

**RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES**

Khalilov Hikmatulla Dilshodovich

Assistant of the Department of Normal and Pathological
Physiology of the Tashkent Medical Academy.

Kayimov Mirzohid Normurotovich

Republic of Specialized Narcology Doctor of
Scientific Applied Medicine Center

Esanov Alisher Akromovich

3rd Year Student of the 1st Medical Faculty of the
Tashkent Medical Academy

Abstract

Diabetes and thyroid disease result from endocrine dysfunction, and both have been shown to interact. Changes in thyroid hormone levels, even within the normal range, can trigger the onset of type 2 diabetes (T2D), especially in people with prediabetes. The aim of this review is to understand the pathological relationship between thyroid-related diseases and T2D.

Thyroid-related diseases and T2D are closely related. Type 2 diabetes can be exacerbated by thyroid disorders, and diabetes can worsen thyroid dysfunction. Insulin resistance has been found to play a critical role in T2D and thyroid dysfunction. Therefore, failure to recognize insufficient thyroid hormone levels in diabetes and insulin resistance in both conditions can lead to poor patient outcomes.

Keywords: hypothyroidism, hyperthyroidism, type 2 diabetes, insulin resistance, thyroid dysfunction, thyroid cancer.

Introduction

Thyroid dysfunction and diabetes mellitus are the most common endocrinopathies with a major impact on cardiovascular health.

Diabetes is a global pandemic. The global prevalence of diabetes has increased as a result of increasing obesity and lifestyle changes. In 2017, the global prevalence of diabetes was 425 million people. The prevalence of diabetes is currently increasing worldwide and is expected to increase to 366 million by 2030, affecting 4.4% of all age groups [1]. On the other hand, the prevalence of thyroid dysfunction in the United States and Europe is 6.6% in adults;

it increases with age and is more common among women than men.

Thyroid disease is also significantly more common in patients with type 2 diabetes mellitus (T2QD), ranging from 9.9% to 48%. In addition, studies also reported a high prevalence of



thyroid disease in the diabetic population at 13.4%, higher among women with type 2 diabetes (31.4%) than men with T2QD (6.9%) (6.9%) [2].

Evidence also suggests a strong link between thyroid disease and diabetes. Researchers have shown that thyroid hormone plays a role in controlling glucose metabolism and pancreas function, and that diabetes can alter thyroid function. For example, in diabetes, it was found that the "response of TTG to thyrotropin-releasing hormone" is reduced, which leads to a decrease in the level of T3 and hypothyroidism [3 .]. A decrease in T3 levels during diabetes has been suggested based on studies to observe a reversible decrease in liver thyroxine concentration and deiodinase activity due to hyperglycemia, which may reduce the conversion of T3 from T4.

Other studies have shown that elevated T3 levels can lead to insulin resistance, even in the short term; thereby contributing to the occurrence of T2QD.

The purpose of the study: to study the relationship between type 2 diabetes and related thyroid diseases.

Materials and methods. An analysis of 17 foreign literature sources was conducted on this topic.

Results

Association between thyroid disorders and T2QD Thyroid hormones directly affect insulin secretion. Hypothyroidism resulted in decreased insulin production by beta cells, whereas hyperthyroidism resulted in increased beta cell sensitivity to catecholamines or glucose due to increased beta cell mass [4].

All these changes occur as a result of thyroid hormone metabolism, which increases the risk of developing T2QD and can lead to diabetic complications.

Hyperthyroidism and T2QD

Increased hepatic glucose production is a major factor in the development of peripheral insulin resistance, glucose tolerance, and hyperinsulinemia [5]. Glucose tolerance in thyrotoxicosis occurs with increased glucose release and glycogenolysis in the liver.]. This process contributes to the development of subclinical diabetes and increased hyperglycemia in type 2 diabetes. Studies have shown that T2QD and hyperthyroidism also share some pathological features.

For example, TD2M is characterized by altered B-cell mass, decreased insulin secretion and increased intestinal glucose absorption, increased glucagon secretion, increased insulin breakdown, insulin resistance, and elevated catecholamine levels.

These factors are also an important part of hyperthyroidism [6]. Among the aforementioned factors, insulin resistance was identified as the most important association between thyroid dysfunction and T2QD. Insulin resistance of the liver is caused by hyperinsulinemia due to excessive production of glucose. In addition, increased hepatic glucose uptake was found to be an important regulator of plasma glucose (PG) concentrations in patients with T2QD



[7]. During insulin resistance, muscle glucose uptake increases, but uptake efficiency decreases.

Decreased absorption of glucose into the muscles and increased release of glucose in the liver leads to deterioration of glucose metabolism.

It is worth noting that insulin resistance can occur in both hyperthyroidism and hypothyroidism.

According to recent discoveries, insulin resistance also impairs lipid metabolism [8]. Thus, insulin resistance is a possible link between thyroid dysfunction and T2QD.

Hypothyroidism and T2QD

Hypothyroidism is characterized by decreased glucose absorption from the gastrointestinal tract, increased peripheral glucose accumulation, gluconeogenesis, decreased hepatic glucose production, and decreased glucose excretion [9]. Hypothyroidism can affect glucose metabolism in type 2 diabetes in several ways. For example, subclinical hypothyroidism can lead to insulin resistance due to decreased insulin-stimulated glucose uptake as a result of translocation of the GLUT 2 gene.

In addition, according to the study, in hypothyroidism, the physiological need for insulin is reduced due to a decrease in the secretion of insulin by the kidneys. In addition, anorexia can help reduce insulin production in hypothyroidism. Furthermore, insulin resistance has been associated with hypothyroidism in a number of preclinical and in vitro studies [10], where peripheral muscles were found to be less sensitive to insulin in hypothyroid conditions [11]. In addition, many authors have studied the direct relationship between insulin resistance and hypothyroidism [12]. The association between T2QD and thyroid cancer is controversial. Large prospective cohort studies have shown an increased differential thyroid cancer risk in women with type 2 diabetes [13].

Another large prospective study and pooled analysis of multiple prospective trials [14] showed no evidence of a significant relationship between thyroid cancer and diabetes.

Furthermore, a preliminary review of the literature suggested that any association between thyroid cancer and T2QD was weak [15].

However, Korean studies have shown that thyroid cancer is rare in patients with early T2QD, and this effect persists for up to six years after T2QD is detected [16].

In addition, a retrospective study published in December 2018 showed a significantly increased risk of thyroid cancer in Chinese women with T2QD [17].

Discussion:

Current evidence suggests that insulin resistance plays a critical role in the association between thyroid dysfunction and T2QD.

Both thyroid dysfunction and T2QD have a bidirectional relationship.

Thyroid disorders such as thyrotoxicosis and hypothyroidism can cause insulin resistance. Insulin resistance can develop in subclinical hypothyroidism as a result of a translocation of the glucose transporter type 2 (GLUT 2) gene resulting in decreased insulin-stimulated glucose uptake.



On the other hand, high levels of T3 activate a number of genes involved in glucose metabolism and insulin resistance.

In addition, insulin resistance and hyperinsulinemia increase the development of thyroid tissue, which can lead to nodular thyroid disease.

In addition, the literature suggests that subclinical hypothyroidism or hyperthyroidism can increase blood pressure and cholesterol levels, impair insulin secretion, and impair micro- and macrovascular function, increasing the risk of peripheral neuropathy, peripheral artery disease, and diabetic nephropathy.

Conclusions:

There is considerable evidence that thyroid disease and T2QD are closely related.

T2QD is characterized by changes in beta-cell mass, decreased insulin secretion and increased intestinal glucose absorption, increased glucagon secretion, increased insulin breakdown, insulin resistance, and increased catecholamine levels.

These factors are also an important part of hyperthyroidism.

All these findings suggest that there is a strong association between thyroid diseases and TD2M and that the risk of these two conditions and their medical complications can be minimized by early screening or recognition of risk factors.

References:

1. The interface between the thyroid gland and diabetes mellitus. Duntas LH, Orgiazzi J, Brabant G. *Clin Endocrinol.* 2011; 75:1–9.
2. Incidence of thyroid dysfunction in patients with diabetes: value of annual screening. Perros P, McCrimmon RJ, Shaw G, et al. *Diabetic Med.* 2005; 12:622–627.
3. The relationship between glycemic control and the hypothalamic-pituitary-thyroid axis in patients with diabetes. Gursoy NT, Tuncel E. *Turk J Endocrinol Metab.* 2009; 12:163–168.
4. Insulin sensitivity and counter-regulatory hormones in hypothyroidism and during thyroid hormone replacement therapy. Stanická S, Vondra K, Pelikánová T, Vlček P, Hill M, Zamrazil V. 2005; 43:715–720.
5. Potential therapeutic applications of thyroid hormone analogs. Brenta G, Danzi S, Klein I. *Nat Clin Pract Endocrinol Metab.* 2007; 3:632–640.
6. Insulin-stimulated rate of muscle glucose uptake in hyperthyroidism: importance of blood flow. Dimitriadis G, Mitrou P, Lambadiari V, et al. *J Clin Endocrinol Metab.* 2008; 93:2413–2415.
7. Pathogenesis of type 2 diabetes. DeFronzo RA. *Med Clin North Am.* 2004; 88:787-835, ix.
8. Relationship between altered thyroid status and insulin resistance. Kapadia KB, Bhatt PA, Shah JS. *J Pharmacol Pharmacother.* 2012; 3:156–160.
9. Effect of the thyroid gland on absorption in the digestive tract. Althausen TL, Stockholm M. *Am J Physiol.* 1998; 123:577–588.



10. Effects of insulin on glucose transport and metabolism in skeletal muscle from hyperthyroid and hypothyroid rats. Dimitriadis G, Parry-Billings M, Bevan S, et al. *Eur J Clin Invest.* 2007; 27:475–483.
11. In rats, hypothyroidism reduces peripheral glucose utilization, a defect partially corrected by central leptin infusion. Cettour-Rose P, Theander-Carrillo C, Asensio C, et al. 2005; 48:624–633.
12. The relationship of glucose utilization to hyperinsulinemia in human hypothyroidism and hyperthyroidism. achon C, Tauveron I, Dejax C, et al. *Clin Sci.* 2013; 104:7–15.
13. Diabetes and thyroid cancer risk in the National Institutes of Health-AARP Diet and Health Study. Aschebrook-Kilfoy B, Sabra MM, Brenner A, et al. *Thyroid gland.* 2011; 21:957–963.
14. Physical activity, diabetes and thyroid cancer risk: a pooled analysis of five prospective studies. Kitahara CM, Platz EA, Beane Freeman LE, et al. *Cancer causes control.* 2012; 23:463–471.
15. Diabetes and thyroid cancer risk: a review of the literature. Shih SR, Chiu WY, Chang TC, Tseng CH. *J Diabetes Res.* 2012;
16. The relationship between type 2 diabetes and thyroid cancer. [September; 2019];Seo YG, Choi HC, An AR, et al. *J. Diabetes Res.* 2017
17. Cancer risk in Chinese diabetic patients: a retrospective cohort study based on case-control data. Fang Y, Zhang X, Xu H, et al. . *Endocr Connect.* 2018; 7:1415–1423.