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A Study of Prevalence of Thyroid Disorders in Patients with Type 2 Diabetes Mellitus

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Research Article

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Abstract

Context: Thyroid disorders are very common in the general population and it is second only to diabetes as the most common condition to affect the endocrine system. In this study we try to observe the prevalence of type of thyroid dysfunction in patients with T2DM.

Aims: Prevalence of thyroid dysfunction in patients with T2DM and its effect on glycemic parameters. Prevalence of autoimmune thyroid disorder in patients with T2DM.

Settings and Design: This study included adults having Type 2 DM. Patients were explained about the nature of study and consent was taken.

Methods and Material: Clearance was taken from the ethics committee. All participants who met the inclusion criteria were subjected to detailed clinical examination and relevant investigation.

Statistical analysis: Chi-square test

Results: 234 patients, 112 male and 122 female were included in the study. The mean BMI was high (26.21 ± 3.13 kg/m^2). The mean HbA1c was 8.81 ± 1.96%, while mean HbA1c in patients with both T2DM and thyroid dysfunction was 9.68±1.35%. Thyroid dysfunction was found in 84 cases (35.8%) out of which most common thyroid abnormality was Overt Hypothyroidism (61.9%) followed by Sub-clinical hypothyroidism (27.3%). Out of 84 cases with thyroid dysfunction Anti-TPO was found positive in 45 cases (53.5%) and the most common thyroid dysfunction associated with positive Anti-TPO was subclinical hypothyroidism (44%).

Conclusion: The study shows high prevalence of thyroid dysfunction among patients with T2DM and its association with poor glycemic control. The study also shows the prevalence of Anti-TPO among patients and its significant correlation with thyroid dysfunction.

Keywords: Thyroid; Diabetes; Autoimmune Thyroid disorder

Key Messages

The study shows the high prevalence of thyroid dysfunction in T2DM Patients. Hence every Diabetic patient and especially patients with poor glycemic control should be screened for thyroid dysfunction.

Introduction

Diabetes Mellitus is a clinical syndrome characterized by hyperglycemia caused by absolute or relative deficiency of insulin. Lack of insulin affects the metabolism of carbohydrate, protein and fat and can cause a significant decrease of water and electrolyte homeostasis [1]. Diabesity defined as obesity and type 2 diabetes is likely to be the greatest epidemic in human history [2]. If the total number of diabetics in the world is to be collected in one country, it would be the third biggest country in the world. In recent years, the prevalence of diabetes, as well as prediabetes, has significantly increased in India. A recent Indian Council of Medical Research sponsored study suggests the widespread seriousness of this condition across rural and urban areas with some areas showing prevalence as high as 13% [3].

Thyroid disorders 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 t issues [4,5].

Autoimmunity has been implicated to be the major cause of thyroid-dysfunction associated diabetes mellitus [6-9]. Autoimmunity in which circulating antibodies exist to numerous body tissue components destroy such tissues was stated to be the underlying mechanism behind the increase prevalence of thyroid disorders in type 1 diabetes mellitus ,despite the fact autoimmune thyroid diseases are known to be highly prevalent in all forms of the diabetes ; no specific reason has been adduced for an increased prevalence of thyroid disorders in type 2 diabetes mellitus [10]. However, insulin; the hormone required for transporting glucose from plasma across cell membranes into the cytosol of many cells (including those of the skeletal muscle) is absolutely deficient in type 1 diabetics and relatively deficient in type 2 diabetics. Some authors have postulated that insulin treatment in type 1 diabetics and insulin resistance with resultant high plasma insulin levels in type 2 diabetes may equally predispose both groups to deranged thyroid function [11,12].

Material and Methods

The present study was conducted in Department of Medicine, S.P. Medical College & Associated Group of P.B.M. Hospitals, Bikaner. This study was a cross sectional study. Participants were recruited from PBM hospital and associated group of hospitals, Bikaner. Ethical approval was obtained from institutional research ethics committee and written informed consent will be taken from all subjects. For each patient the following data were collected: Age, Sex, biochemical parameters (complete blood count including hemoglobin, total and differential leukocyte count, total platelet count, HBA1c, fasting plasma glucose, blood urea, serum creatinine, aspartate aminotransferase, alanine aminotransferase, total bilirubin, T3, T4. TSH, Anti TPO antibody. The results were analyzed and tabulated using SPSS 2018 software and Microsoft Excel.

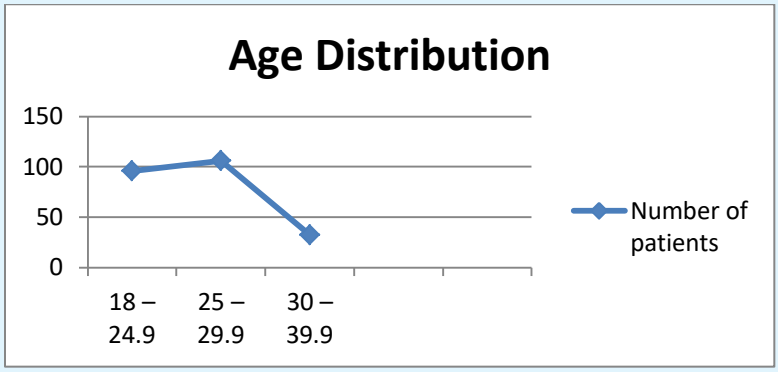
Sample Size

234 OPD and IPD patients of medicine department of S.P. Medical College & Associated group of P. B.M. Hospital, Bikaner during the study period and meeting the inclusion, exclusion criteria will be included in this study.

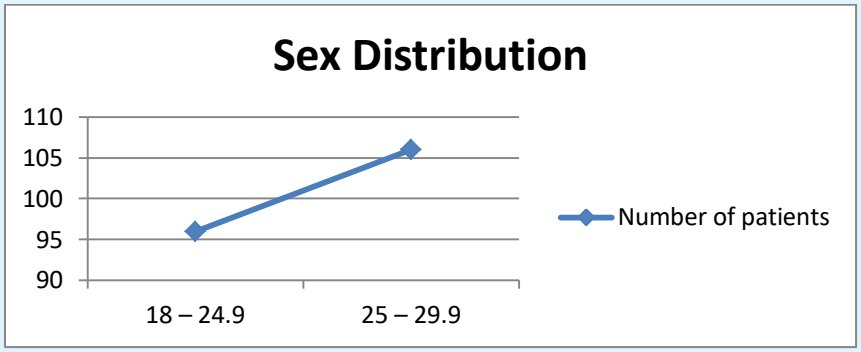
Results

General characteristic	No of patients	95% confidence level
Age group (Yrs)		
41-50	45	14.39-24.87%
51-60	86	30.56-43.28%
61-70	70	24.12-36.22%
71-80	27	7.74-16.34%
>80	6	0.95-5.50%
Gender		
Male	112	41.31-54.47%
Female	122	45.53-58.69%
BMI (kg/m2)		
18 - 24.9	96	35.13-48.20%
25 - 29.9	106	38.07-51.25%
30 - 39.9	32	9.68-18.99%

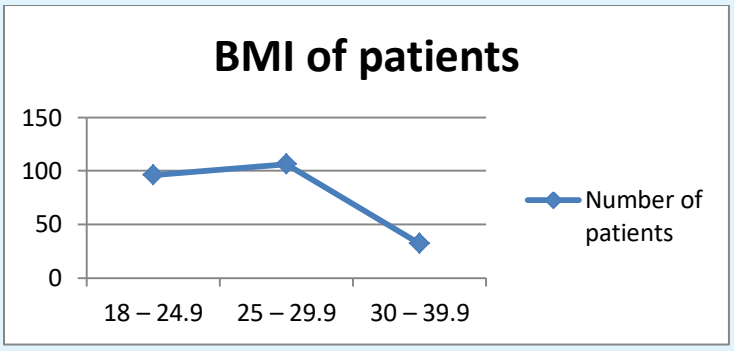
Table 1: General characteristic of patients.



Graph 1: Age Distribution.



Graph 2: Sex Distribution.



Graph 3: BMI of Patients.

Age of the patients varied from 40 years to 85 years with maximum number of patients was observed in the age group 51-60 years. The mean ages of the patients were 58.18 ± 10.08. 112 patients were males and 122 patient's females. The mean BMI of T2DM cases was high (26.21 ± 3.13 kg/m^2). The mean value of HbA1c was 8.81 ± 1.96%. The mean value of FBS was 126.31 ± 29.76 mg/dl and of PPBS 216.29 ± 52.82 mg/dl (Figure 1 & Table 2).

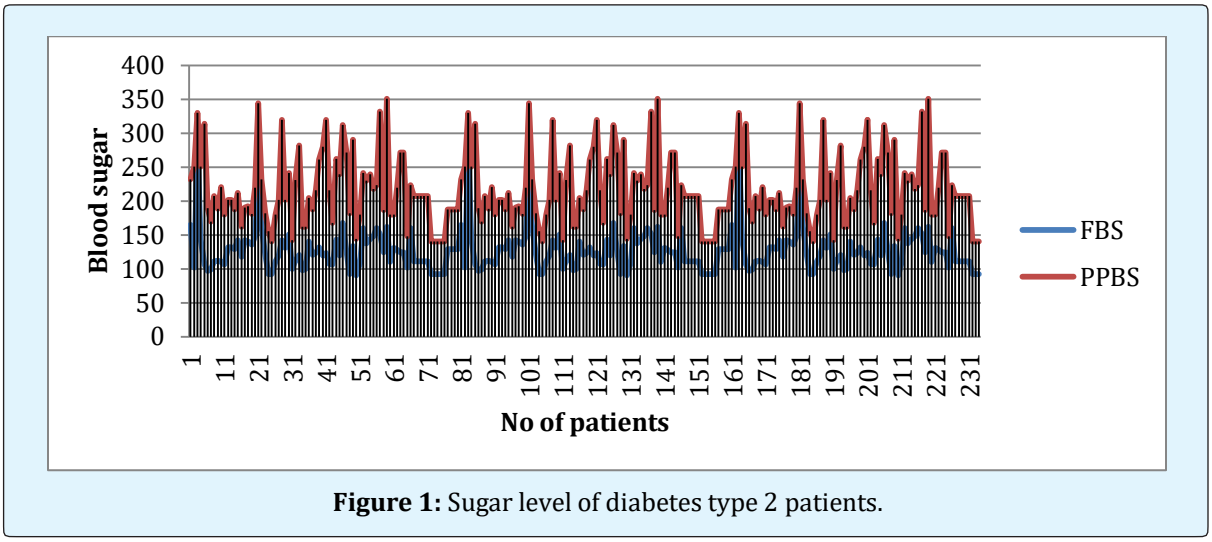


Figure 1: Sugar level of diabetes type 2 patients.

Anti TPO	No. of cases	95% confidence level
Positive	45	75.13-85.61%
Negative	189	14.39-24.87%

Table 2: Anti TPO prevalence.

In present study maximum 80.73% cases of TYPE2DM were anti TPO negative and only 19.23% cases were anti TPO positive (Table 3).

Thyroid Disorder	No. of cases	95% confidence level
Euthyroid	150	57.59-70.25%
Hyperthyroidism	6	0.95-5.50%
Overt Hypothyroidism	52	17.06-28.10%
Secondary Hypothyroidism	3	0.27-3.70%
Sub clinical Hypothyroidism	23	6.33-14.38%

Table 3: Distribution of cases, according to thyroid hormone abnormality.

In present study maximum 64.10% cases of TYPE2DM were normal thyroid function and 22.22% cases were overt hypothyroidism, 9.83% cases were sub clinical hypothyroidism, 1.28% cases secondary hypothyroidism and 2.56% cases were hyperthyroidism (Table 4).

Thyroid Disorder	Anti TPO		Total
	Positive	Negative	
Euthyroid	0	150	150
Hyperthyroidism	3	3	6
Overt Hypothyroidism	13	39	52
Secondary Hypothyroidism	3	0	3
Subclinical Hypothyroidism	20	3	23
Total	45	189	234

Table 4: Association between thyroid disorder and anti TPO.

In the present study out of 23 cases of Subclinical Hypothyroidism, 20 cases were ant-TPO positive and 3 cases were anti-TPO negative. The association between thyroid disorder and anti-TPO was statistically significant (Figure 2).

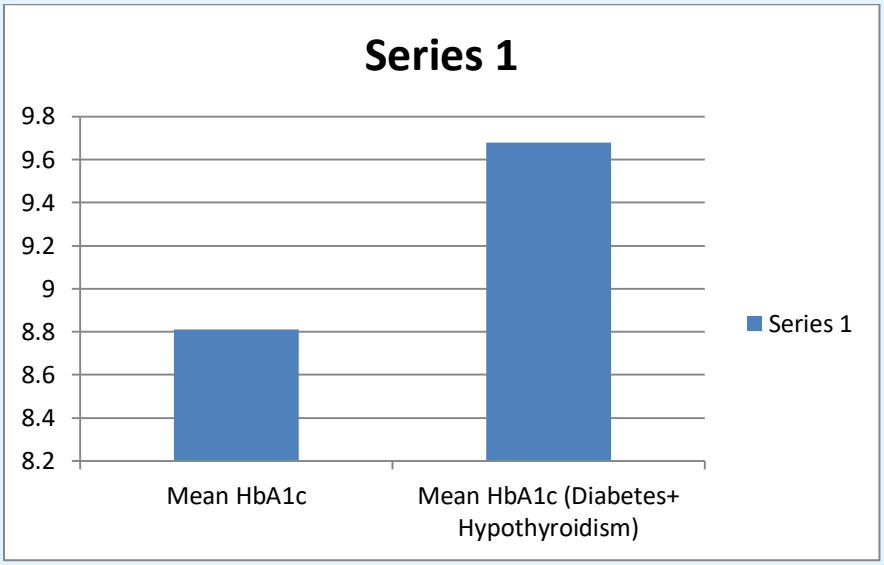


Figure 2: The mean HbA1c was 8.81 ± 1.96%, while mean HbA1c in patients with both T2DM and thyroid dysfunction was 9.68±1.35%.

Discussion

Among the endocrine disorders, diabetes occupies the major share. India is the diabetes capital of the world. Diabetes mellitus is a complex multifactorial disease with varying etiologies, but in most of the cases there is a genetic predisposition. It has been associated with various physiologic changes in different organ systems of the human body [13-16]. The varying complications are associated with the morbidity and mortality associated with diabetes. According to recent estimates, the overall prevalence of T2DM was 4.3% in India. In a recent study, about 42 million people in India suffer from thyroid diseases. The prevalence of overt hypothyroidism in India is 3.9% [17]. The prevalence of subclinical hypothyroidism is also high in our study, the value being 22.22%. The prevalence of subclinical and overt hyperthyroidism in India is 1.6% and 1.3%. T2DM has an intersecting underlying pathology with thyroid dysfunction. Altered thyroid hormones have been described in patients with diabetes, especially those with poor glycemic control. In diabetic patients, the nocturnal TSH peak is blunted or abolished and the TSH response to thyrotropin releasing hormone is impaired. Reduced T3 levels have been observed in uncontrolled diabetic patients and it's become normal with improvement in glycemic control. This “low T3 state” could be explained by the impairment in the peripheral conversion of T 4 to T 3. The abnormal thyroid hormone level may also be the outcome of various medications that the diabetic patients were receiving [18]. For example, it is known that insulin, an anabolic hormone enhances the level of FT4 while it suppresses the level of T3 by inhibiting hepatic conversion of T4 to T3 [19]. On the other hand, some of the oral hypoglycemic agents such as the phenylthioureas (sulfonylureas) are known to suppress the level of FT4 and T4, while causing raised levels of TSH [20].

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 the art evidence suggests a pivotal role of insulin resistance in underlining the relationship 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.

In present study maximum 80.73% cases of TYPE2DM were anti TPO negative and only 19.23% cases were anti

TPO positive. A study conducted by Palma CC et al in 2013 year prevalence of anti TPO antibodies was 10.8%. In our study maximum 64.10% cases of Type 2 DM were normal thyroid function and 22.22% cases were overt hypothyroidism, 9.83% cases were sub hypothyroidism, 1.28% cases secondary hypothyroidism and 2.56% cases were hyperthyroidism. Kiran Babu, et al. 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 MF, et al. reported 31.4% thyroid dysfunction in T2DM cases. Out of these, Subclinical hypothyroidism was most common (48.3%), followed by subclinical hyperthyroidism (24.2%) 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 from 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 hypothyroidism. 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 [21]. A possible role of dysregulated metabolism of leptin has been implicated for such pathology. 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 the pathological basis occurs which gives us cue to an array of physiological aberrations which are common in hyperthyroidism and T2DM [8,22].

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). Both T2DM and hypothyroidism are associated with high BMI and insulin resistance while hyperthyroidism is mostly associated with a low BMI because of high metabolic rate [22,23]. So because of common pathophysiology of T2DM is more commonly associated with hypothyroidism compare to hyperthyroidism. So it is evident from the above discussion that thyroid dysfunction was more common in T2DM than non-diabetics and thus patients with T2DM particularly patients with uncontrolled blood sugar should be screened for Thyroid dysfunction [24-26]. The results of the present study are more or less correlated with the various national and international studies.

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