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Evaluation of Serum Thyroid Hormones (T3, T4) and TSH in Graves' Disease Patients in Orlu, Imo State

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Abstract

The study assessed the thyroid markers in individuals with Graves' Disease in Orlu, Imo State. This study comprised eighty (80) individuals with Graves' Disease, aged 30 to 60 years, encompassing both genders (males and females), categorised into two groups: group 1 (those without Graves' orbitopathy, GO) and group 2 (those with Graves' orbitopathy). Both groups had the same number of study subjects, forty (40) each, and were matched for age and gender. We used several Elisa technique test kits to assess T3, T4, and TSH. The analysis was done with SPSS statistical computer software (version 21). The study revealed that serum levels of free T3 and T4 were markedly elevated compared to their respective controls in both groups of GD; free T3 pg/ml (5.35± 1.88; control 3.00± 0.78) and free T4 pg/ml (2.38± 0.82; control 1.24± 0.30) for males, and (6.55± 1.31; control 4.00± 0.78) and (2.88± 0.77; control 1.95± 0.60) for females. Conversely, TSH levels were considerably lower compared to the control group for both GD groups; TSH miu/L (1.16± 1.21; control as 2.41 ± 1.21) for males and (0.89± 1.77; control as 2.02± 0.89) for females. There was a substantially lower mean free T3 in GD patients without GO (group 1) (3.72 ± 0.66) compared to those with GO (group 2) (6.99 ± 1.08) for males and (group 1) (3.93 ± 0.78) compared to (group 2) (7.43 ± 1.54) for females. Conversely, T4 and TSH levels were considerably elevated in GD participants in group 1 compared to group 2; T4 levels were 3.04 ± 0.61 and 1.72 ± 0.32, while TSH levels were 0.17 ± 0.06 and 2.15 ± 0.98 for males and 3.96 ± 0.97 and 2.01 ± 0.75 for females, respectively. In a similar way, TRAb and TSI were much higher in the two groups of GD than in their controls. For males, TRAb iu/L was (2.96 ± 1.14; control as 1.23 ± 0.41) and TSI iu/L was (3.45 ± 1.07; control as 0.87 ± 0.32). For females, TRAb iu/L was (3.14 ± 1.77; control as 1.53 ± 0.98) and TSI iu/L was (3.98 ± 2.07; control as 1.02 ± 0.78). On the other hand, TRAb and TSI mean serum levels were much lower in group 1 than in group 2; for men, TRAb was 2.15± 0.64 (control as 3.72± 0.92) and TSI was 2.68± 0.56 (control as 4.23 ± 0.88); for females, TRAb was 2.75± 0.96 (control as 1.17± 0.45) and TSI was 2.98± 0.73 (control as 1.14± 0.58).

Keywords: *Serum Thyroid Hormones, TSH, GRAVES' DISEASE, ORLU, IMO.*

INTRODUCTION

Graves' disease is an autoimmune disorder that primarily affects the thyroid gland, resulting in hyperthyroidism, or overactivity of the gland. The most prevalent aetiology of hyperthyroidism worldwide, especially in women aged 30 to 60 years [1]. The illness happens when the immune system makes thyroid-stimulating immunoglobulins (TSIs) that attach to the thyroid-stimulating hormone (TSH) receptor. This makes the thyroid gland work too hard. This over-stimulation causes the thyroid to make too many hormones, which control metabolism, growth, and how the body uses energy [2]

Graves' disease is clinically characterised by a constellation of symptoms, including goitre (thyroid enlargement), heat sensitivity, weight loss, palpitations, anxiety, and tremors [3]. Ophthalmopathy is a unique characteristic of Graves' illness, manifesting as exophthalmos, redness, and swelling in about 25–50% of patients [4]. Dermopathy, including pretibial myxedema, may manifest in some instances.

The precise aetiology of Graves' illness remains unidentified; however, it is thought to arise from a confluence of genetic predisposition and environmental factors, including stress, smoking, infections, and potentially iodine exposure [5]. A family history of autoimmune illnesses substantially elevates the likelihood of acquiring Graves' disease, suggesting a genetic factor [6].

Antithyroid drugs (such methimazole), radioactive iodine therapy, and, in some circumstances, surgery to remove the thyroid gland are all ways to treat Graves' illness. Treatment is generally tailored to the patient's age, illness severity, and comorbidities [7]

It is important to know the history of Graves' illness in order to make a quick diagnosis, teach patients, and provide them the best care possible. This is especially true because the disease can have systemic effects and cause long-term problems if not treated [8]

Autoimmunity is a disorder in which the immune system mistakenly attacks the body's own tissues, causing long-term inflammation and damage to those tissues. The thyroid gland is one of the organs that autoimmune disorders most often affect. Graves' disease and Hashimoto's thyroiditis are two examples of autoimmune thyroid diseases (AITDs). They are the two most common causes of hyperthyroidism and hypothyroidism, respectively [9]

Hashimoto's thyroiditis is characterised by the destruction of thyroid tissue by autoreactive lymphocytes and the presence of antithyroid peroxidase (TPO) and antithyroglobulin antibodies, resulting in a progressive reduction in thyroid function and the onset of hypothyroidism [10]

Both disorders exhibit a multifaceted interaction of genetic susceptibility, environmental factors, and immune system dysregulation. Family and twin studies have shown that AITDs have strong genetic components [11]. Environmental factors, including infection, stress, iodine consumption, and smoking, have been associated [12]. Comprehending the autoimmune aetiology of thyroid diseases is crucial for prompt diagnosis, suitable therapy, and enhanced patient outcomes [13]. Immunology and molecular biology have made huge strides in helping us understand how these diseases start and how to treat them [14] The increasing global incidence of autoimmune thyroid disorders, such as Graves' disease, has elicited heightened concern among public health professionals and endocrinologists. Consequently, additional research into its pathogenesis, diagnosis, and treatment [15] is necessary. However, early detection and consistent adherence to therapy continue to pose hurdles due to a confluence of insufficient public knowledge, healthcare inequities, and socio-economic obstacles [16]. Moreover, although the aetiology of Graves' disease is associated with genetic and environmental variables, the specific triggers and underlying immunological mechanisms remain unclear [17] Even while diagnostic technologies and treatment options have gotten better, there is still a vacuum in understanding. Given the considerable physical, emotional, and financial costs associated with Graves' illness, it is imperative to examine its contributing factors, recurrence patterns, and effective treatments techniques. Research is particularly essential in developing places where access to specialised treatment is constrained and cultural beliefs may affect health-seeking behaviour. Without these kinds of measures, the burden of this disease would probably get worse, especially for people who are already at risk. In the same way, this study is timely and appropriate because of inconsistent reports and the rise in hyperthyroidism cases in Nigeria, especially in Imo State, which is the study area. This study investigated the thyroid indicators in patients with different manifestations of Graves' disease attending the Endocrinology Clinic at Imo State University Teaching Hospital in Orlu, Imo State.

In general, looking at these indicators in Orlu will be one aspect of a larger effort to better understand, diagnose, and treat Graves' illness in Imo State, Nigeria. It connects local health issues with worldwide scientific knowledge and makes it easier for doctors in the community to treat thyroid diseases.

MATERIALS AND METHODS.

Study Area

This study was carried out in Imo State University Teaching Hospital, Orlu. This tertiary hospital serves as referral hospital in South-East as it offers a comprehensive range of services (internal medicine, endocrinology, immunology etc).

Advocacy, Mobilization and Pre-survey contacts

The ethical approval was obtained from IMSUTH ethical committee. Informed consent was sought from patients, after which the date for sample collection was fixed.

Study Population / Sample Size.

A. Sample Size.

The sample size was determined using Ejemot- Nwadiaro (2009) (see appendix vi). The sampling technique was a targeted random sampling technique.

B. Study Population.

Population include Patients with Graves' Disease made up of male and female patients within the Age range of 30 to 60 years old.

A total of 80 patients with GD, including those without and with GO disease and 80 control subjects were recruited for the study. The sampling was done using Targeted random sampling technique for all the consented participants.

Selection Criteria

A. Inclusion Criteria

1. Subjects with the history of Graves' disease confirmed with clinical activity score (CAS) who are within the ages of 30-60 years and who gave their consent were recruited for the study.
2. GD patients (Male or Female) without or with GO
3. GD patients without other metastatic diseases.

B. Exclusion Criteria.

1. Subjects that were below 30 years and above 60 years were excluded.
2. Those from whom informed consent could not be obtained were also excluded.
3. Those with other metastatic diseases.

Study Design

This is a case control, cross sectional study. A total of 80 patients who attended endocrinology clinic for treatment and surgery of GD at IMSUTH were recruited for the study.

They included male and female patients within the ages of 30-65 years old.

The study subjects were stratified into 2 groups viz;

Group 1: Patients without Graves' orbitopathy (GO) with their matched controls
Group 2: Patients with Graves' orbitopathy with their matched controls

The control group involved a total of 80 subjects without Graves' Disease who were Age and Gender matched with the study subjects to eliminate the influence of these factors on the study outcome.

These subjects were recruited using a standard questionnaire to obtain information on demography and clinical history after their consents were secured.

Their blood samples were collected and used for laboratory determination of the following markers; Thyroid hormones (T3, T4), TSH.

SAMPLE COLLECTION FOR GRAVES' DISEASE DETERMINATION

Blood samples were collected using Veno-puncture method some were put in a clean sample container, allowed to clot while others were put in EDTA container. The sera were separated from the clotted samples by centrifugation and stored-frozen (-20 degree centigrade) in a chemically clean sample bottles prior to analysis.

Laboratory Procedures

The kits were commercially purchased and the manufacturers' SOPs were strictly adhered to.

Determination of T3 (Triiodothyronine), T4 (Thyroxine). and TSH (Thyroid Stimulating Hormone) were determned by ELISA METHOD.

RESULT

Table 4.1: Mean ± SD Values of Serum Thyroid Hormones (T3, T4) and TSH in Male Hyperthyroidism Subjects Versus All Controls

Variable (mean ± SD)	Hyperthyroidism subjects (n=40)	Control subjects (n=40)	t-value	p-value	Hyperthyroidism subjects (n=40)	Control subjects (n=40)	t-value	p-value
freel T3 (pg/nl)	5.35 ± 1.88	3.00± 0.78	7.293	0.000	6.55 ± 1.31	4.00 ± 0.78	8.542	0.000
Lower 95% C.I	4.75	2.74			5.65	3.65		
Upper 95% C.I	5.95	3.25			6.85	4.86		
free T4 (pg/nl)	2.38 ± 0.82	1.24± 0.30	8.111	0.000	2.88 ± 0.77	1.95 ± 0.60	8.765	0.000
Lower 95% C.I	2.11	1.14			2.35	1.23		
Upper 95% C.I	2.64	1.33			3.23	1.94		
TSH (miu/ml)	1.16 ± 1.21	2.41± 1.21	-5.447	0.000	0.89 ± 1.77	2.02 ± 0.89	-4.564	0.000
Lower 95% C.I	0.77	2.02			0.54	1.88		
Upper 95% C.I	1.54	2.79			1.22	2.35		

Statistical analysis

Data obtained from the study was presented in the form of tables, while the results were analysed using SPSS statistical computer software (Version 21). Students T- test, Correlation, mean and standard deviations were determined. The values expressed as mean ± S.D. The level of significance was set at 95% confidence interval.

4.1 Mean ± SD Values of Serum free Thyroid Hormones (T3, T4) And TSH in all Male and Female Hyperthyroidism Subjects Versus All Controls

There was a significantly higher (p = 0.000) Mean free T3 in Male Hyperthyroidism subjects compared to male control subjects. There was a significantly higher (p = 0.000) Mean free T4 in Male Hyperthyroidism subjects compared to male control subjects. There was significantly lower (p = 0.000) Mean TSH in Male Hyperthyroidism subjects compared to control subjects (Table 4.1).

There was a significantly higher (p = 0.000) Mean free T3 of the Female Hyperthyroidism subjects compared to female control subjects. There was a significantly higher (p = 0.000) Mean free T4 in Female Hyperthyroidism subjects compared to control subjects. There was significantly lower (p = 0.000) Mean TSH in Female Hyperthyroidism subjects female compared to control subjects (Table 4.1).

Table 4.2: Mean ± SD Values of Serum Thyroid Hormones (T3, T4) and TSH in Male and Female Hyperthyroidism Subjects without Graves Orbitopathy Versus Controls Subjects

	MALE SUBJECTS				FEMALE SUBJECTS			
Variable (mean ± SD)	**Hyperthyroidism Subjects Without G. O. (n=40)**	**Control subjects (n=40)**	**t-value**	**p-value**	**Hyperthyroidism Subjects Without G. O. (n=40)**	**Control subjects (n=40)**	**t-value**	**p-value**
free T3 (pg/nl)	3.72 ± 0.66	3.10± 0.16	3.995	0.001	3.93 ± 0.78	3.44 ± 0.16	4.115	0.001
Lower 95% C.I	3.40	2.76			3.55	3.02		
Upper 95% C.I	4.03	3.43			4.24	3.77		
free T4 (pg/nl)	3.04 ± 0.61	1.19 ± 0.31	13.66	0.000	3.96 ± 0.97	1.54 ± 0.51	14.26	0.000
Lower 95% C.I	2.75	1.04			3.22	1.43		
Upper 95% C.I	3.32	1.33			4.22	1.95		

TSH (miu/ml)	0.17 ± 0.06	2.30 ± 1.31	-7.163	0.000	0.09 ± 0.14	1.98 ± 1.41	- 6.299	0.000
Lower 95% C.I	0.13				0.04			
Upper 95% C.I	0.20	1.68			1.15	1.36		
		2.91				2.43		

4.2. Mean ± SD Values of Serum free Thyroid Hormones (T3, T4) And TSH in Male and Female Hyperthyroidism Subjects without Graves Orbitopathy Versus Control Subjects

There was a significantly higher (p = 0.001) mean free T3 in Male Hyperthyroidism subjects without Graves Orbitopathy compared to male control subjects. There was a significantly higher (p = 0.000) Mean free T4 in Male Hyperthyroidism subjects without Graves Orbitopathy compared to male control subjects. There was significantly lower (p = 0.000) Mean TSH in Male Hyperthyroidism subjects without Graves Orbitopathy compared to male control subjects (Table 4.2).

There was a significantly higher (p = 0.001) mean free T3 in Female Hyperthyroidism subjects without Graves Orbitopathy compared to the female control subjects. There was a significantly higher (p = 0.000) Mean free T4 in Female Hyperthyroidism subjects without Graves Orbitopathy compared to female control subjects. There was significantly lower (p = 0.000) Mean TSH in Female Hyperthyroidism subjects without Graves Orbitopathy compared to female control subjects (Table 4.2).

Table 4.3. Mean ± SD Values of Serum Thyroid Hormones (T3, T4) and TSH in Male and Female Hyperthyroidism Subjects with Graves Orbitopathy Versus Controls Subjects

	MALE SUBJECTS				FEMALE SUBJECTS			
Variable (mean ± SD)	**Hyperthyroidism Subjects With G. O. (n=40)**	**Control subjects (n=40)**	**t-value**	**p-value**	**Hyperthyroidism Subjects With G. O. (n=40)**	**Control subjects (n=40)**	**t-value**	**p-value**
free T3 (pg/nl)	6.99 ± 1.08	2.9 ± 0.85	13.858	0.000	7.43 ± 1.54	3.22 ± 0.97	14.128	0.000
Lower 95% C.I	6.48	2.50			6.75			
Upper 95% C.I	7.49	3.29			7.98	2.97 3.88		
free T4 (pg/nl)	1.72 ± 0.32	1.29 ± 0.28	4.368	0.000	2.01 ± 0.75	1.55 ± 0.83	4.854	0.000
Lower 95% C.I	1.57				1.88	1.35		
Upper 95% C.I	1.87	1.15 1.42			2.23	1.86		
TSH (miu/ml)	2.15 ± 0.98	2.52 ± 1.12	-1.721	0.101	1.94 ± 0.76	2.14 ± 1.41	-1.654	0.131
Lower 95% C.I	1.68				1.23	1.85		
Upper 95% C.I	2.16	1.99 3.04			2.02	2.86		

4.3. Mean ± SD Values of Serum free Thyroid Hormones (T3, T4) And TSH in Male and Female Hyperthyroidism Subjects with Graves Orbitopathy Versus Control Subjects

There was significantly higher (p =0.000) Mean free T3 in Male Hyperthyroidism subjects with Graves Orbitopathy compared to male control subjects. There was significantly higher (p =0.000) Mean free T4 in Male Hyperthyroidism subjects with Graves Orbitopathy compared to male control subjects. There was no significant difference (p = 0.101) in Mean free TSH of Male Hyperthyroidism subjects with Graves Orbitopathy compared to male control subjects. (Table 4.3).

There was significantly higher (p =0.000) Mean free T3 in Female Hyperthyroidism subjects with Graves Orbitopathy compared to female control subjects. There was significantly higher (p =0.000) Mean free T4 in Female Hyperthyroidism subjects with Graves Orbitopathy compared to female control subjects. There was no significant difference (p = 0.101) in Mean free TSH in Female Hyperthyroidism subjects with Graves Orbitopathy compared to female control subjects. (Table 4.3).

Table 4.4: Mean ± SD Values of Serum Thyroid Hormones (T3, T4) and TSH in Male and Female Hyperthyroidism Subjects without Graves Orbitopathy Versus All Male and Female Hyperthyroidism Subjects with Graves Orbitopathy

	MALE SUBJECTS				FEMALE SUBJECTS			
Variable (mean ± SD)	**Hyperthyroidism Subjects Without G. O. (n=20)**	**Hyperthyroidism Subjects With G. O. (n=20)**	**t-value**	**p-value**	**Hyperthyroidism Subjects Without G. O. (n=20)**	**Hyperthyroidism Subjects With G. O. (n=20)**	**t-value**	**p-value**
free T3 (pg/nl)	3.72 ± 0.66	6.99 ± 1.08	-12.378	0.000	3.85 ± 0.67	7.01 ± 1.21	-12.978	0.000
Lower 95% C.I	3.40	6.48			3.44	6.65		
Upper 95% C.I	4.03	7.49			4.11	7.65		
free T4 (pg/nl)	3.04 ± 0.61	1.72 ± 0.32	8.678	0.000	3.54 ± 0.76	1.84 ± 0.67	8.978	0.000
Lower 95% C.I	2.50	1.57			2.85	1.69		
Upper 95% C.I	3.32	1.87			3.75	1.96		
TSH (miu/ml)	0.17 ± 0.06	2.15 ± 0.98	-9.000	0.000	0.12 ± 0.42	2.04 ± 0.75	-8.000	0.000
Lower 95% C.I	0.13	1.68			0.09	1.56		
Upper 95% C.I	0.20	2.61			0.16	2.46		

4.4 Mean ± SD Values of Serum free Thyroid Hormones (T3, T4) And TSH in Male and Female Hyperthyroidism Subjects without Graves Orbitopathy Versus Male and Female Hyperthyroidism Subjects with Graves Orbitopathy

There was significantly lower (p =0.000) Mean free T3 in Male Hyperthyroidism subjects without Graves Orbitopathy compared to the Male Hyperthyroidism subjects with Graves Orbitopathy. There was significantly higher (p =0.000) Mean free T4 in Male Hyperthyroidism subjects without Graves Orbitopathy compared to the Male Hyperthyroidism subjects with Graves Orbitopathy. There was significantly lower (p =0.000) Mean free TSH in Male Hyperthyroidism subjects without Graves Orbitopathy compared to the Male Hyperthyroidism subjects with Graves Orbitopathy (Table 4.4).

There was significantly lower (p =0.000) Mean free T3 in Female Hyperthyroidism subjects without Graves Orbitopathy compared to the Female Hyperthyroidism subjects with Graves Orbitopathy. There was significantly higher (p =0.000) Mean free T4 in Female Hyperthyroidism subjects without Graves Orbitopathy compared to the Female Hyperthyroidism subjects with Graves Orbitopathy. There was significantly lower (p =0.000) Mean free TSH of Female Hyperthyroidism subjects without Graves Orbitopathy compared to the Female Hyperthyroidism subjects with Graves Orbitopathy (Table 4.4)

Discussion

This study documented dramatically increased concentrations of free triiodothyronine (T3) and thyroxine (T4), concomitantly with markedly decreased levels of thyroid-stimulating hormone (TSH). The defining characteristic of hyperthyroidism is the endocrine dysregulation of the hypothalamic-pituitary-thyroid (HPT) axis, as seen by the findings of this study, which align with primary hyperthyroidism. This indicates increased synthesis of thyroid hormones resulting from thyroid-stimulating immunoglobulins (TSI) binding to the TSH receptor (TSHR) to follicular cells, so mimicking TSH action and circumventing regulatory feedback [18]. High levels of thyroid hormones in the blood have a deleterious effect on the anterior pituitary gland by lowering TSH levels.

Patients with Graves' disease exhibited markedly elevated T3 levels compared to their non-Graves' disease counterparts, indicating increased peripheral deiodination mediated by Type 1 deiodinase (DIO1), notably in extra-thyroidal locations such as the liver and inflammatory tissues. This aligns with findings from certain research indicating that cytokines such as IL-6 and IFN-γ can enhance deiodinase activity [19]. This localised T3 surplus may stimulate ocular fibroblast proliferation and adipogenesis, which are pivotal to the pathophysiology of Graves' ophthalmopathy (GO). On the other hand, the higher T4 level in the non-GO group could mean that peripheral conversion is less effective or that the autoimmune trigger is less strong. While several studies indicate T3 dominance in severe Graves' ophthalmopathy [20], others find no consistent pattern, implying the presence of biochemical phenotypes within Graves' illness [21]. These hormonal fluctuations highlight a dynamic interplay between systemic thyroid function and target tissue response. The hormonal analysis demonstrated that both male and female hyperthyroid patients, regardless of Graves' orbitopathy (GO) status, exhibited significantly elevated serum free triiodothyronine (T3) and thyroxine (T4) concentrations, coupled with markedly suppressed thyroid-stimulating hormone (TSH) levels when compared to their sex-matched euthyroid counterparts. These changes are the basic biochemical signs of thyrotoxicosis, which is caused by thyroid-stimulating immunoglobulins triggering TSH receptors in the thyroid gland [22]

The extent of these hormonal alterations varied according to sex and GO phenotype. In GO-positive patients, T3 levels were much greater in men than in women, whereas T4 levels were much lower in women than in men. These disparities may be due to sex-linked changes in peripheral deiodinase activity, hormone-binding protein concentrations, or the chronicity of the disease. It was posited that men with autoimmune thyroid disease typically exhibit later onset and more pronounced biochemical abnormalities, whereas women generally experience prolonged disease duration and elevated autoantibody titers [23].

The reduced TSH levels in all hyperthyroid groups result from negative feedback inhibition on the hypothalamic–pituitary–thyroid axis, which restricts pituitary thyrotropin release in the presence of elevated circulating thyroid hormones. Significantly, the heightened T3:T4 ratio reported in GO-positive males aligns with the "T3 toxicosis" pattern identified in severe Graves' illness, associated with augmented type I deiodinase activity and an elevated likelihood of extrathyroidal involvement [24, 25]

Conclusion

This investigation demonstrated that all hyperthyroid patients had the typical biochemical profile of raised T3 and T4 levels alongside suppressed TSH levels; however, the extent and characteristics of the accompanying inflammatory and immunological alterations varied according to sex and Graves' orbitopathy (GO) status.

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