



A study of prevalence of autoimmune thyroid disorder in type 2 diabetes mellitus

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Abstract

Background: *Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia.*

Methods: *From the patients admitted 100 representative cases of Type 2 DM are taken as subjects for the study. The diagnosis of diabetes is based on revised criteria according to consensus panel of experts from the national diabetes data group and WHO.*

Results: *82.00% cases of Type 2 DM were normal thyroid function and 6.00% cases were hypothyroidism, 12.00% cases were sub hypothyroidism. Hypothyroidism and subclinical hypothyroidism is more evident in the elderly age groups (55–65 years). Among those diabetic patients with thyroid dysfunction (subclinical hypothyroidism and hypothyroidism), 15 (83.33%) out of 18 were females.*

Conclusion: *Thyroid dysfunction was more in female than male. Maximum number of T2DM patients had subclinical hypothyroidism.*

Keywords: *T2DM, Hypothyroidism, Thyroid function.*

Introduction

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the

individual with diabetes and on the health care system. The worldwide prevalence of Diabetes Mellitus has risen dramatically over the past two decades from an estimated 30 million cases in 1985 to 285 million in 2010. Based on the current trends, International Diabetic Federation projects that 438 million individuals will have Diabetes by 2030¹.

Thyroid disorder are also very common in the general population and it is second only to diabetes as the most common condition to affect the endocrine system. Many thyroid abnormalities may co-exist and interact with diabetes mellitus.

Diabetes mellitus affects thyroid functions at many sites, from hypothalamic control of thyroid stimulating hormone (TSH), release to T3 production from T4 in the target tissues.

Thyroid hormones affect glucose metabolism via several mechanisms. Hyperthyroidism has long been recognized to promote hyperglycemia². During hyperthyroidism, the half life of insulin is reduced most likely secondary to an increased rate of degradation and an enhanced release of biologically inactive insulin precursors^{3,4}.

Thyroid disorders remain the most frequent autoimmune disorder associated with type 1 diabetes mellitus (T1DM). Hyperthyroidism is typically associated with worsening of glycemic control and increased insulin requirement whereas diabetes patients with hypothyroidism go rapidly into hypoglycemia with aggressive management. Another aspect is that even subclinical hypothyroidism can exacerbate the coexisting dyslipidemia commonly found in type 2 diabetes mellitus (T2DM) and further increase the risk of cardiovascular diseases.

Materials and Methods

From the patients admitted 100 representative cases of Type 2 DM are taken as subjects for the study. The diagnosis of diabetes is based on revised criteria according to consensus panel of experts from the national diabetes data group and WHO.

Inclusion Criteria

Patients of Type 2 DM.

Exclusion Criteria

Seriously ill-patients, patients previously diagnosed with thyroid disorder and on medication, patients with the chronic conditions known to alter thyroid function, such as hepatic dysfunction, pregnancy and psychiatric illness were excluded from the study.

Method of data collection

- The blood sample of diabetes patients including controls group was taken after fasting for 10-12 hours.

- 5-10ml of venous blood was drawn from the antecubital vein by aseptic technique in plain vial.
- Serum was separated from the collected sample for biochemical analysis. Thyroid profile investigations that included serum T3, T4, TSH analyzed by standard kits.

Statistical analysis

Descriptive statistics were presented as mean \pm standard deviation for continuous measures while absolute values and percentages for categorical measures. All analyses were conducted using the Statistical Package for the Social Sciences (SPSS) 20 statistical software. A $P < 0.05$ was considered to be statistically significant throughout the analysis.

Results

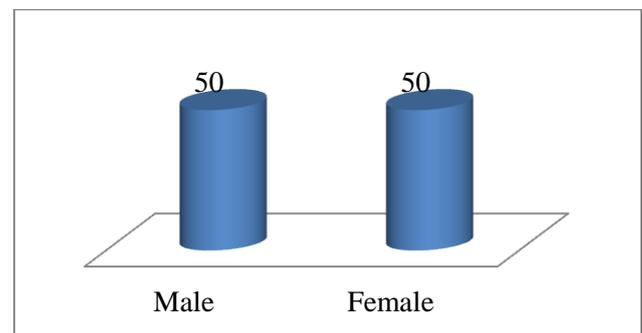


Table no.1 Age wise distribution of thyroid disorders in type 2 DM

Age group	Hypothyroidism	Subclinical hypothyroidism	Total
35-45	0	2	2
46-55	2	4	6
56-65	4	6	10
Total	6	12	18

Hypothyroidism and subclinical hypothyroidism is more evident in the elderly age groups (55–65 years).

Table no.2 Sex wise distribution of thyroid disorders in type 2 DM

Sex	Hypothyroidism	Subclinical hypothyroidism	Total
Male	1	2	3
Female	5	10	15
Total	6	12	18

Among those diabetic patients with thyroid dysfunction (subclinical hypothyroidism and hypothyroidism), 15 (83.33%) out of 18 were females.

Discussion

The most probable mechanism leading to hyperglycemia in thyroid dysfunction could be attributed to perturbed genetic expression of a constellation of genes along with physiological aberrations leading to impaired glucose utilization and disposal in muscles, overproduction of hepatic glucose output, and enhanced absorption of splanchnic glucose. These factors contribute to insulin resistance. Insulin resistance is also associated with thyroid dysfunction. Both hyperthyroidism and hypothyroidism have been associated with insulin resistance which has been reported to be the major cause of impaired glucose metabolism in T2DM. The state of art evidence suggests a pivotal role of insulin resistance in underlining the relation between T2DM and thyroid dysfunction. A plethora of preclinical, molecular, and clinical studies have evidenced an undeniable role of thyroid malfunctioning as a comorbid disorder of T2DM.⁵

In our study maximum 82.00% cases of Type 2 DM were normal thyroid function and 6.00% cases were hypothyroidism, 12.00% cases were sub hypothyroidism.

Kiran Babu *et al*⁶ reported 28% of thyroid dysfunction in T2DM case with 13.2% having hypothyroidism, 8.8% having hyperthyroidism and low T3 syndrome in 5.8%. Celani M F *et al*⁷ reported 31.4% thyroid dysfunction in T2DM cases. Out of these, Subclinical hypothyroidism was most common (48. 3%), followed by subclinical hyperthyroidism (2 4. 2%) and by definite hypothyroidism (23. 1%). Definite hyperthyroidism was found in 4 patients (4. 4%).

Dysregulated glucose disposal and metabolism in adipocytes, muscles, and liver, along with impaired insulin secretion by the pancreatic beta cells, constitute the four major organ system abnormalities which play a definitive role in the pathogenesis of T2DM. It is worth considering that insulin resistance has been a proven condition in hyperthyroidism as well as hypothyroidism⁵

Insulin resistance has been shown to be caused in hypothyroidism in various *in vitro* and preclinical

studies where it was found that peripheral muscles became less responsive in hypothyroid conditions. A possible role of dysregulated metabolism of leptin has been implicated for such pathology.⁵

The pathological features of T2DM include increased intestinal glucose absorption, reduced insulin secretion, and change in the cell mass. Further, symptoms also include increased insulin degradation, increased glucagon secretion, increased hepatic glucose production, enhanced catecholamines, and insulin resistance. These factors have been investigated to be an integral part of hyperthyroidism as well. Hence, an intersection of pathological basis occurs which gives us cue to an array of physiological aberrations which are common in hyperthyroidism and T2DM⁵

Insulin resistance and cell function are inversely correlated with TSH which may be explained by insulin-antagonistic effects of thyroid hormones along with an increase in TSH. The higher serum TSH usually corresponds to lower thyroid hormones via negative feedback mechanism. As TSH increased, thyroid hormones decreased and insulin antagonistic effects are weakened. These observations demonstrate that insulin imbalance is closely associated with thyroid dysfunction and the phenomenon is mediated via cell dysfunction (T2DM)⁵

Conclusion

Thyroid dysfunction was more in female than male. Maximum number of T2DM patients had subclinical hypothyroidism.

References

1. Longo D.L. Fauci A.S. Harrison's principles of Internal Medicine. 18th edition. Mc Graw Hill ; 2012: 2969.
2. Maxon HR, Kreines KW, Goldsmith RE, Knowles HC. Long-term observations of glucose tolerance in thyrotoxic patients. *Archives of Internal Medicine*. 1975;135(11):1477–1480.
3. O'Meara NM, Blackman JD, Sturis J,

- Polonsky KS. Alterations in the kinetics of C-peptide and insulin secretion in hyperthyroidism. *Journal of Clinical Endocrinology and Metabolism*.1993; 76(1):79–84.
4. Dimitriadis G, Baker B, Marsh H, et al. Effect of thyroid hormone excess on action, secretion, and metabolism of insulin in humans. *The American journal of physiology*. 1985;248(5):593–601.
 5. Chaoxun Wang. The Relationship between Type 2 Diabetes Mellitus and Related Thyroid Diseases. *Journal of Diabetes Research*. 2013. Volume 2013. 1-9. doi: 10.1155/2013/390534 (accessed 1st June, 2014).
 6. Kiran N, Amin P, Tayebbeh S, Pegah E. Prevalence of thyroid dysfunction and its management in diabetic patients attending outpatient clinic of KIMS hospital. *Intenational Research Journal of Pharmacy*. 2013;4(9): 132-5.
 7. Celani MF, Bonati ME, Stucci N. Prevalence of abnormal thyrotropin concentrations measured by a sensitive assay in patients with type 2 diabetes mellitus. *Diabetes Res*. 1994;27(1):15-25.