	<0.001
TC >200 mg/dL	86%	53%	<0.001
HDL-C <40mg/dL	54%	22%	<0.001
LDL-C >130 mg/dL	79%	50%	<0.001

Table-3: Type of thyroid disorders in study group

Thyroid disorder	Study Group (n=100)		
	Total cases	Females	Males
Hypothyroidism (Primary)	4	3	1
Hypothyroidism (Subclinical)	9	7	2
Hyperthyroidism (Primary)	0	0	0
Hyperthyroidism (Subclinical)	6	4	2
Total	19	15	4

Table-4: Significant correlation of thyroid hormones with BMI, HbA1C and lipid profile parameters in the study group (r and p values, Pearson Correaltion)

	TSH	T3	T4	FT3	FT4
BMI		r= -0.337 p=0.001	r= -0.227 p=0.023	r= -0.314 p=0.001	
HbA1c	r= +0.372 p<0.001	r= -0.218 p=0.029		r= -0.208 p=0.039	
TG	r= +0.254 p=0.011	r= -0.272 p=0.006	r= -0.261 p=0.009	r= -0.282 p=0.004	r= -0.280 p=0.005
HDL	r= -0.258 p=0.010		r= +0.247 p=0.013		r= +0.235 p=0.005

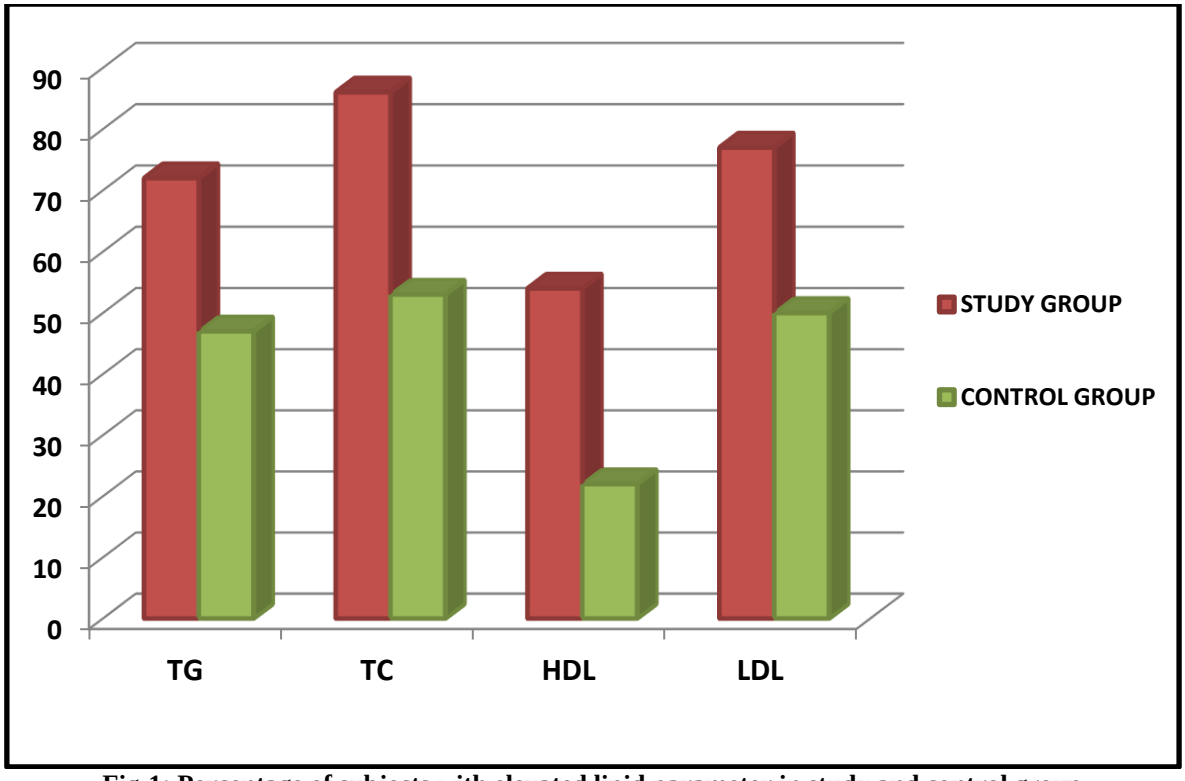

Fig-1: Percentage of subjects with elevated lipid parameter in study and control group

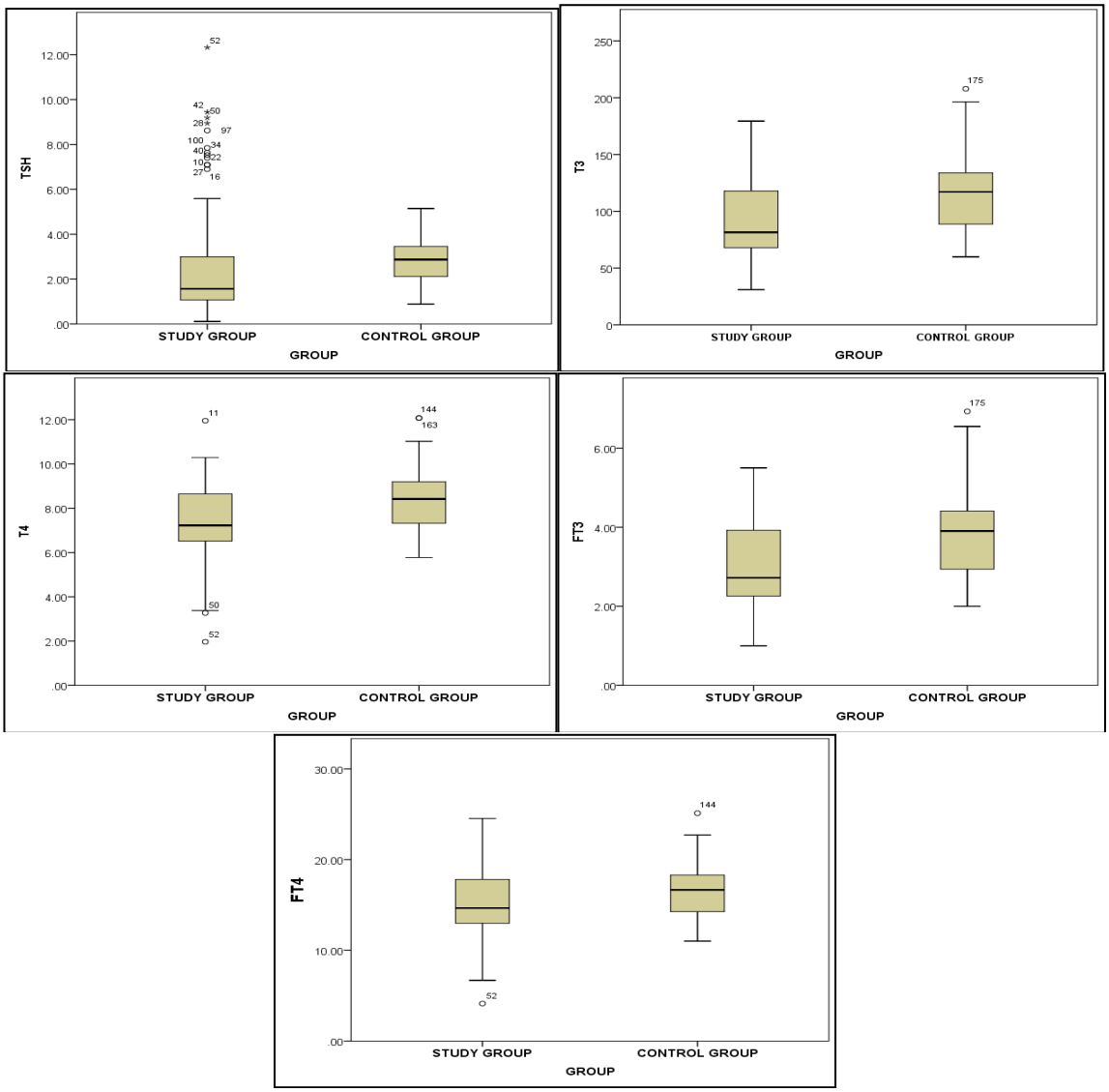

Fig-2a-2e showing the comparison of thyroid profile parameters in study and control groups

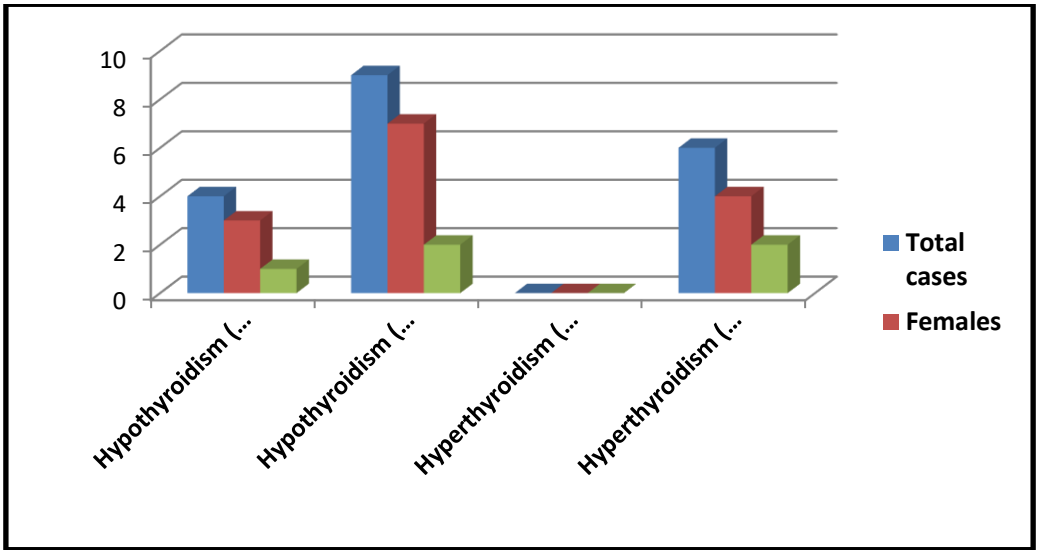

Fig-3: Distribution of thyroid profile abnormalities in study group

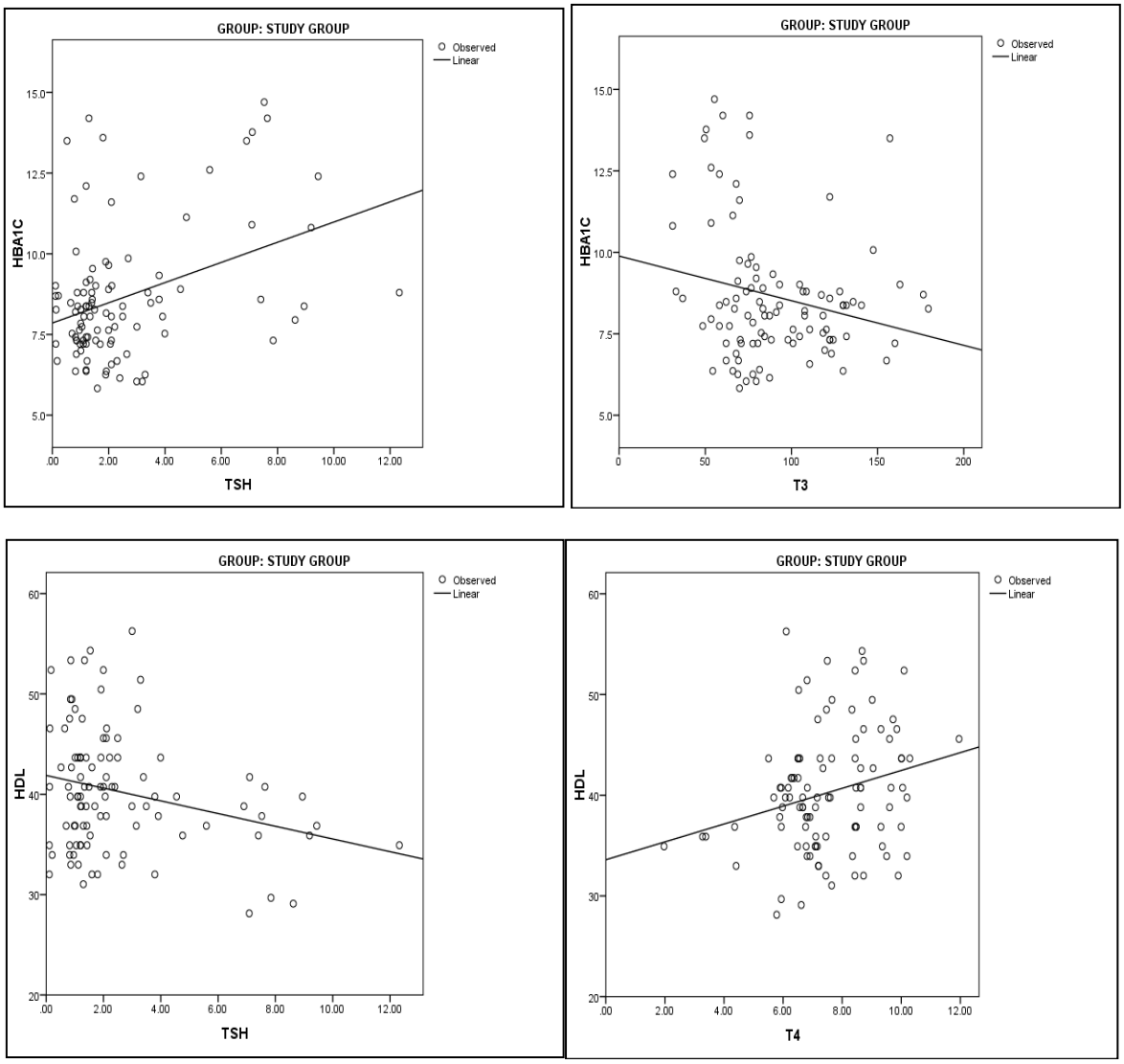

Fig-4a-4d: Correlation of HbA1c with TSH and T3 and HDL with TSH and T4

DISCUSSION

Age and gender

The study group were similar with respect to age and gender distribution of subjects (table 1).

BMI and Waist Hip ratio:

BMI and waist hip ratio (indicators of obesity) of study group was significantly higher than control group (table 1).) BMI is a measure of obesity and total body fat which is an important cause of insulin resistance [23]. BMI does not reflect body fat distribution, whereas the intra-abdominal deposition of adipose tissue is considered to be a major contributor to the development of hypertension, insulin resistance, DM and dyslipidemia. Waist-to-hip ratio (WHR) is increasingly being accepted as the better anthropometric indicator of abdominal adiposity and metabolic risk [23].

Fasting blood sugar and HbA1c

Levels were significantly higher in study diabetic group as compared to controls (table 1). These reflect the poor glycemic control and the deranged metabolic profile of the study subjects [24].

Lipid profile

The mean levels of all lipid profile parameters were significantly higher in study group than controls. HDL-C was significantly lower (table 1). The prevalence of dyslipidemia (classified as per the NCEP ATP III guidelines, given in methodology section) was also significantly higher for all the parameters in the study group (table 2 and figure 1). Dyslipidemia occurs in type 2 diabetes due to the selective insulin resistance in various pathways in the liver and adipose tissue [25].

Thyroid profile

All the parameters (TSH, T4, T3, FT4, FT3) were significantly lower in the study group than controls though the mean±SD levels were in the normal range (table 1). The concept now emerging is that lower thyroid hormone levels within the euthyroid range may adversely affect atherosclerosis [16]. The prevalence of thyroid abnormalities was 19% (table 2 and figure 2). The findings are similar with those reported earlier in the studies [4,9,12-14,15-20]. There is however variability in the changes in different thyroid parameters in different studies. These may be due to the fact that mostly in case control cross-sectional studies thyroid profile has been estimated at a single point in time. Patients may have deranged thyroid hormone levels due to clinically undetected non-thyroidal illnesses. Thus thyroid profile needs to be repeated 6-8 weeks later which is a limitation of the current study. Further there can be variability due to local dietary and environmental factors like consumption of goitrogens, environmental pollutants. Differences can arise due to racial and ethnic differences also. Cross laboratory quality control and standardization of reference ranges and methods used is also a significant factor [4,9,12-14,15-20]. Wu *et al*. reported a 6.6% prevalence of thyroid disorders in general population [9]. The exact prevalence and pattern of thyroid disorder in general population of India is difficult to estimate and depends on age, sex, ethnic and geographic factors and especially on iodine intake. Menon *et al*. reported a 12.2% prevalence of goiter in adult population of an iodine sufficient region in India [26]. Abraham *et al*. reported a 15.8% prevalence of thyroid abnormalities in women in south India [27]. In the present study we observed thyroid function abnormalities in 19% (28.3% in females and 8.5% in males) of diabetics. The most common abnormality was subclinical hypothyroidism (Table 2). A number of studies have also indicated a higher than normal prevalence of thyroid disorders in type 2 diabetic patients, with the hypothyroidism being the most common disorder [7, 13].

It has been proposed that decreased TRH secretion in diabetics was responsible for the occurrence of low thyroid hormones levels [28]. Suzuki *et al*. concluded that abnormal thyroid hormones level found in type-2 diabetes mellitus was because of the presence of thyroid hormone binding inhibitor (THBI) which decreases the conversion of T_4 to $T_3$and dysfunction of the hypothalamus-hypophyseal-thyroid-axis [29]. It was also proposed that the presence of sub-clinical hypothyroidism and hyperthyroidism may result from hypothalamus-hypophyseal-thyroid-axis disorders due to stress [29]. Many diabetics show a low T_3 syndrome suggesting that, there might be impairment in the extrathyroidal conversion of T_4 to T_3. This could well be enhanced by the poor diabetic control [15]. Poorly controlled diabetes, both type 1 and type 2 , may induce a 'low T3 state' characterized by low serum total and free T3 levels, increase in reverse T3 (rT3) but near normal serum T4 and TSH concentrations [30]. Thus lower TSH levels are found despite near normal T4 levels and low T3 profile. Impaired TSH response to TRH or lack of nocturnal TSH peak has been described in patients with uncontrolled diabetes mellitus or with low residual insulin secretion. A global impairment of hormonal secretion (TSH and insulin) associated with diabetes mellitus or some neuroendocrine damage induced by glycaemic variations in diabetes has also been suspected. Low serum T3 is due to reduced peripheral conversion of thyroxin totriiodothyronine via 5' monodeiodination (type 1 deiodinase) reaction [15, 31]. These changes in thyroid hormone metabolism probably reflect both decreased T4 transport and deiodinase 1 and deiodinase 2 actions [31]. Insulin, an anabolic hormone decreases T3 levels by decreasing hepatic conversion of T4 to T3. Intrapituitary T3 and, thus, TSH regulation are derived principally by 5'D-type II deiodinase from circulating T4. Thus these modestly decreased T3 levels fail to stimulate TSH increase [32, 33]. This 'low T3 state' is also seen due to many non-thyroid illness, drugs, fasting and starvation. Diabetes may also be considered a state of 'cellular starvation' [31-33]. Glucocorticoids also inhibit TSH secretion [31, 33]. Also in a longitudinal population-based study, metformin use was associated with an increased incidence of low TSH levels in patients with treated hypothyroidism, but not in euthyroid patients. Thus metformin, a commonly used drug may also affect TSH levels [34].

Thyroid profile – correlations with HbA1c, BMI and lipid profile

The correlations observed are summarized in table 4 and figures 4a-4d. In the study subjects, we observed TSH levels to correlate positively and significantly with HbA1c, the most important marker of glycemic control. In euthyroid individuals with diabetes mellitus, the serum T_3 levels, basal TSH levels and TSH response to thyrotropin releasing hormone (TRH) may all be strongly influenced by the glycemic status [15]. TSH responses and 'low T_3 state' may normalize with improvement of glycemic status but even with good diabetic control, the normal nocturnal TSH peak may not be restored in C-peptide negative patients i.e. those with totally absent pancreatic beta cell function [15]. Fujii *et al* investigated thyroid hormone abnormalities in serum in 47 patients with type 2 diabetes mellitus. No significant differences in T_4 level were found between normal subjects and diabetics. A group of diabetics whose fasting blood sugar levels were over 250 mg/dL showed significantly higher reverse T_3 and lower T_3 levels than healthy controls. These findings suggested that the reduction of T_3 and the increase of reverse T_3 may indicate an adaptation to limit catabolism in diabetics [35]. Significant negative correlation of T3, T4

and FT3 with BMI suggest effect of obesity on the thyroid hormones or it may be due to the confounding affects of increased insulin levels and deranged glycaemic control (table 4).

The correlations of thyroid hormones with lipid profile parameters are summarized in table 4. The correlations suggest that thyroid changes may be associated with poorer TG and HDL levels in diabetics. The concept is now emerging that lower thyroid hormone levels within the euthyroid range may adversely affect atherosclerosis [35, 36]. The present study could not compare the lipid profile parameters in hypothyroid and hyperthyroid diabetics versus the euthyroid diabetics due to the less number of cases with diabetes and hypothyroidism or diabetes and hyperthyroidism to achieve statistical significance. Further study in a large number of type 2 diabetics is thus recommended the study such affect. Superimposed hypothyroidism in DM increases the cardiovascular risk. However hyperthyroid state does not significantly alter the lipids. TG levels in diabetics significantly and negatively correlated with both T3 and T4 and positively with TSH. Thyroid hormones increase the rate of lipolysis in adipose tissue by potentiating the action of catecholamines on hormone sensitive lipase (by upregulating β-adrenergic receptors on adipocyte membrane). The increased flux of FFA to liver leads to increase in triglyceride synthesis. They may stimulate lipogenesis by increasing the activity of malic enzyme, ATP citrate lyase and glucose 6-phosphate dehydrogenase[36]. Arner *et al*. described that in isolated adipocytes insulin receptor number is increased by 70% in hypothyroidism and decreased by 40% in hyperthyroidism. The effect of insulin on lipolysis and glucose oxidation is increased fourfold in hypothyroidism and decreased fivefold in hyperthyroidism. The maximum insulin-induced glucose oxidation was inhibited by 60% in hypothyroidism and enhanced by 180% in hyperthyroidism. They observed that thyroid hormone concentration was significantly correlated with insulin receptor number, insulin responsiveness, and insulin sensitivity. It has been suggested that thyroid hormones regulate the effect of insulin on adipose tissue, which occurs at the receptor and postreceptor levels [37].

CONCLUSIONS

Given the significant prevalence of thyroid abnormalities in type 2 diabetics and their significant correlation with HbA1c and lipid parameters observed in this study, it will be beneficial clinically to assess thyroid hormones in type 2 diabetics as failure to recognize the presence of abnormal thyroid hormone levels in diabetes may be a primary cause of poor management often encountered in some treated diabetics. Further investigations in larger number of subjects with longitudinal serial testing are needed to understand the intimate mechanisms of lipid and glucose metabolism in type 2 diabetes with respect to thyroid function.

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