ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 11, Issue, 05, pp.3654-3656, May, 2019

DOI: https://doi.org/10.24941/ijcr.35193.05.2019

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

PREVALENCE OF THYROID DISORDERS IN TYPE 2 DIABETES MELLITUS- HOSPITAL BASED STUDY

[1]Surendra Kumar Ghintala and [2,*]Dr. Mohd Arif

[1]Senior Resident, Department of Medicine, Pandit Deendayal Upadhyaya Medical College, Churu, Rajasthan, India
[2]Assistant Professor, Department of Medicine, Pandit Deendayal Upadhyaya Medical College, Churu, Rajasthan, India

ARTICLE INFO

Article History:
Received 10th February, 2019
Received in revised form
24th March, 2019
Accepted 10th April, 2019
Published online 30th May, 2019

Key Words:
Glycosylated Hemoglobin,
Thyroid disorders, Diabetes mellitus.

**Corresponding author: Mohd Arif*

ABSTRACT

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 significant decrease of water and electrolytes homeostasis Various 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. The study was carried out in Department Of Medicine, Pandit Deendayal Upadhyaya Medical College, Churu, Rajasthan, from 11 Oct. 2017 to 10 Oct. 2018 for a period of one year. It was a hospital based cross sectional observational study. We have selected 120 cases (60 males and 60 females) of T2DM following the inclusion and exclusion criteria of our study protocol from medicine ward. Serum glucose was estimated by GOD-POD method. Serum HBA1c was measured by HPLC, and thyroid hormones were measured by ECL technology. It was a hospital based cross sectional observational study.It was observed that high incidence of abnormal thyroid hormone levels were seen among T2DM subjects. Out of all patients having thyroid dysfunction, maximum number of T2DM patients had subclinical hypothyroidism.

***Citation*: *Surendra Kumar Ghintala and Dr. Mohd Arif,* 2019.** "Prevalence Of Thyroid Disorders In Type 2 Diabetes Mellitus- Hospital Based Study", *International Journal of Current Research*, 11, (05), 3654-3656.

INTRODUCTION

The burden of diabetes mellitus is increasing worldwide in both the developing and developed world. The disease reduces both a person's quality of life and life expectancy and imposes a large economic burden on health care system and on families (Narayan, 2003; American Diabetes Association, 2003) The estimated number of people with diabetes worldwide is expected to double from 171 million in 2000 to 366 million in 2030 (Susana, 2012) According to the Diabetes Atlas 2006 published by the International Diabetes Federation, the number of people with diabetes in India currently around 40.9 million is expected to rise to 69.9 million By 2025 unless urgent preventive steps are taken (Mohan et al., 2007). 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. Thyroid hormones affect glucose metabolism via several mechanisms. Hyperthyroidism has long been recognized to promote hyperglycemia (Maxon et al., 1975) 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 (O'Meara et al., 1993; Dimitriadis et al., 1985). 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. Thyroid disorders remain the most frequent autoimmune disorders associated with type 1 diabetes mellitus (T1DM). Hypothyroidism is typically associated with worsening of glycemic control and increase insulin requirement whereas diabetic patients with hypothyroidism go rapidly into hypoglycemia with aggressive management. Another aspect in 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. Autoimmunity has been implicated to be the major cause of thyroid-dysfunction associated diabetes mellitus thyroid dysfunction is quite common in diabetic patients and can produce significant metabolic disorder. Therefore regular screening for thyroid abnormalities in all diabetic patients will allow early treatment of subclinical thyroid dysfunction. Moreover not many studies have been

done on thyroid dysfunction in type 2 diabetes mellitus in this part of the country.

MATERIAL AND METHOD

The study was carried out in Department Of Medicine, Pandit Deendayal Upadhyaya Medical College, Churu, Rajasthan, from 11 Oct. 2017 to 10 Oct. 2018 for a period of one year. It was a hospital based cross sectional observational study.
We have selected 120 cases (60 males and 60 females) of T2DM following the inclusion and exclusion criteria of our study protocol from medicine ward.

Specimen Collection: Unhaemolysed serum or plasma can be used for the testing. Anti-coagulants like EDTA and heparin can be used. Preferable sodium fluoride should be used as anti-coagulant. It is recommended to use freshly collected samples for assay. Separated plasma samples can be stored for 3 days at 2-8ºC.

Blood Sugar Estimation God-Pod Method Principle: Glucose is oxidized by glucose oxidase (GOD) to produce gluconate and hydrogen peroxide. The hydrogen peroxide is then oxidatively coupled with 4 amino antipyrene and phenol in the presence of peroxidase to yield a red quinoeimine dye that is measured at 505nm. The absorbance at 505 nm is proportional to concentration of glucose in the sample.

Glucose +2H2O + O2GOD Gluconate + H2O2
H2O2 H2O + O
O + 4-AAP + Phenol POD Quinonimine

Absorbance of the colored solution is directly proportional to the glucose concentration, when measured at 505nm.

HbA1c Estimation Method: HbA1C is estimated by NSGP certified method [CATION EXCHANGE HIGH PERFORMANCE LIQUID CHROMATOGRAPHY, SYSTEM D-10 FROM BIORAD, USA] and Estimated average glucose is calculated by the following Equation : e AG(mg/dl)=28.7XA1C-46.7.

Method For Estimating Thyroid Hormones: Thyroid hormones (FT3, FT4 and TSH) were estimated by The Roche Diagnostics Elecsys 2010 Immunoassay System in our hospital. It is a fully automated, random access, software controlled system for immunoassay analysis. The analyze is specially designed for performing assays utilizing electro chemiluminescent (ECL) technology and is marketed by Roche Diagnostics.

Statistical Analysis: Pearson's chi- square test is used to see if there is any association between categorical variables .We compared continuous variables with Student's t test. Statistical analysis was performed with IBM SPSS Statistics version 21 Software. p-value less than or equal to 0.05 was considered as significant

DISCUSSION

In the present study, out of the 120 diabetic patients, 49 (40.9%) patients had thyroid dysfunction and 71 (59.1%) patients were found to be euthyroid. The findings of our study are consistent with studies of Papazafiropoulou et al 8 (overall prevalence of thyroid disorders was 12.3%), Nobre et al 9 (overall prevalence of thyroid disorders was 12.7%) and Radaiedeh et al 10 (overall prevalence of thyroid disorders was 12.5%). Subclinical hypothyroidism was the most prevalent disorder in diabetic patients in our study, occurring in 16.7%, followed by hypothyroidism in 8.3%, hyperthyroidism in 3.3%, and sick euthyroid syndrome in 12.5% of total 120 diabetic patients. Thus, among thyroid dysfunction maximum prevalence was found to be of subclinical hypothyroidism whereas subclinical hyperthyroidism was least found. Our results are in concordance with the results of Perros et al 11, Celani et al 12, Nobre et al 9. 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

Table 1. Age and Sex wise distributation of the T2DM cases

AGE GROUP (in years)	NO. OF CASES				TOTAL	
	MALE		FEMALE			
	NO.	(%)	NO.	(%)	NO.	(%)
31—40	2	33.3	4	66.7	6	5.0
41—50	17	56.7	13	43.3	30	25.0
51—60	25	51.0	24	49.0	49	40.8
61—70	14	45.2	17	54.8	31	25.8
71 — 80	2	50.0	2	50.0	4	3.4
TOTAL	**60**		**60**		**120**	**100**

Table 2. Thyroid dysfunctions in the T2DM cases and prevalence of auto-immune antibodies

THYROID DYSFUNCTION	CASES (120)		ANTI TPO ANTIBODY		ANTI TG ANTIBODY	
	No.	%	No.	%	No.	%
Normal	71	59.1	15	21.1	10	14
Subclinical Hypothyroidism	20	16. 7	10	50	6	30
Overt hypothyroidism	10	8. 3	5	50	3	30
Primary Hyperthyroidism	4	3. 3	2	50	1	25
Sick Euthyroid Syndrome	15	12. 5	4	26.6	2	13.3

Reduced T3 levels have been observed in uncontrolled diabetic patients and it become normal with improvement in glycaemic control. This "low T3 state" could be explained by impairment in peripheral conversion of T4 to T3. 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. On the other hand some of the oral hypoglycaemic agents such as the phenylthioureas (sulfonylureas) are known to suppress the level of FT4 and T4, while causing raised levels of TSH. 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 T2DM. 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 Low BMI because of high metabolic rate, because of common pathophysiology of T2DM and hypothyroidism, it is more common than hyperthyroidism in T2DM. 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 co-morbid disorder of T2DM.

Conflict of Interest: There is no conflict of interest between authors.

REFERENCES

American Diabetes Association. 2003. Economic costs of diabetes in the US in 2002. Diabetes care. 26.917-32.

Celani MF., Bonati ME., Stucci N. 1994. Prevalence of abnormal thyrotropin concentrations measured by a sensitive assay in patients with type 2 diabetes mellitus. *Diabetes Res.,* 27 (1): 15-25.

Dimitriadis G., Baker B., Marsh H. et al., 1985. Effect of thyroid hormone excess on action, secretion, and metabolism of insulin in humans. *The American journal of physiology.* 248(5):593–601.

Maxon HR., Kreines KW., Goldsmith RE., Knowles HC. 1975. Long-term observations of glucose tolerance in thyrotoxic patients. *Archives of Internal Medicine.,* 135(11):1477–1480.

Mohan, V., Sandeep, S., Deepa, R., Shah B., & Varghese, C. 2007. Epidemiology of type 2 diabetes: *Indian scenario. The Indian journal of medical research.* 125. 217

Narayan KM., Boyle JP., Thompson TJ., Sorensen SW., Williamson DF. 2003. Lifetime risk for diabetes mellitus in the United states. JAMA. 290.1884- 90.

Nobre EL., Jorge Z., Pratas S. et al., 2008. Profile of the thyroid function in a population with type-2 diabetes mellitus. *Endocrine Abstracts.,* 3: p298.

O'Meara NM., Blackman JD., Sturis J., Polonsky KS. 1993. Alterations in the kinetics of C-peptide and insulin secretion in hyperthyroidism. *Journal of Clinical Endocrinology and Metabolism.* 76(1):79–84.

Papazafiropoulou A., Sotiropoulos A., Kokolaki A. et al., 2010 Prevalence of thyroid dysfunction among greek type 2 diabetic patients attending an outpatient clinic. *J Clinical Medicine Research.,* 2 (2): 75-8.

Perros P., McCrimmon RJ., Shaw G., Frier BM. 1995. Frequency of thyroid dysfunction in diabetic patients: value of annual screening. *Diabet Med.,* 12 (7): 622-7.

Radaiedeh AR., Nusier MK., Aar FL. et al., 2004. Thyroid dysfunction in patients with type 2 diabetes mellitus in Jordan. *Saudi Med J.,* 25 (8): 1046-50.

Susana A Moran, KM Venkat Narayan. 2012. Chapter 11: Diabetes mellitus in developed countries. AK Das, PV Rao, SV Madhu, V Mohan(eds). RSSDI Textbook of diabetes mellitus. 2nd edition. New Delhi. Jaypee. p163.

****