

(RESEARCH ARTICLE)



Effect of autoantibodies and cytokines on thyroid disorders in Thi-Qar

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Abstract

Introduction: The thyroid gland is one of the important endocrine gland that have many functional activates in the body. It is usually located in the front of the neck and is shaped like a butterfly. It goes all the way down to the platysma, stern thyroid, and Stern hyoid muscles. In all vertebrates, the thyroid gland and its hormones perform several important activities.

Methodology: Fifty patients diagnosed with hyperthyroidism disease (25 males and 25 females). 50 subjects in good health as controls (25 males and 25 females).

Results and dissuasion: When compared to controls, the findings of our investigation (17.04 ± 5.10 vs. 1.15 ± 0.25 U/l) demonstrate a substantial rise ($P \geq 0.00$) of TSH-receptor Ab levels in the subjects (17.04 ± 5.10 vs. 1.15 ± 0.25 U/l). The findings revealed a substantial rise ($P \geq 0.00$) in FT4 level in subjects (6.16 ± 0.62 vs. 1.40 ± 0.24 ng/dl) compared to controls. In our investigation, the T3 level demonstrates a considerable rise ($P \geq 0.00$) of patients (2.81 ± 0.20 vs. 0.32 ± 0.07 ng/dl) vs. control. The findings of our investigation, which were presented in table 3.2, clearly demonstrated a substantial drop ($P \geq 0.00$) of TSH level in subjects was (0.19 ± 0.6 vs. 3.19 ± 0.53 IU/L) compared to controls.

Conclusions: TSH-R Ab is the better hyperthyroidism marker, especially in individuals having elevated TNF plus IL-6, in both males as well as females. The close relationship among TSH-R Ab elevation, FT-4 as well as FT-3 concentration in patients having hyperthyroidisms, which is associated with increased immune response activity manifested in TNF and IL-6 accompanied by a decrease in the concentration of TSH in general comparisons and between males and females as a control group.

Keywords: TSH-R Ab (thyroid stimulation hormone receptor antibody); TNF (tumor necrosis factor); INL-6 (interleukin - 6); INL-10 (interleukin - 10).

1. Introduction

1.1. Thyroid Glands

The thyroid gland is one of the important endocrine gland that have many functional activates in the body that located at the front of the neck and resembles a butterfly endocrinology gland. It goes all the way down to the Stern hyoid muscles and stern thyroid. The thyroid gland is involved in many important physiological functions in the body, including regulating the balance of metabolic processes in cells and energy distribution, as well as organ growth [1]. On a cellular level, thyroid hormones exert their influence after being attached to the thyroid hormone receptor via

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predetermined procedures. Substrates of energy Availability, hormones, and other physiologically energetic substances come together to keep plasma thyroid hormone at the optimum amount in order to maintain energy homeostasis [2]. Thyroid hormone activities on metabolism are regulated at the tissue level by Thyroid hormone receptors, trans-membrane transporters, and deiodinases [3].

1.2. Hypothyroidism

Hypothyroidism affects 5% of the general population, with another 5% going unreported [4]. More common in females than in males [5]. This is recognized with a decreasing blood concentration of FT₄ and a TSH serum level who isn't increased among appropriate patients [6]. Symptoms of central hypothyroidism as well as indicators include exhaustion, depression, cold sensitivity, dyspnea, skin dryness, constipation, bradycardia, and hyporeflexia are also symptoms of goiter, however, they are less than those of primary hypothyroidism [7].

1.3. Hyper-thyroidism

Hyper-thyroidism is characterized as a great deal of production as well as release of thyroid hormone is produced by the gland that makes up the thyroid, resulting in an inappropriate rise in blood levels. A disproportionate amount of thyroid hormone will result in an immediate metabolic state [8].

2. Methodology

2.1. Patients group

Fifty patients diagnosed with hyperthyroidism disease (25 males and 25 females).

2.2. Control group

50 subjects in good health as controls (25 males and 25 females).

2.3. Data collection

2.3.1. Inclusion criteria

The hyperthyroidism patients who were diagnosed by physicians of the center.

2.3.2. Exclusion criteria

The patients with renal, liver failure, congestive heart disease and those who treats with thyroxin.

2.3.3. Sample gathering

This study was achieved by gathering 50 blood samples includes to 50 patients (25 male and 25 female) with diagnosed AMI and 50 subjects as control (25 male and 25 female).

2.4. Statistical analytical tests

Statistical tests were conducted using SPSS version 23 and included the variance test, standard deviation, T-test, and ANOVA based on the probability level ($P \geq 0.01$).

3. Results and dissuasion

3.1. Biochemical characteristics

3.1.1. Ages Distribution

The findings (Table 1) reveal that there are no significant variations at patients in contrast to the control group depending on the age periods . the probability level (P -value = 0.63)

3.1.2. TSH-R ab

When compared to controls, the findings of our investigation ($17.04 \pm 5.10 / 1.15 \pm 0.25$ U/l) demonstrate a substantial rise ($P \geq 0.00$) of Ab-TSH receptor levels in the subjects (17.04 ± 5.10 vs. 1.15 ± 0.25 U/l). This finding may be attributed

to an increased immunological reaction to the thyroid gland caused by the production of high levels of TNF as well as IL-6, which may drive This auto immunological-Ab is produced by the immune system's cells, and this concept supports that the condition is a chronic clinical state.

Table 1 Biochemical differences between patients and control group.

Variables	Control	Patients	P (value)
	Mean \pm SD	Mean \pm SD	
Age	22.28 \pm 2.8	20.20 \pm 9.1	0.63
TSH-R ab	1.15 \pm 0.24	17.04 \pm 5.10	0.000
FT-4	1.40 \pm 0.24	3.16 \pm 0.62	0.000
FT-3	0.32 \pm 0.07	0.81 \pm 0.20	0.000
TSH	3.19 \pm 0.58	0.19 \pm 0.06	0.000
TG	22.16 \pm 6.80	40.14 \pm 9.62	0.040

The findings of our investigate are consistent with those of Chen *et al*, [9] who found elevated titer of anti TSH-R Ab (52.35 19.07 p0.001), who stimulates high level of thyroid hormone production [10] , who found an increase in TSH-R Ab (3.77 18.4 p0.001) Al-Mawlah *et al* [11] who found elevated concentration of TSH-R that have thyroid gland surface displayed elevated concentration of TSH-R Ab, increasing thyroid mass as well as manufacturing (12.11 \pm 1.15 p<0.0001) and Mikoś *et al* [12] concur alongside our research, which demonstrated that TSH-R Ab reduces TSH's functional activity and causes disease by binding to the TSH a receptor. (> 10 IU /L).

3.1.3. Free-T4

The findings shown in table (3.2) also revealed a substantial rise (P= 0.00) by FT 4 level in subjects (6.16 \pm 0.62 vs. 1.40 \pm 0.24 ng /dl) compared to controls. We believe that the rise in FT4 levels is due to an increase in TSH R-AB, which destroys TSH receptors on the thyroid gland's surface, causing the thyroid gland to cease producing T4, causing T4 levels to remain high.

We concurred with Cheng *et al*. [9] who noted that rising TSH-R Ab was related to a rise in FT4 (3.32 3.44 ng/dl p \geq 0.001), Zhu *et al*. [10] who stated an enhance in FT4 (23.6 6.15 ng/dl p \geq 0.001), Diana *et al*. [13] whose reported an elevate in FT4 (2.0 1.2 ng/dl p0.001) and Elfadil *et al* [14] who demonstrated a markedly elevated FT4 in Sudanese individuals (32.61 \pm 36.46 ng/dl, p \geq 0.00).

3.1.4. T3 /Free

In our investigation, the T3 level (shown in table 3.2) demonstrates a considerable rise (P \geq 0.00) of patient (2.81 \pm 0.20 vs. 0.32 \pm 0.07 ng/ dl) vs. control. The level of FT3 was increasing as a result of an immunological reaction to thyroid gland surface, which was influenced by FT4 concentration. Zhu *et al*. [10] findings corroborate our inspection's results, that's show FT-3 had been (9.64 3.07 pmol/l p \geq 0.001). results corroborate our findings, which indicate that FT-3 is rising (34.0 5.6 ug/l p \geq 0.001). Diana *et al* [13] findings indicate that FT-3 is enhancing (5.00 1.50 ng/dl p 0.001), and Elfadil *et al* [14] findings indicate which FT-3 Ab significantly increased in Sudanese individuals (21.4 \pm 20.6 pg/mL p< 0/00).

3.1.5. TSH

The findings of our investigation, which were presented in table 3.2, clearly demonstrated a substantial drop (P 0.00) of TSH level in subjects was (0.19 0.6 vs. 3.19 0.53 IU/L) compared to controls. This result indisputably suggests that the TSH level was low due to a rise in the levels of the FT-4 as well as FT-3 hormones. Because of the mirrored link between them, TSH levels remain low while T4 and T3 levels rise. Cheng *et al* [9] concur about our results who TSH hormone levels were lowered due to elevated FT4 (0.02 \pm 0.03 P 0.001); Khamisi *et al* [15] concur with our results how TSH levels were decreasing (0.005 \pm 0.004 ug/ L p 0.001); Al-Mawlah *et al* [11] demonstrated that T4 activity was high (0.68% 0.14 P< 0.0001), and Zhu *et al* [10] demonstrated a reduction in TSH hormone levels of 0.01 \pm 0.02 p< 0.001).

3.1.6. TG

Table (3.2) also revealed a substantial rise (P 0.04) in patients' thyroglobulin levels (40.41 9.62 vs. 22.16 6.80) ng/m. We proposed that by activating auto Ab (TSH-R Ab), we may eliminate the thyroid gland's receptors while maintaining the function of the peroxidase enzyme, which transformed TG into T4. As a result, TG incidence remains significant. Khamisi *et al* [15] concurred alongside our results, that demonstrated the thyrocyte travel vesicles that released into the extra-follicular space by a process regulated by TSH or TRAb are responsible for the rising level of T.G (132–124 g/L El Din *et al* [16] demonstrated concerning with our investigate results regarding TG (412.88 ± 142.5 pg/l p<0.001) due to the actions of autoimmune antibody in opposition to cells of the thyroid gland [10]. agreed with our findings that increasing in T.G Ab (22.5 4.00 ng/dl p0.001).

3.2. Immune characteristics

Table 2 Evaluation of some Immune parameters between patient and control

Variables	Control	Patients	P values
	Mean ±SD	Mean ±SD	
INL-6	18.14 ± 1.29	28.03 ±2.80	0.000
IL-10	12.66 ± 1.58	22.87 ± 3.76	0.000
TNF	8.63 ± 0.80	18.78 ± 3.95	0.000

3.2.1. IL-6

When compared to controls, the findings of our investigation (shown in table 2) demonstrate a substantial rise (P=0.00) of INL-6 level in patient (28.03± 2.80 vs. 18.14± 1.29 ng/dl). This might be related to an increase in the immunological reaction to oneself, which confuses thyroid gland cells toward T-cells, resulting in the creation of auto-Abs. Al-Mawlah *et al* [17] discovered that subjects with hyperthyroidism had higher serum concentrations of IL-6 (3.82 4.03 pg/ml, p 0.001), and Kumar *et al* [18] found that orbital IL-6 may originate from the activity of adipogenesis within tissue and adipocytes themselves. Salvi *et al* [19] found that elevated levels of INL-6 elevated thyroid follicular cell proliferation. contrary to studies mentioned above, our findings contradict those of Cheng *et al* [9], who found no significant changes in INL-6 levels.

3.2.2. IL-10

Our results in table (2) also revealed a substantial rise (P 0.00) in IL-10 levels in patients (22.87 3.76 vs. 12.66 1.58 ng/ml) compared to controls. This rise in IL-10 concentration may be attributed to an increase in the second self-immune response for T-cells against thyroid gland follicular cells, which leads to the creation of self Ab. This study's findings were supported by Cheng *et al.* [9], Khamisi *et al* [15], El Din *et al* [16] and Mikoś *et al* [19]

3.2.3. TNF

Our results in table (2) also revealed a substantial rise (P 0.00) of TNF level in subjects (18.78 3.95 vs. 8.63 0.80 ng/ml) compared to controls. We believe that the increase in TNF levels is due to a faulty translation for monocytes, which drive the initial immune response, such that it attacks thyroid gland cells as self Ag and then completes its work by stimulating T-cells. Our results are in line with those of Cheng *et al* [9], who discovered a connection between TNF, a primary immune stimulant, and TSH-R Ab. TNF was shown to be (13.8 3.87 pg/ml, p 0.001) by Zhu *et al* [10], and TNF inhibits the growth and development of thyroid cells by Mikoōs *et al* [12]. Cheng *et al* [9] concurred with our findings that elevated TNF contributes to thyroid impulsivity.

3.3. Biochemical characteristics between male and female vs control group

3.3.1. Thyrotropin Receptor Antibody according to the sex

The findings of our investigation, as shown in table (3), demonstrate a substantial rise (P 0.001) of TSH-receptor Ab level in thyroid of men was (15.36± α 5.12 α vs. α 1.03± α± 0.23 α U/L α) and female α (17.98 α ±6.89 α vs. α 1.26± α 0.17 α U/L α). We think that a significant rise in TSH-R Ab is associated with an immune response decreases due to the decreased effect of thyroid hormones. Elfadil *et al* [14] demonstrated that the significantly elevated TSH-R Ab in Sudanese patients was (9.32 7.71 U/Ml p 0/00). Leschik *et al* [20]. Researcher Abdel Abbas *et al* [21] mentioned that THS-R Ab has an important role in influencing natural receptors by stimulating cyclic adenosine monophosphate, so

measuring TSH-R Ab can help us diagnose patients with hyperthyroidism, in addition Diana *et al* [13] agreed with our investigation while they had recorded that TSH-R Ab increased in both euthyroid as well as hyperthyroid subjects.

Table 3 Biochemical differences of male's vs females

Variables	Gender	Patients	Control	P-value
		Mean \pm SD	Mean \pm SD	
Thyrotropin Receptor Antibody (U/L)	Male	15.36 \pm 5.12	1.03 \pm 0.23	0.000
	Female	17.98 \pm 6.89	1.26 \pm 0.17	0.000
Thyroxine (T4) (ng/dl)	Male	3.31 \pm 0.81	1.35 \pm 0.29	0.001
	Female	3.08 \pm 0.47	1.46 \pm 0.23	0.004
Triiodothyronine (T3) (ng/dl)	Male	2.78 \pm 0.21	0.33 \pm 0.08	0.001
	Female	3.75 \pm 0.22	0.31 \pm 0.07	0.000
Thyroid-stimulating hormone (μ IU/L)	Male	0.20 \pm 0.06	3.32 \pm 0.49	0.000
	Female	0.18 \pm 0.04	3.03 \pm 0.64	0.000
Thyroglobulin (Mg/dl)	Male	36.62 \pm 11.64	21.58 \pm 6.78	0.820
	Female	42.11 \pm 12.07	23.15 \pm 7.44	0.077

3.3.2. The Free-T4 between male and female

When compared to controls, our results in table (3) demonstrated a substantial rise ($P \geq 0.001$) in free T4 thyroid levels of thyroid men (3.31 \pm 0.81 vs. 1.35 \pm 0.29 ng/dl) and female (3.08 \pm 0.47 / 1.46 \pm 0.23 ng /dl). We believe that the increase in free T4 levels is due to the development of TSH -R Ab or to a minimize levels of TSH . Cheng *et al* . [9] concurred with our findings, indicating that elevated cytokines were linked to elevated FT-4. Leschik *et al* [20] demonstrated the high value of FT-4 (2.3 \pm 0.17 ng/dl P 0.00), and Díez *et al* [22] demonstrated the association between hyperthyroidism and elevated FT-4 (49.34 \pm 13.20 pmol/L p = 0.001).

3.3.3. Free-T3 between male and female

In our investigation, the free T3 level was found to be significantly higher ($P \geq 0.001$) of FT-4 male subjects was (2.78 \pm 0.21 vs. 0.33 \pm 0.08 ng /dl) while females were (3.75 \pm 0.22 vs. 0.31 \pm 0.07 ng /dl). As with FT4, we believe that the rise of FT-3 is due to the similar process that produces the increases of FT-4. Leschik *et al* [20] demonstrated the elevated level of FT-3 (10.71 \pm 1.38 ng/dL $P \geq 0.00$), Kumar *et al* [18] hypothesized who TSH -R Ab could have the potential to facilitate InL -6 production and secretion by mature adipocytes, as well as Salvi *et al* [19] agreed this study that recorded; those who have hyperthyroidisms resulted of elevated with FT-3 (12.85 \pm 10.61 pmol/l $p \geq 0.001$).

3.3.4. TSH between male and female

The findings of our investigation, as shown in table (3), clearly demonstrated a substantial drop ($P \geq 0.001$) of TSH level of thyroid men subjects was (0.20 \pm 0.06 vs. 3.32 \pm 0.49 IU/L) vs women were (0.18 \pm 0.04 vs. 3.03 \pm 0.64 IU/L). There is little question that the large decrease in TSH is closely connected to the increase in thyroid secretion caused by immunological response. Elfadil *et al* [14] demonstrated the critical decrease of TSH with Sudanese patients (0.02 \pm 0.01 U/L p 0.00) and Díez *et al* [22] concurred with our findings that TSH (0.05 \pm 0.05 mU/L p 0.001) was reduced by hyperthyroid production. Diana *et al* [13] illustrates this decrease in TSH to 1.6 \pm 0.01 ng/dl p 0.001).

3.3.5. TG between male and female

The findings of our investigation, as shown in table (3), clearly demonstrated a substantial rise (P 0.001) of thyroglobulin level of thyroid's men subjects were (36.62 \pm 11.64 vs. 21.58 \pm 6.78 ng/mL) while women were (42.11 \pm 12.07 vs. 23.15 \pm 7.44 ng/mL). We propose that the increase in TG is related to the high activity of the thyroid gland, which consumes a large quantity of TG as a precursor to thyroid hormones. Diana *et al* [13] indicated an increase in T.G (519 - 7 UI/L $p \geq 0.001$), Hashim *et al* [23] indicated an increase in T.G (25.295.83UI/L p 0.001), and El Din *et al* [13] indicated an increase in T.G (412.88 \pm 142.5 pg/l p 0.001) owing for the auto immune's action Abs towards cells of thyroid gland .

3.4. Immune characteristics between male and female

Table 4 Immune differences of male's vs females

Variables	Gender	Patients	Control	P-value
		Mean \pm SD	Mean \pm SD	
InL-6	Male	28.08 \pm 2.51	18.35 \pm 1.09	0.000
	Female	28.00 \pm 2.99	17.93 \pm 1.45	0.001
InL-10	Male	23.17 \pm 3.82	12.63 \pm 1.65	0.001
	Female	22.71 \pm 3.77	12.69 \pm 1.55	0.000
TNF	Male	20.54 \pm 4.62	8.62 \pm 0.78	0.000
	Female	17.78 \pm 3.18	8.64 \pm 0.83	0.000

3.4.1. InL-6 comparing of males & females

The results of our study showed that the IL-6 level in male subjects (28.08 2.51 vs. 18.35 1.09 ng/dl) was significantly higher than that in female subjects (28.00 2.99 vs. 17.93 1.45 ng/dl) (P 0.001). We propose that a high level of IL-6 is linked to an increase in TNF as the key motor of immune response or into an increase in TSH-R Ab. The results of our investigation corroborated the findings of Salvi *et al* [19], which stated that people with hyperthyroidism have elevated levels of IL-6 (3.2 0.4 pg/ml, p 0.001), Shukla *et al* [24] which reported that the serum concentration of IL-6 increased in subjects with hyperthyroidism (11.12.3 pg/ml, p 0.001), and Kumar *et al* [18] who noted that adipogenesis activity within tissue could be a source of orbital IL-6. Contrary to the studies mentioned above, our findings contradict those of Cheng *et al* [9], who found no significant changes in IL-6 levels.

3.4.2. IL-10 comparing of male & female

In our study, the IL-10 level (shown in table 4) reveals a substantial rise (P 0.001) of IL-10 male patients (23.17 3.82 vs. 12.63 1.65 ng/dl) and females (22.71 3.77 vs. 12.69 1.55 ng/dl). This rise in IL-10 content in both females and males may be owing to an increase in the second self-immune response to thyroid gland follicular cells for T-cells, which leads to the creation of self-Ab. Our data of this study was agreed with Cheng *et al* [9], Khamisi *et al* [15], El Din *et al* [16] and Mikoś *et al* [12]

3.4.3. TNF level comparing of male & female

The TNF-level measured through our work, as shown into (Table 3.4), demonstrates a substantial rise P value 0.001 in TNF males subjects was (20.54 \pm 4.62 vs. 8.62 \pm 0.78 ng/dl) while females were (17.78 \pm 3.18 vs. 8.64 \pm 0.83 ng/dl). We believe that the increase in TNF is attributable to the immune system's tenacity in attacking the thyroid gland's follicular cells. Our research's conclusions supported the findings of Mikoś *et a* [12], which demonstrated that TNF inhibits the growth and development of thyroid cells. Díez *et al* [22] concurred with our findings, which established that patients with thyroid disorders release TNF at significant levels (3.36 \pm 1.21 pg/ml, P<0.001), and Salvi *et al* [19] agreed with our findings, which showed increasing in the patients thyroid hormones via elevated of TNF concentration (4.4 \pm 0.4 pg/ml, p \geq 0.001).

3.5. Association between thyroid disease severity and the parameters analyzed

Tables 1 and 3 shows that the TSH-R Ab is a more reliable protein marker of hyperthyroidism disease, and more correlated with the severity of illness. In addition theses tables shows correlations between TSH-R Ab levels and some immune, physiological parameters comprehend the link between each parameter and the severity of the illness. A t-test was used to correlate TSH-R Ab with the parameters under investigation; a significant association was shown at (P \geq 0.05).

3.5.1. Association of TSH-R Ab and FT-4

As demonstrated in Figure (1) and table (5), our data of this investigation revealed that the substantial positive association of TSH-R Ab and FT-4, (r= -0.91) as well as (P 0.05).

Table 5 Association of TSH-R Ab and FT-4

Thyrotropin Receptor Antibody	R	P Value	Results
FT4	0.91	0.05	Positive correlation

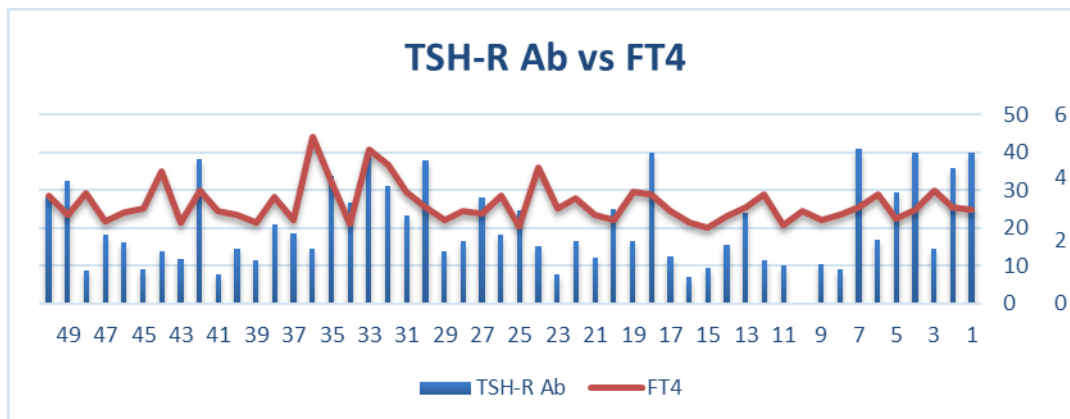


Figure 1 The Association between FT4 and TSH-R

3.5.2. Association of TSH-R Ab and FT3

Figure (2) and table (6) clearly indicate a substantial positive connection between TSH-R Ab and FT3, ($r = -0.76$) as well as ($P 0.05$).

Table 6 A relationship between TSH-R Ab and FT-3

Thyrotropin Receptor Antibody	R	P Value	Results
FT3	0.76	0.05	Positive correlation

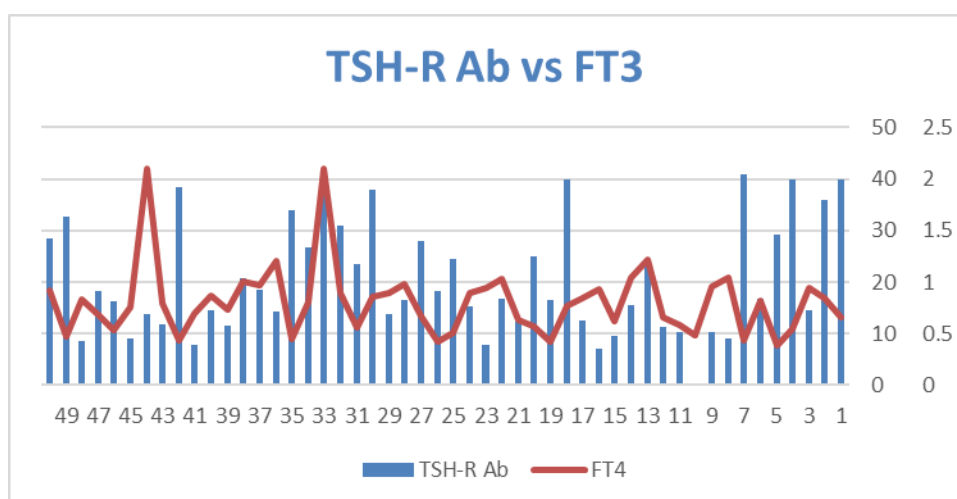


Figure 2 The Association between FT3 and TSH-R Ab

3.5.3. Association of TSH-R Ab and TSH

Our results show a significant negative correlation ($r = -1.00$), ($P 0.05$), between TSH-R Ab and TSH in Figure (3) and Table (7) of this investigation.

Table 7 A relation of TSH-R Ab and TSH

Thyrotropin Receptor Antibody	R	P Value	Results
TSH	-1.00	0.05	Negative correlation

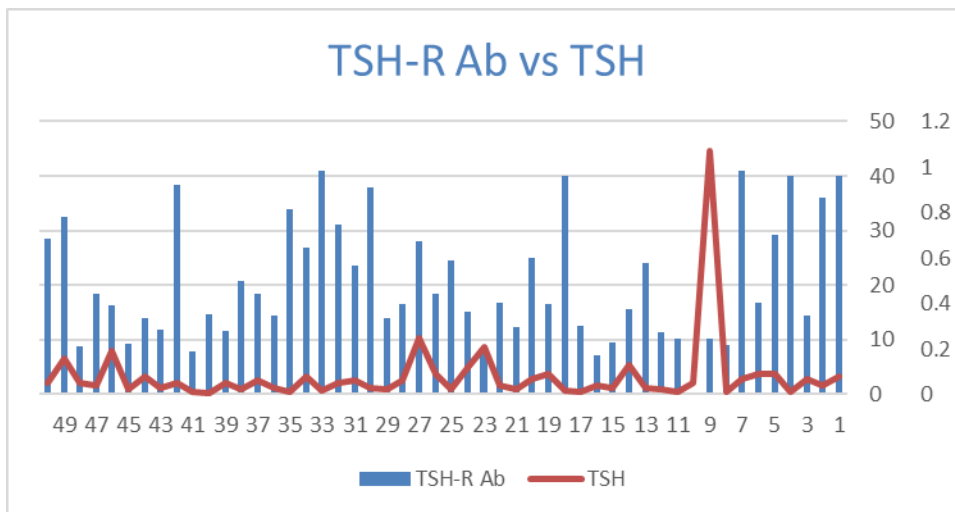


Figure 3 The Association between TSH and TSH-R Ab

3.5.4. Association of TSH-R Ab and TG

Figure (4) and table (8) indicate a substantial positive connection between TSH-R Ab and T.G (r= 1.00) as well as (P= 0.05).

Table 8 A relation of TSH-R Ab and T.G

Thyrotropin Receptor Antibody	R	P Value	Results
TG	0.53	0.05	Positive correlation

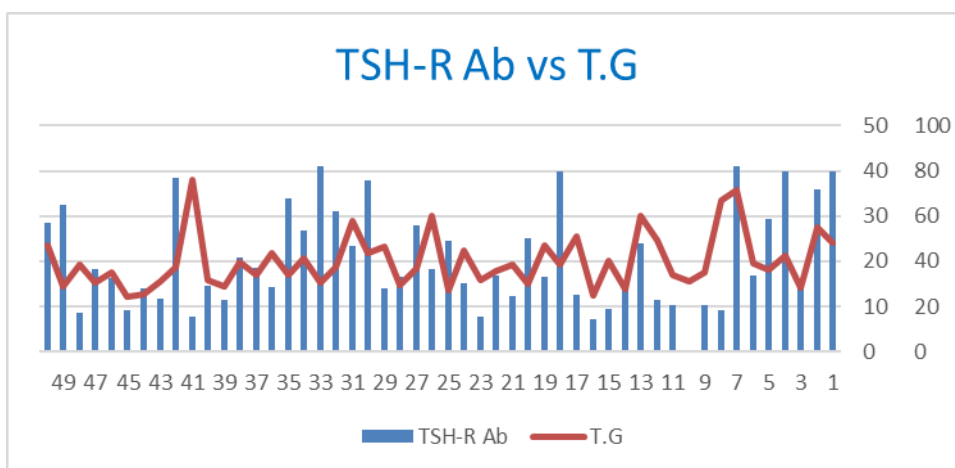


Figure 4 The Association between TG and TSH-R Ab

3.5.5. Association of TSH-R Ab and TNF

Figure (5) as well as Table (9), ($r = -1.00$), ($P < 0.05$), reveal a substantial positive connection among TSH-R Ab and TNF.

Table 9 A relation of TSH-R Ab and TNF

Thyrotropin Receptor Antibody	R	P Value	Results
TNF	0.55	0.05	Positive correlation

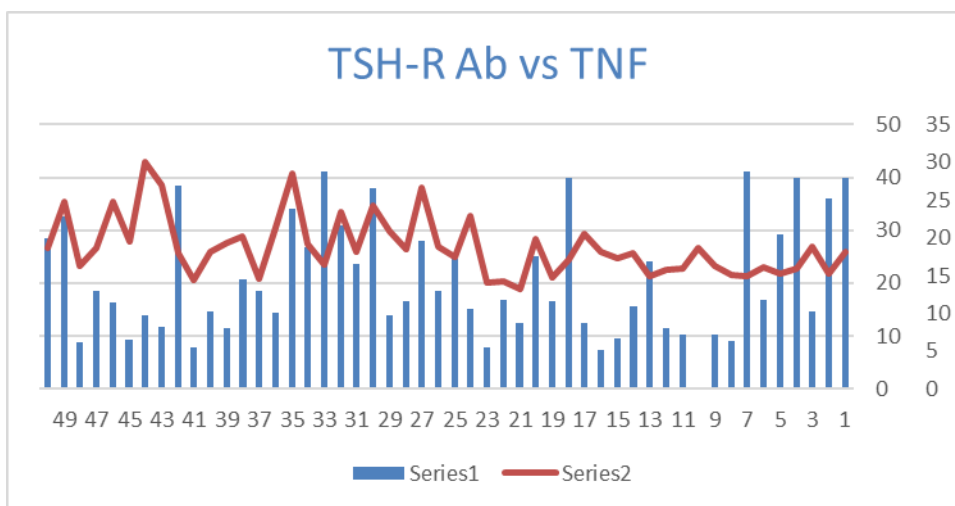


Figure 5 The Association between TSH-R Ab and TNF

3.5.6. Association of TSH-R Ab and IL-6

Figure (6) shows a substantial positive connection of TSH-R Ab as well as IL-6 ($r = -1.00$), ($P < 0.05$).

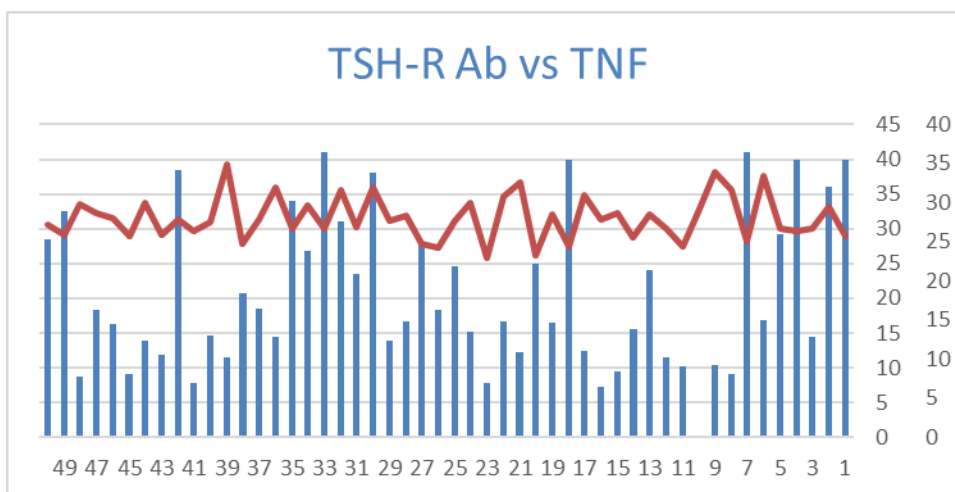


Figure 6 The Association between IL-6 and TSH-R Ab

We believe that positive connection among TSH-R Ab, FT-4, FT-3, as well as T.G seen into figures (3.1, 3.2, and 3.4, respectively) is as a result of the continued existence of the strong immunological response towards thyroid follicular cells caused by increased TNF and IL-6 concentrations. A similar explanation exists for the negative relationship of TSH-R Ab as well as TSH hormone. Our findings match with those of El Din *et al* [16], who found a link among TSH-R Ab capacities and serum FT-4, FT-3, as well as TSH. This association may be noted through the activity of TSH-R Ab within the receptors of TSH, which prompts the thyroid cell toward secrete an excessive quantity of thyroid gland

hormones, leading to hyperthyroidism, as well as which puts the association of our results' investigate in agreement to Zhu *et al* [10], who claimed that TNF as well as TSH-R Ab are positively associated.

4. Conclusions

TSH-R Ab is the better hyperthyroidism marker, especially in individuals having elevated TNF plus IL-6, in both males as well as females. There is a strong correlation between elevated TSH-R Ab, elevated FT-4 and FT-3 concentration, and hyperthyroidism in patients. This is linked to increased immune response activity, which is shown in TNF and IL-6, and decreased TSH concentration in both general comparisons and when comparing males and females versus controls. Because of thyroid secretion hyperactivity in both genders, there are no significant changes between TSH-R Ab and FBS, HDL, and a substantial reduction with S.C, TG, and LDL-C. TSH-R Ab has a substantial positive connection into FT-4, FT-3, T.G, TNF, plus IL-6, but a negative correlation into TSH.

Compliance with ethical standards

Acknowledgement

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Disclosure of conflict of interest

Authors have no conflict of interest to be declared.

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