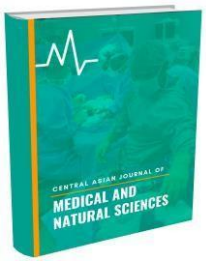

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Article

Investigation The Relashinship Between Hashimoto Disease and TPOA in Patients' Women

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Abstract: Background; Hashimoto thyroiditis is an autoimmune illness that destroys thyroid cells through cell and antibody-mediated immunological mechanisms. In developed countries, it causes most hypothyroidism. However, iodine deficiency is the leading cause of hypothyroidism worldwide. Hashimoto thyroiditis is caused by antithyroid antibodies attacking thyroid tissue, creating fibrosis. The diagnosis might be difficult; hence the disease is occasionally identified late. Most lab tests show elevated thyroid-stimulating hormone (TSH), decreased thyroxine (T4), and enhanced antithyroid peroxidase (anti-TPO) antibodies. This exercise discusses Hashimoto thyroiditis' pathogenesis, diagnosis, and treatment, as well as the interprofessional team's role. Methodology: The study comprised 50 women with Hashimoto thyroditis, as well as a control group of 46 healthy, the patient and control who came to teaching Hospital between September 2022 and April 2023 in Basrah, Southern Iraq. TPA was measured using an Automated enzyme-linked fluorescence immunoassay (ELFA). Thyroid hormone (FT3, FT4, and TSH) was also measured using an Automated enzyme-linked fluorescence immunoassay (ELFA). Results; TPOA levels were significantly higher in patients compared to age-matched healthy controls, and FT4 and FT3 and levels were significantly lower in Hashimoto thyroiditis patients compared to age-matched healthy controls, according to the current study. Instead, and TSH are increased. Conclusions: From our results, we can conclude the relashinship between TPOA levels and Hashimoto thyroditis disease in women in Basra Governorate.

Keywords: Anti-thyroid peroxidase (anti-TPO) antibodies, Hashimoto disease, biomarkers, hypothyroidism.

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1. Introduction

Hashimoto thyroiditis is an autoimmune disease that destroys thyroid cells by cell and antibody-mediated immune processes. It is the most common cause of hypothyroidism in developed countries. In contrast, worldwide, the most common cause of hypothyroidism is an inadequate dietary intake of iodine [1].

The etiology of Hashimoto disease is very poorly understood. Most patients develop antibodies to a variety of thyroid antigens, the most common of which is anti-thyroid peroxidase (anti-TPO). Many also form antithyroglobulin (anti-Tg) and TSH receptor-blocking antibodies (TBII). These antibodies attack the thyroid tissue [2].

The development of Hashimoto disease is thought to be of autoimmune origin with lymphocyte infiltration and fibrosis as typical features. The current diagnosis is based on clinical symptoms correlating with laboratory results of elevated TSH with normal to low

thyroxine levels. It is interesting to note, however, that there is little evidence demonstrating the role of antithyroid peroxidase (anti-TPO) antibody in the pathogenesis of autoimmune thyroid disease (AITD). Anti-TPO antibodies can fix complement and, in vitro, have been shown to bind and kill thyrocytes []3]

Thyroid peroxidase (TPO) antibodies are a type of thyroid antibody. Thyroid peroxidase is an enzyme which helps to make thyroid hormones (T3, T4 and TSH). The body's immune system makes antibodies in response to non-self proteins. These non-self proteins are called antigens. Making antibodies is important to protect us from diseases. However sometimes the immune system identifies our own proteins as non-self, making autoantibodies [4].

Thyroid antibodies develop when the immune system identifies our own thyroid cells and tissues as non-self. This can cause inflammation and affect thyroid function. Normal TPO antibodies range is less than 30 international units per millimeter (IU/ml). Thyroid peroxidase antibodies are considered high if above this. Autoimmune thyroid disorders cause high thyroid peroxidase antibodies. The two main autoimmune thyroid disorders are Grave's disease and Hashimoto's thyroiditis [5].

A goiter was used as a diagnostic tool for thyroid diseases and either an iodine deficient or excess was blamed. An autoimmune etiology wasn't proposed until 1956, a long time afterwards [6]. The only clinical tools available to doctors back then were measuring serum protein-bound iodine as a surrogate for thyroid hormone (TH) concentration, measuring iodine uptake into the gland with radioactive iodine using a geiger counter, and measuring radioiodine discharge following treatment with perchlorate [7].

Low FT4 and high TSH readings are characteristic of patients with primary hypothyroidism. Iodine deficiency is the leading cause of primary hypothyroidism. Hashimoto's thyroiditis is the most common cause in iodine-rich areas; hence, TPO-Ab testing is also recommended [8].

Hypothyroid patients have an increased risk of myocardial damage and pericardial effusions compared to euthyroid controls with similar clinical characteristics [9].

2. Materials and Methods

This is a cross-sectional study included 50 women with Hashimoto thyroditis. The median age of the participants in the study was 35. A control group of 49 healthy, normal-Thyroid function test was also included in the study. A group visited Al-Faiha specialized diabetes, endocrine, and metabolism center (FDEMC) and AL-sader teaching Hospital. Doctors at Al-Faiha specialized diabetes, endocrine, and metabolism center (FDEMC) and AL-sader teaching Hospital examined patients for this study between September 2022 and April 2023 in Basrah, Southern Iraq. We excluded men and also excluded Patients with Graves disease, thyrotoxicosis, renal failure, a thyroid tumor, children, patients aged 55 and under, and patients aged 18 and above were not included in the study. Blood samples for the measurement of serum TPOA, FT3, FT4, TSH, A total of 5 ml of blood was drawn from each patient and control subject were placed in sterile gel tubes and allowed to coagulate at room temperature for 30 minutes before being centrifuged for 15 minutes at a speed of 3000 rpm to separate the components. The serum should be separated and kept at a temperature of -20 degrees Celsius until use. TPOA, FT3, FT4 and TSH was assayed by an Automated enzyme-linked fluorescence immunoassay (ELFA), according to the operational automated of Biomerieux, Firance.

Statistical Analysis

The statistically significant differences were determined using SPSS (version 26).

3. Results and Discussion

Result

Table (1) also show highly significant increase the levels of TPOA in the patients group (450.44±400.43) compared to control (0.89±0.14)(P<0.001).

Table 1. Differences in TPOA, FT4, FT3 and TSH, between patients group and control group.

Parameters	Control group (n=46)		Patients group (n=50)		P. value
	Mean	SD	Mean	SD	
TPOA	0.89	0.14	450.44	400.43	<0.001
FT3	4.65	0.59	4.19	1.15	0.01
FT4	15.39	2.45	11.63	4.05	<0.001
TSH	2.16	1.14	16.25	13.23	<0.001

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

Discussion

When comparing thyroid hormones between the control group and the patients, a significant decrease was observed in both FT3 and FT4 in patients group compared to control (4.65±0.59 vs. 4.19±1.15, P=0.01) (15.39±2.45 vs. 11.63±4.05, P<0.001) respectively, while there is a significant increase in serum levels of TSH in patients group compared to control (2.16±1.14 vs. 16.25±13.23, P<0.001), while there is a significant icrease in serum levels of TPOA in patients group compared to control (450.44±400.43 vs. 0.89±0.14, P<0.001).

Most patients develop antibodies to a variety of thyroid antigens, the most common of which is anti-thyroid peroxidase (anti-TPO). Many also form antithyroglobulin (anti-Tg) and TSH receptor-blocking antibodies (TBII). These antibodies attack the thyroid tissue, eventually leading to inadequate production of thyroid hormone. There is a small subset of the population, no more than 10-15% with the clinically evident disease, that are serum antibody-negative. Positive TPO antibodies presage the clinical syndrome [10][11]. This research is consistent with [12].

The relationship of hypothyroidism with autoimmunity is a matter of great controversy, as the most common cause of autoimmune thyroid disease that leads to tissue damage in the gland and thus affects the activity of the thyroid gland. Hashimoto's thyroditis, and therefore Hashimoto's disease can be diagnosed by examining TPOAand knowing its levels in affected people [13]

The development of Hashimoto disease is thought to be of autoimmune origin with lymphocyte infiltration and fibrosis as typical features. The current diagnosis is based on clinical symptoms correlating with laboratory results of elevated TSH with normal to low thyroxine levels. It is interesting to note, however, that there is little evidence demonstrating the role of antithyroid peroxidase (anti-TPO) antibody in the pathogenesis of autoimmune thyroid disease (AITD). Anti-TPO antibodies can fix complement and, in vitro, have been shown to bind and kill thyrocytes. However, to date, there has been no correlation noted in human studies between the severity of disease and the level of anti-TPO antibody concentration in serum. We do, however, know that positive serum anti-TPO antibody concentration is correlated with the active phase of the disease. Other

theories implicated immune complexes, containing thyroid directed antibodies, as culprits of thyroid destruction [13] [14].

TPO is a membrane-bound enzyme responsible for the iodination of tyrosyl residues in the thyroglobulin molecule. It is known as the microsomal antigen due to its intracellular location. TPO activity is not blocked by anti-TPO Abs in healthy individuals, nor do they interfere with the blocking activity of anti-TPO Abs in patients with AITD. While anti-TPO Abs in patients with AITD can fix complement, destroy thyroid cells, and competitively inhibit enzymatic activity, the risk factors for anti-TPO Abs presence in GD include a higher age, iodine deficiency or excess, a family or personal history of AITD, along with idiopathic and autoimmune causes. Furthermore, paradoxically, smoking and pregnancy appear to decrease the risk of positive anti-TPO Abs [15] [16] [17].

Anti-TPO Abs are more prevalent and more indicative of thyroid disease than anti-thyroglobulin antibodies (anti-TG Abs) [18]. In AITD patients, anti-TPO Abs are found in about 90-95% of patients, while being present in about 80% of GD patients and only in 10-15% of non-AITD patients [19]. Anti-TPO Abs in Hashimoto's thyroiditis play a role in thyroid cell damage; however, they do not play an established role in GD (DeGroot, 2015). Anti-TG Abs do not cause thyroid cell destruction and can be detected in about 10% of healthy young subjects and 15% of patients who are greater than 60 years of age, as well as in about 60-80% of patients with Hashimoto's thyroiditis and 50-60% of patients with GD [19].

In summary, prevalence of anti-TPO and anti-Tg antibodies is high in patients with GD and HT, while anti-TSHR antibodies are common in GD patients but relatively rare in patients with HT. This may suggest that anti-TSHR antibodies are produced under more specific situations than the other antibodies. This difference is also reflected in some factors that have opposite effects in GD and HT, like, for instance, smoking and stress (see Factors with Opposite Effects on GD and HT). There are additional differences in development and manifestation of the diseases. GD is usually characterized by rapid onset of the symptoms and is, except for elderly people with less typical symptoms, diagnosed and treated quite fast [20]. Established treatments normalize titers of TSHR antibodies in adults within 2 years, while treatment of children and adolescents requires longer treatment times [21].

HT develops gradually over months and years with very high antibody titers in some patients [22]. Symptoms can be mild, and patients might not seek medical advice. Even when treatment has been initiated, titers of anti-TPO antibodies decrease only slowly (e.g., over 5 years) upon treatment with levothyroxine, and anti-TPO antibody titers remain in the pathological range [23].

Thyroid peroxidase (TPO) antibodies are a type of thyroid antibody. Thyroid peroxidase is an enzyme which helps to make thyroid hormones (T3, T4 and TSH). The body’s immune system makes antibodies in response to non-self proteins. These non-self proteins are called antigens. Making antibodies is important to protect us from diseases. However sometimes the immune system identifies our own proteins as non-self, making autoantibodies. The immune system then attacks our own body proteins in error. This is known as an autoimmune disease. Thyroid antibodies develop when the immune system identifies our own thyroid cells and tissues as non-self. This can cause inflammation and affect thyroid function [24],[25][26].

4. Conclusion

In summary, we conclude that high TPO levels have a direct effect on the function of the thyroid gland, as all research has proven high levels of TPOA in people with Hashimoto’s disease, and it had a direct role in causing the disease. Therefore, one of the most important recommendations is to monitor TPOA and conduct periodic examinations for people Who have a higher risk of thyroid disease.

Declaration
Ethical approval is Not applicable.
Consent to participate is Not applicable.
Consent for publication is Not applicable.
Competing interests, the authors declare no competing interests

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