Short Communication

The Leptin and Resistin Levels in Obese and Overweight Diabetic Patients with Hypothyroidism

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Abstract

Background: White adipose tissue (AT) produces leptin, which binds to receptors on the surface of cells. AT releases the endocrine hormone resistin, which interferes with glucose homeostasis maintenance. Type 2 diabetes mellitus (T2DM) is the result of this condition. Resistin has been found to be associated with visceral obesity and diabetes. The two primary classifications of hypothyroidism are primary hypothyroidism and secondary (central) hypothyroidism. **Objectives:** To investigate the relationship between hypothyroidism and adipokines in obese and overweight diabetic patients with hypothyroidism. **Materials and Methods:** Fifty overweight women and 50 obese women, a control group of 70 healthy, visited Al-Habbobi and AL-Nasseriah Teaching Hospital. This study was conducted during October 12022, and February 22023. Leptin and resistin were estimated by enzyme-linked immunosorbent assay; thyroid hormone (free triiodothyronine [FT3], free thyroxine [FT4], thyroid-stimulating hormone [TSH], and C. peptide) by (electrochemiluminescence). Fasting blood sugar by colorimetric method, hemoglobin A1C assay by fluorescence immunoassay. **Results:** The current study showed that leptin and resistin were significantly decreased in obese and overweight diabetic patients compared with the control group, and FT4 was significantly decreased in obese and overweight diabetic patients compared with the control group. On the contrary, FT3 and TSH are elevated in both groups compared to the control group. **Conclusions:** Adipokines, such as leptin and resistin, as well as the thyroid profile consisting of FT4, FT3, and TSH, play a significant role in the diagnostic and monitoring processes of individuals who are both obese and overweight and have diabetes.

Keywords: Adipocytokines, biomarkers, hypothyroidism, obesity, overweight, type 2 diabetes mellitus

Introduction

The prevalence of obesity is a pervasive global health issue. Obesity significantly contributes to the heightened susceptibility of individuals to cardiovascular disease, type 2 diabetes mellitus (T2DM), musculoskeletal disorders, gastrointestinal ailments, respiratory complications, and psychological issues that might impact their everyday functioning. There is a possibility that it could elevate the likelihood of mortality. The use of either a simple or moderate weight loss regimen has been shown to effectively mitigate the risk of numerous diseases.[1]

Obesity and overweight are terms used to describe abnormal or excessive weight growth that is harmful to one's health. Abnormal or excessive fat accumulations that pose a health risk are described as being overweight or obese, according to the World Health Organization. Overweight is defined as having a Body Mass Index (BMI) of 25–29.9 kg/m^2, and obesity is defined as a body mass index of 30 kg/m^2 or higher. Rates of overweight and obesity can be found in both developed and developing countries.[2]

There is a close link between obesity and type 2 diabetes. Overall, 90% of patients with type 2 diabetes are overweight. There is a close link between obesity's metabolic and cardiovascular features. Obesity-related chronic inflammation contributes significantly to the development of insulin resistance, a key pathogenesis of type 2 diabetes.[3]

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Adipose tissue (AT) comprises adipose cells, cells of the immune system, endothelial cells, and stem/stromal cells. These cell types help the tissue serve as an endocrine organ, energy storage, and energy metabolism player.[4]

DM is a condition that can affect how your body processes carbohydrates, proteins, and fats. Failure of the beta-Langerhans islet cells in the pancreas to secrete insulin over time or in large amounts, or problems with peripheral tissue uptake of insulin, cause this condition. The two main groups of the disease are T1DM and T2DM.[5]

Noninsulin-dependent, type 2, or adult-onset diabetes characterizes insulin-resistant people with a family history of diabetes. They can survive without insulin therapy. This type of diabetes probably has a wide variety of root causes. Patients do not experience autoimmune destruction of cells, and they do not have any of the other known causes of diabetes, but the exact causes remain unknown.[6]

Primary hypothyroidism and secondary (central) hypothyroidism are the two main types of this condition. When the thyroid gland itself is unable to produce enough thyroid hormone, this condition is known as primary hypothyroidism. When the thyroid gland is healthy, but the hypothalamus or pituitary gland is malfunctioning, the condition is known as secondary or central hypothyroidism.[7]

White AT is responsible for the production of the peptide hormone leptin; leptin exerts its effects by interacting with cell-surface leptin receptors. Leptin receptors are found in neuronal, hepatic, pancreatic, cardiac, and intestinal tissue. Resistin, a hormone secreted by AT, impairs glucose homeostasis. It causes T2DM. Resistin is linked to visceral obesity and diabetes.[8]

The aim of this research was to examine the correlation between thyroid hormone and adipokines in a population of obese and overweight individuals with diabetes and hypothyroidism.

Materials and Methods

Study design and patients

This case–control study included 50 overweight women and 50 obese women who visited Al-Habbobi and AL-Nasiriyia Teaching Hospitals, Thi-Qar province. The median age of the participants in the study was 35. A control group of 70 healthy, normal-weight women was also included in the study. Doctors at Al-Habbobi Teaching Hospital and Al-Nassriah Teaching Hospital examined patients for this study during October 12022 and February 22023. We excluded men and also excluded women under the age of 30 years without taking into account the incidence of a specific disease, especially thyroid dysfunction.

Blood samples for the measurement of serum leptin, resistin, free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH), hemoglobin A1C (HBA1C), fasting blood sugar (FBS), and C. peptide. A total of 5 mL of blood was drawn from each patient and control subject; 2 mL were placed in an EDTA tube for HBA1C testing within 30 min, and the remaining 3 mL were placed in sterile gel tubes and allowed to coagulate at room temperature for 30 min before being centrifuged for 15 min at a speed of 3000 rpm to separate the components. The serum should be separated and kept at a temperature of -20°C until use. Leptin and resistin were assayed by an enzyme-linked immunosorbent assay, according to the operational manual of Snlog, China. FT3, FT4, TSH, and C. peptide performed with immunoassay using electrochemiluminescence (ECLIA) kits are compatible with analyzers for immunoassays such as the Elecsys and the Cobas E411, according to the operational manual of ROCH, Germany. HBA1C is performed by fluorescence immunoassay using AFIAS-6, according to the operational manual of Bodytech, S. Korea. FBS is performed by the colorimetric method by using a spectrophotometer, according to the operational manual of BIOLABO, France.

Statistical analysis

The statistically significant differences were determined using SPSS version 26 (SPSS, IBM Company, Chicago, Illinois).

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients' verbal and analytical approval before conducting the study. The study protocol and the subject information and consent form were reviewed and approved by a local committee on publication ethics at Thi-Qar Health Directorate under reference No. 839/11 on August 14, 2022.

Results

Table 1 shows that results revealed a significant decrease of leptin and resistin in obese diabetic patients in comparison with a control group (117.91 ± 21.21 vs. 212.10 ± 32.58 pg/mL, $P < 0.001$) (0.16 ± 0.05 vs. 0.25 ± 0.03 ng/mL, $P < 0.001$), respectively.

Table 2 shows that results revealed a significant decrease of leptin in the overweight diabetic patient in comparison with the control group (175.03 ± 67.09 vs. 212.10 ± 32.58 pg/mL, $P = 0.001$), respectively. The results of this study showed that there was a significant decrease in resist in between overweight diabetic patients in comparison with the control group (0.20 ± 0.07 vs. 0.25 ± 0.03 ng/mL, $P < 0.001$), respectively.

Table 3 shows that results revealed a significant increase of FT3 in obese diabetic patients in comparison with the control group (5.81 ± 0.36 vs. 3.94 ± 0.26 pmol/L, $P < 0.001$), respectively. The results of this study showed that there is a

Table 1: Differences in adipocytokine (hormone), leptin, and resistin levels between obese diabetic patients and control

Parameters	Control mean ± SD	Obese diabetic mean ± SD	*P* value
Leptin (pg/mL)	212.10 ± 32.58	117.91 ± 21.21	<0.001
Resistin (ng/mL)	0.25 ± 0.03	0.16 ± 0.05	<0.001

Compared with healthy control: *** $P < 0.001$, ** $P < 0.01$

Table 2: Differences of the adipocytokine (hormone) indicators between control and overweight diabetic patients

Parameters	Control mean ± SD	Overweight diabetic mean ± SD	*P* value
Leptin (pg/mL)	212.10 ± 32.58	175.03 ± 67.09	0.001
Resistin (ng/mL)	0.25 ± 0.03	0.20 ± 0.07	<0.001

Compared with healthy control: *** $P < 0.001$, ** $P < 0.01$

Table 3: The serum levels of thyroid hormone (FT3, FT4, and TSH) in obese diabetic patients compared to the control group

Parameters	Control mean ± SD	Obese diabetic mean ± SD	*P* value
FT3 (pmol/l)	3.94 ± 0.26	5.81 ± 0.36	<0.001
FT4 (pmol/l)	16.12 ± 0.42	14.12 ± 0.46	<0.001
TSH (μIU/mL)	1.47 ± 0.51	2.83 ± 0.07	<0.001

Compared with healthy control: *** $P < 0.001$, ** $P < 0.01$

Table 4: Differences in the thyroid hormone levels between control and overweight diabetic patients

Parameters	Control mean ± SD	Overweight diabetic mean ± SD	*P* value
FT3 (pmol/L)	3.94 ± 0.26	4.80 ± 0.43	<0.001
FT4 (pmol/L)	16.12 ± 0.42	15.27 ± 0.42	<0.001
TSH (μIU/mL)	1.47 ± 0.51	2.42 ± 0.16	<0.001

Compared with healthy control: *** $P < 0.001$, ** $P < 0.01$

significant decrease in FT4 among obese diabetic patients in comparison with the control group (14.12 ± 0.46 vs. 16.12 ± 0.42 pmol/L, $P < 0.001$), respectively. Moreover, there is an increase significant between the levels of TSH in the obese diabetic patient compared with a control group (2.83 ± 0.07 vs. 1.47 ± 0.51 μIU/mL, $P < 0.001$), respectively.

Table 4 shows that results revealed a significant increase of FT3 in overweight diabetic patients in comparison with the control group (4.80 ± 0.43 vs. 3.94 ± 0.26 pmol/L, $P < 0.001$), respectively. The results of this study showed that there is a significant decrease in FT4 between overweight diabetic patients in comparison with the control group (15.27 ± 0.42 vs. 16.12 ± 0.42 pmol/L, $P < 0.001$), respectively. Moreover, there is a significant increase in the levels of TSH in overweight diabetic patients compared with the control group (2.42 ± 0.16 vs. 1.47 ± 0.51 μIU/mL, $P < 0.001$), respectively.

Discussion

The results showed a significant decrease in leptin concentration between the diabetic obese and overweight group compared to the control group *P* value (<0.001). This research is agreed with[9] ($P > 0.001$, 0.009, and 0.012). This decreases because of mutations in LEP or the LR gene, which are known as congenital leptin deficiencies.[10] Genetic mutations in the leptin pathway can be a cause of human obesity and leptin deficiency.[11] Outside of the hypothalamus, leptin interacts with the mesolimbic dopamine system, which is involved in motivation for and reward of feeding, and the nucleus of the solitary tract of the brainstem to contribute to satiety. Leptin in regulating energy and food consumption. When leptin decreases, the body gives a clear nervous signal of hunger and insufficient energy, which leads to increased appetite and food intake, in addition to lack of exercise, lack of adherence to a healthy diet, and consumption of carbohydrates in large quantities with sugars. This leads to obesity.

This result was incompatible with.[9] The results show increases significantly in leptin concentration in obese diabetics compared to the control group ($P > 0.001$). As leptin reduces appetite and body weight, the paradoxical coexistence of obesity and hyperleptinemia suggests the pathology of "leptin resistance." Leptin resistance can be due to a defect in the intracellular mechanism or due to impairment in transport through the blood–brain barrier.[12]

The results of our research showed a significant decrease in the concentration of resistin in the diabetic obese and overweight group, compared to the control group ($P < 0.001$). This research is agreed with[13] ($P < 0.001$). The resistin gene represents a potential candidate for the etiology of insulin resistance and type 2 diabetes, although

the expression of the resistin gene in fat cells and AT from overweight subjects has been reported to be almost absent.

This search is disagreed with.[14] It was found in this research on humans that the proportion of resistin increases with weight gain and is associated with a positive correlation with BMI.[15] Although it seems that resistin levels increase with obesity, it is questioned whether such serum resistin increases are accountable for the insulin resistance that appears to be associated with increased adiposity. Many researchers in their respective studies have shown that this is indeed the case by finding positive correlations between resistin levels and insulin resistance.[16]

The results in this research showed, after calculating the concentration of thyroid hormones, that there is a significant increase in the hormone FT3 in obese and overweight diabetic patients included in the research compared to the control group ($P < 0.001$). The results also showed a significant decrease in the hormone FT4 in obese and overweight diabetic patients included in the research compared to the control group ($P < 0.001$). Also, there was a significant increase in hormone TSH in obese and overweight diabetic patients included in the research compared to the control group ($P < 0.001$). The results of this search are consistent.[17] the result showed TSH levels are at the upper limit of the normal range or slightly increased in obese children, adolescents, and adults and are positively correlated with BMI and low FT4 with a moderate increase in T3 or free T3 (FT3) levels has been reported in obese subjects. One theory suggests an increased deiodinase activity leading to a high conversion rate of T4 to T3.[18]

Further explanation is that inflammatory cytokines secreted from AT, such as tumor necrosis factor-alpha, interleukin (IL)-1, and IL-6, inhibit sodium/iodide symporter mRNA expression and iodide uptake activity. Leptin stimulates TRH secretion in the hypothalamus. Leptin produced in the pituitary is capable of inhibiting TSH secretion. Leptin secretion from ATs is regulated by stimulatory factors such as TSH, glucocorticoids, T4 and T3, and insulin, as well as inhibitory factors such as cold and free fatty acids.[19] One of these adipokines is leptin, which seems to be a promising link between obesity and alterations of thyroid hormones. The interrelation between leptin and TSH is bidirectional since TSH stimulates leptin secretion; in addition, leptin regulates TSH secretion as it promotes thyrotropin-releasing hormone gene expression directly in the paraventricular nucleus, ultimately stimulating TSH release.[20]

Conclusion

We can conclude from the results obtained for some adipokines (leptin, resistin) and thyroid hormones (FT3, FT4, and TSH) that there is a strong link between thyroid and thyroid hormones and leptin and can be used for treatment and follow-up of people with obesity and overweight with diabetes around the world.

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Conflicts of interest

There are no conflicts of interest.

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