Research Article

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THE EFFECT OF DIABETES ON THE ALEVEL OF THYROID GLAND HORMONES IN PATIENTS WITH UNCONTEOLLED TYPE 2 DIABETES IN THI-QAR PROVINCE, IRAQ.

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Abstract

Background: Diabetes mellitus and thyroid dysfunction represent two prominent endocrine disorders frequently encountered in clinical practice. The objective of this research was to examine the prevalence of hypothyroidism among hospitalized individuals diagnosed with type 2 diabetes mellitus, along with its associated factors.

Methods: A total of 120 in patients suffering from type 2 diabetes mellitus were admitted to the metabolic diseases unit. The findings of this investigation indicated a notable elevation of T3 and T4 levels in patients with uncontrolled diabetes (GI) when compared to those with controlled diabetes (GII) and the control group. Conversely, the levels of TSH exhibited a significant reduction in patients with uncontrolled diabetes (GI) relative to both the controlled diabetic patients (GII) and the control group. Utilizing a significance threshold of ($P \leq 0.05$), this study revealed a substantial decrease in the concentrations of thyroid hormones, alongside a reduction in thyroid stimulating hormone levels in patients with uncontrolled diabetes when contrasted with the controlled diabetic patients and the control group..

Aims of Study: Evaluation the risk degree of developing of metabolic disorders complications in patients with DM and their prevalence in Thi-Qar province.

Conclusion: It was concluded from this study that there is a clear relationship between high blood glucose levels and low T3 and T4 levels.

Keywords: thyroid gland; patients; type 2 diabetes; Thi-Qar Province, Iraq.

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Introduction

In the intricate domain of type 2 diabetes, a multitude of interrelated factors, which encompass the relentless progression of age, the accumulation of excess body weight, the female gender, experiences of hospitalization, as well as the existence of thyroid peroxidase antibody positivity, collectively conspire to substantially increase the risk associated with the onset of hypothyroidism, thereby complicating the overall health profile of affected individuals. The presence of diabetes exerts a considerable influence on the functionality of the thyroid gland, as it modifies the concentration of thyroid-stimulating hormone (TSH) and disrupts the conversion process of thyroxine (T4) into triiodothyronine (T3) within the peripheral tissues of the body, thereby potentially leading to significant physiological ramifications [1]. In the cohort of euthyroid individuals diagnosed with diabetes, it is observed that the anticipated nocturnal elevation of TSH may be entirely absent or markedly diminished, while, concurrently, the physiological response of TSH to thyrotropin-releasing hormone (TRH) may become considerably impaired, indicating a disruption in typical endocrine feedback mechanisms. Nevertheless, it is essential to acknowledge that the persistent state of hyperglycemia can accumulate various detrimental effects over time, ultimately culminating in the manifestation of thyroid dysfunction, which necessitates careful consideration in clinical evaluations. As a consequence, when one undertakes the analysis of thyroid function tests, it becomes critically important to understand that, similar to the impact of other acute systemic conditions, the occurrence of diabetic ketoacidosis can precipitate a reduction in the levels of T3 and T4, while the concentration of TSH may paradoxically remain within the normal physiological range, thus complicating the interpretation of thyroid function in diabetic patients.

Additionally, the pathological conditions characterized by hyperinsulinemia, coupled with the phenomenon of insulin resistance, have been shown to significantly stimulate the proliferation of thyroid tissue, thereby enhancing the occurrence of nodular thyroid disease, which ultimately contributes to the development of goiter, as substantiated by various scholarly articles [2]. Furthermore, individuals diagnosed with diabetes who also present with goiter orbitopathy are confronted with an escalated risk of experiencing dysthyroid optic neuropathy, a condition that has been observed to be markedly more prevalent when compared to their nondiabetic counterparts, thereby underscoring the intricate relationship between these medical conditions. A plethora of empirical studies has unequivocally revealed that the complex interplay between diabetes and thyroid function can be mutually influential, demonstrating that disturbances in one system can have significant ramifications on the other. For instance, the emergence of early-stage type 2 diabetes or even prediabetes can serve as a catalyst for the hyperplasia of thyroid tissue, which ultimately culminates in the notable swelling of the thyroid gland and the subsequent formation of nodules that may require medical intervention. Conversely, it is important to note that thyroid dysfunction can substantially disrupt glucose metabolism within the context of diabetes, thereby complicating the clinical management of both conditions. Moreover, it has been firmly established within the literature that the prevalence of subclinical hypothyroidism tends to increase significantly with age, indicating a potential area of concern for geriatric populations. Notably, males and females exhibit distinct tendencies toward the manifestation of thyroid dysfunction, and the association between obesity and hypothyroidism has been prominently highlighted in various studies [3]. A comprehensive review of 36 studies has concluded that females with type 2 diabetes mellitus (T2DM) who are aged over 60 years are demonstrably more susceptible to developing subclinical hypothyroidism, suggesting a need for targeted screening in this demographic. In addition, a cross-sectional observational study conducted in India, which involved a substantial sample size of 1,508 patients diagnosed with T2DM, revealed a significantly heightened risk of hypothyroidism among older type 2 diabetics, particularly those aged over 65 years, with an alarming odds ratio of 4.2 indicating a strong association. A distinct disparity in the prevalence of hypothyroidism was also noted between female and male patients (OR 4.82 vs 2.60), as well as among obese versus normal-weight individuals (OR 2.56 vs. 3.11), thereby highlighting the multifaceted nature of this health issue and its varying impact across different subpopulations [4].

Materials and Methods

Subjects

The target population of this study was 120 patient individuals who are already diagnosed as DM which referred to the Nasiriyah Endocrine and Diabetes Centre in Thi-Qar province, Iraq.The ages of patients and healthy individuals ranged between 45-65 years. The patients were divided into two groups, the first group consisted of 60 patients with uncontrolled diabetes(GI), while the second group consisted of the controlled diabetic patients (GII), in addition to 40 healthy individuals who were considered a control group.The patients are already diagnosed as DM by the consultant medical staff according to history inspection clinical examination and biochemical investigation.

Statistical analysis

Statistical analysis was performed using software statistical package for social sciences (SPSS) version 20, unpaired T test, Pearson's correlation. P value of less than 0.05 was considered as statistically signifi cant at 95% confidence Statistical analysis Statistical analysis was performed using software statistical package for social sciences (SPSS) version 20, P value of less than 0.05 was considered as statistically significant at 95% confidence intervals.

Instruments used for assessment

Plasma glucose by GOD-POD method[semi autoanalyser (BTS-350), fully autoanalyser(BIOSYSTEM A-25)],glycohemoglobin (HbA1C) measured by bio –rad D-10 instrument . Thyroid Profi le by Cobas automated device.

Results

FBS and HbA1C

The present results of blood sugar in GI and GII patients Showed a significant increase ($P \leq 0.05$) in and GII compared with the control group. Also, in GI patients the results showed there was a significant increase ($P \leq 0.05$) compared with GII. The level of HbA1C in blood of patients GI and GII showed a significant increase at ($P \leq 0.05$) in GI and GII compared with the control group. Also, the results showed a significant increase at ($P \leq 0.05$) in GI compared with the GII as summarized in (Table 1).

TSH, T3 and T4

The results of this study showed, as shown in Table 1, that there was a significant decrease at ($P \leq 0.05$) in levels of T3,T4 and TSH in GI compared with the GII and control group. On the other hand, the results showed there was a significant decrease at ($P \leq 0.05$) in levels of T3,T4 and TSH in GII compared with the control group(Table 1).

Table 1: Level of T3,T4 and TSH

Multiple Comparisons					
LSD					
Sig.	**Std. Error**	**Mean Difference (I-J)**	**Category (J)**	**Category (I)**	**Dependent Variable**
0.000	0.154457632	3.50233*	Type (2)	**GI**	**FBS**
0.000	0.172688882	5.89983*	Control Group		
0.000	0.154457632	-3.50233*	GI	**GII**	
0.000	0.172688882	2.39750*			
0.000	0.172688882	-5.89983*	GI	**Control Group**	
0.000	0.172688882	-2.39750*			
0.000	0.133436751	4.73633*	Type (2)	**GI**	**HbA1c**
0.000	0.149186823	6.60417*	Control Group		
0.000	0.133436751	-4.73633*	GI	**GII**	
0.000	0.149186823	1.86783*			
0.000	0.149186823	-6.60417*	GI	**Control Group**	
0.000	0.149186823	-1.86783*	GII		
0.000	2.279328591	-19.59400*	GII	**GI**	**T3**
0.000	2.548366836	-52.25467*	Control Group		
0.000	2.279328591	19.59400*	GI	**GII**	
0.000	2.548366836	-32.66067*	Control Group		
0.000	2.548366836	52.25467*	GI	**Control Group**	
0.000	2.548366836	32.66067*	GII		
0.000	0.134106865	-2.30137*	GII	**GI**	**T4**
0.000	0.149936033	-6.00852*	Control Group		
0.000	0.134106865	2.30137*	GI	**GII**	
0.000	0.149936033	-3.70715*	Control Group		
0.000	0.149936033	6.00852*	GI	**Control Group**	
0.000	0.149936033	3.70715*	GII		
0.000	0.071126849	-1.38677*	GII	**GI**	**TSH**
0.000	0.079522234	-2.19677*	Control Group		
0.000	0.071126849	1.38677*	GI	**GII**	
0.000	0.079522234	-.81000*	Control Group		
0.000	0.079522234	2.19677*	GI	**Control Group**	
0.000	0.079522234	.81000*	GII		
***. The mean difference is significant at the 0.05 level.**					

Discussion

Diabetes can cause thyroid problems. Diabetes can increase the risk of developing thyroid disorders in general. [5] The most important information about this includes the following: Hypothyroidism is more common in people with type 2 diabetes than in normal people, according to a clinical study published in the journal Endocrine Reviews in 2019. [5] High levels of insulin in the blood, which are associated with insulin resistance, can lead to increased proliferation of thyroid cells and the formation of nodules within the gland; Which contributes to disturbances in thyroid hormone levels, according to a clinical study published in the journal Endocrine Reviews in 2019. [5-7] Type 1 diabetes is an autoimmune disease, and it has been observed that this disease is accompanied by autoimmune diseases that affect the thyroid gland, including: [8-12] Hashimoto's thyroiditis: This is a type of hypothyroidism, which most people with type 1 diabetes suffer from at some point in their lives.

Elevated hepatic glucose production plays a crucial role in the onset of peripheral insulin resistance, glucose intolerance, and hyperinsulinemia [12-14]. In the realm of thyrotoxicosis, an augmentation in hepatic glucose synthesis, along with heightened glycogenolysis, promotes glucose tolerance [15-17]. This intricate interaction catalyzes the advancement of subclinical diabetes and intensifies hyperglycemia in patients diagnosed with type 2 diabetes. Research has indicated that both type 2 diabetes mellitus (T2DM) and hyperthyroidism share common pathological characteristics. For example, T2DM is distinguished by variations in beta-cell mass, diminished insulin secretion, and an increase in intestinal glucose absorption, in addition to a rise in glucagon secretion, amplified insulin degradation, insulin resistance, and heightened catecholamine concentrations. These elements are equally pivotal in the pathology associated with hyperthyroidism [18].

Among the factors previously mentioned, insulin resistance stands out as the most prominent bridge linking thyroid malfunctions and T2DM. The phenomenon of hepatic insulin resistance is primarily fueled by an overproduction of glucose, rather than merely being a

consequence of fasting hyperinsulinemia. Furthermore, it has been established that an increase in hepatic glucose output serves as a vital factor in the rise of fasting plasma glucose (FPG) levels among individuals afflicted with T2DM [19,20]. Within the realm of insulin resistance, glucose levels in muscle may surge; nevertheless, the effectiveness of glucose absorption diminishes. The intricate dance between reduced glucose absorption in muscle tissue and heightened hepatic glucose output leads to a decline in overall glucose metabolism. Notably, insulin resistance can manifest in both states of hyperthyroidism and hypothyroidism. Recent findings indicate that insulin resistance also hampers lipid metabolism [21]. As a result, insulin resistance is proposed as a possible linchpin linking thyroid dysfunction and T2DM.

In a comparable context, another study uncovered that the malfunction of beta cells and the body's resistance to insulin are intricately linked to TSH levels, possibly illuminated by the insulin-blocking features of thyroid hormones alongside an increase in TSH. Generally, lowered concentrations of T3 and T4 result in a decline in TSH levels through a cycle of positive feedback. As TSH levels diminish, thyroid hormone concentrations also nosedive, leading to weakened insulin-inhibiting effects; on the other hand, when TSH levels retreat, thyroid hormone levels surge, amplifying the insulin-inhibiting influence. The presence of thyroid dysfunction in our analysis unveiled that individuals battling uncontrolled diabetes show a significant drop in thyroid hormone levels when compared to the control group, even while remaining within the normal laboratory range [22].

Conclusion

Numerous studies have shown that insulin resistance is a critical link between thyroid dysfunction and type 2 diabetes mellitus (T2DM) [4]. There appears to be a bidirectional relationship between T2DM and thyroid dysfunction, each of these clinical entities influences the other. Should be a fire inducing spark Thyrotoxicosis and Hypothyroid states are pathological, they can cause insulin resistance which is equivalent to what that same flame does when it spreads.

Under these conditions, insulin resistance could occur as a result of an attenuated rate at which insulin stimulates glucose transport (insulin-responsive facilitative GLUT 4 translocation) or in the case if subclinical hypothyroidism due to levels had no effect on Haemoglobin A1 However might correlate with such other genes associating risperidone and r=0. In contrast, T3 induced expression of many genes that are key for glucose metabolism and the development of insulin resistance at elevated concentrations. In addition, the vicious cycle of insulin resistance and hyperinsulinemia promotes thyroid tissue growth leading to nodular morphology with development of goiter. In this sense, the current literature indicates that subclinical hypothyroidism and hyperthyroidism are associated with an increase in blood pressure and levels of cholesterol and can also impair insulin secretion as well as micro-vascular-macro vascular systems integrity [15–18], facilitating events like peripheral neuropathy/arterial disease/diabetic nephropaty. Taken together, these observations make a strong case for the complex interplay between thyroid disease and T2DM such that by screening diligently or targeting some risk factors one can act to decrease their burden in further compounding medical morbidity.

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Not Applicable

Ethical Considerations

Not required

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