Original Research Paper

Volume - 10 | Issue - 7 | July - 2020 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Biochemistry

# A STUDY OF THYROID PROFILE IN TYPE 2 DIABETES MELLITUS.

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**ABSTRACT** The study aims to estimate the thyroid profile in type 2 diabetes mellitus and to identify any thyroid dysfunction so that early intervention in the management would delay the onset of the complications of type 2 Diabetes. 80 type 2 diabetes patients and 40 age and sex-matched controls were included in the study. The thyroid profile was estimated using electrochemiluminescent immunoassay in the Cobas e411 analyzer. HbA1c was estimated by particle enhanced immunoturbidometric method A statistically significant decrease in serum T3 and T4 levels was observed in cases when compared to the controls ($P< 0.05$).A statistically significant increase in HbA1c and serum TSH was observed in cases when compared to the controls ($P < 0.05$). The major thyroid dysfunction observed in type 2 diabetes mellitus is of hypothyroidism. With thyroxine therapy, the atherogenic potential can be decreased, which will delay the onset of cardiovascular complications in type 2 diabetes mellitus.

**KEYWORDS :** : subclinical hypothyroidism, type 2 diabetes mellitus, cardiovascular risk.

## INTRODUCTION

Type 2 diabetes mellitus is an endocrine disease of the pancreas, with the metabolic defects which include impaired insulin secretion, peripheral insulin resistance, and increased basal hepatic glucose output[1], which contributes to hyperglycemia. Insulin resistance increases with weight gain. Leptin, resistin, and adiponectin produced by adipocytes contribute to insulin resistance. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs. Lipoprotein degradation catalyzed by lipoprotein lipase in adipose tissue is low in diabetes, hence the plasma chylomicrons and VLDL levels are elevated, resulting in hypertriacylglycerolemia. Hyperglycemia may also negatively impact lipoproteins (particularly LDL and VLDL) through increased glycosylation and oxidation, facilitates the development of aggressive atherosclerosis[2]. Diabetes is a major risk factor for the development of CAD with a higher incidence of MI in patients with DM than those without[3,4]. The major cause of mortality among diabetic patients is cardiovascular and complications. The number of people with diabetes in India increased from 26·0 million in 1990 to 65·0 million in 2016.[5] Thyroid disease is common in the general population and the prevalence increases with age. The diagnosis of thyroid dysfunction in diabetic patients based solely on clinical manifestations can be difficult. 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. The recognition and treatment of thyroid disorders in patients with type 2 diabetes may benefit glycemic control and attenuate cardiovascular risk.

The study aims to estimate the thyroid profile in type 2 diabetes mellitus and to identify any thyroid dysfunction so that early intervention in the management would delay the onset of the complications of type 2 Diabetes.

## MATERIALS AND METHODS

The study was conducted in the Department of Biochemistry of the central laboratory, KIMS& RF, Amalapuram. 80 type 2 diabetes mellitus patients from the Department of General medicine, KIMS& RF, and 40 age and sex-matched controls were included in the study. Prior permission was taken from the Institutional Ethics Committee of KIMS& RF, Amalapuram, to conduct the study. All of the subjects provided their informed consent as approved by the ethics committee.80 Type 2 diabetes cases since 9±0.3 years with the age of 57.6±0.7 years were included in the study. Patients were on oral hypoglycaemic drugs. Patients with Type 1 Diabetes, known thyroid disease, with chronic renal failure, diabetic nephropathy, with acute illness, with hepatic dysfunction and patients on treatment with drugs interfering with thyroid function(amiodarone, propranolol, corticosteroids, lithium, beta-blockers,iodides, interferon-alpha, and oral contraceptives)were excluded from the study.

5 ml of the venous blood sample after 8-12 hours of fasting was drawn. Serum Triiodothyronine($T_3$), thyroxine ($T_4$) and thyroid-stimulating hormone (TSH) were estimated by electrochemiluminescence immunoassay in Cobas e411analyser. Serum total cholesterol was estimated by CHOD/PAP method. HbA1c was estimated by particle enhanced immunoturbidometric method.

### Statistical analysis

Results are shown as Mean ± S.D. (standard deviation). To analyze statistically significant differences in means of continuous variables between 2 groups, a student t-test was used. $P \leq 0.05$ was considered statistically significant.

## RESULTS

**Table 1 : Comparison (Mean ± S.D.) of Thyroid profile in cases and controls.**

| PARAMETER | Type 2 Diabetes (80) | Controls(40) | P value |
|---|---|---|---|
| T3 ( ng/dl) | 70.25±45.25 | 102.46±10.24 | 0.02 |
| T4 (µg/dl) | 6.34±3.18 | 8.32±1.02 | 0.008 |
| TSH ( µIU/mL) | 6.36±2.48. | 2.84±0.54 | < 0.001 |

The above table shows a statistically significant decrease in serum T3 and T4 in cases when compared to controls.

A statistically significant increase is observed in TSH in cases when compared to controls.

**Table 2 : Comparison (Mean ± S.D.) of HbA1c and serum cholesterol in cases and controls.**

| PARAMETER | Type 2 Diabetes (80) | Controls(40) | P value |
|---|---|---|---|
| HbA1c ( %) | 7.9±1.8 | 5.7±1.1 | < 0.001 |
| Cholesterol (mg/dl) | 260.66 ± 24.98 | 165.89±12.74 | < 0.001 |

The above table shows a statistically significant increase in HbA1c and serum cholesterol when compared to controls.

**Table 3: Type of abnormal thyroid profile in the cases.**

| | |
|---|---|
| Total cases with abnormal thyroid profile | 17 (21 %) |
| Sub clinical hypothyroidism | 11 (14 %) |
| Overt hypothyroidism | 6 (8 %) |

The above table shows that out of 17, that is 21 % cases with abnormal thyroid profile; 14% showed sub clinical hypothyroidism and 8% showed overt hypothyroidism.

**Table 4 : comparison of HbA1c within cases with abnormal thyroid profile to those with euthyroid status.**

| PARAMETER | Abnormal thyroid profile ( 17) | Euthyroid (63) | P value |
|---|---|---|---|

| | | | |
|---|---|---|---|
| HbA1c | 8.0±1.7 | 5.2±1.0 | 0.001 |

The above table shows a statistically significant increase in HbA1c in cases with abnormal thyroid profile when compared to those with euthyroid status.

## DISCUSSION

Poor glycemic control in type 2 diabetes mellitus patients, with the presence of chronic hyperglycemia, is reflected by the elevated levels of HbA1c. 90% of the total T4 production occurs in the thyroid gland and it accounts for the secretion of 80µg of T4 /day, whereas 9% of the total T3 is produced in the thyroid gland, and it accounts for the secretion of 4µg of T3 /day from the thyroid gland.

Out of 100% of total T4,33% is converted to T3, 45% is converted to reverse T3 in the tissues by the selenium-containing enzyme 5' deiodinase. The remaining 22% of T4 gets metabolized in the liver and is excreted in the bile. Low total T3 observed in type 2 diabetes cases reflects the poor peripheral conversion of T4 to T3 by the enzyme 5' deiodinase.

This could be the effect of long-term hyperglycemia on the peripheral deiodination of T4 to T3. Poorly managed diabetes has been found to induce a "low T3 state" characterized by low serum total and free T3 levels6,7,. There are alterations in the hypothalamus-pituitary-thyroid axis due to hyperglycemia. The presence of altered thyroid hormone levels in type 2 diabetes may also be due to modified TRH synthesis and release and may depend on the glycemic status. Glycemic status is influenced by insulin, which is known to modulate TRH and TSH levels. The major alterations in thyroid hormones from the pituitary are due to the reduction in TSH stimulation of the thyroid gland. Poorly controlled diabetes may also result in impaired TSH response to TRH or loss of the nocturnal TSH peak. Despite the normal peripheral TSH metabolism, T3 and T4 production and the iodide uptake by the thyroid are diminished. There are important structural changes in the thyroid gland and the pituitary that are accompanied by marked alterations in their secretary activities. Stress, associated with diabetes, may also cause changes in the hypothalamic-pituitary-thyroid axis. It appears that subclinical hypothyroidism results from abnormal functioning of the hypothalamus-pituitary-thyroid axis.

The poorer glycemic control, the more is the possibility of thyroid dysfunction, as it is observed that HbA1c is significantly elevated in the type 2 diabetes cases with thyroid dysfunction when compared to the type 2 diabetic cases with euthyroid status. Serum cholesterol levels are elevated in type 2 diabetes cases. Type 2 diabetic patients with subclinical hypothyroidism are associated with an increased risk of nephropathy and cardiovascular events, but not with retinopathy8. Patients with diabetic nephropathy have an increased risk of cardiovascular disease (CVD).

Subclinical hypothyroidism is characterized by an increased prevalence of elevated serum lipid levels, atherosclerosis, ischemic heart disease, and hypertension often associating with the presence of anti-thyroid antibodies. Thyroid hormone replacement therapy in patients with hypothyroidism has been shown to reduce several surrogate markers of CVD. Thyroid hormone replacement therapy may decrease the risk of CVD in diabetic nephropathy patients with Subclinical hypothyroidism9. It is important to reveal subclinical thyroid diseases in time for effective treatment and for stopping the cardiovascular damages before manifestations of cardiovascular diseases.

Adequate thyroxine replacement will reverse the lipid abnormalities, thus reducing the impending cardiovascular complications. Hyperglycemia is the principal cause of diabetic complications. Effective blood glucose control is the key to preventing complications and improving the quality of life for patients with diabetes.

## CONCLUSION

Subclinical hypothyroidism and overt hypothyroidism are the thyroid disorders identified in the type 2 diabetes mellitus cases involved in this study. Poorer the glycemic control with increased HbA1c levels, higher are the chances of thyroid disorders; early detection and treatment of thyroid dysfunction will reduce the morbidity and mortality due to cardiovascular and nephrological complications in type 2 diabetes mellitus.

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