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Clinical Study on The Effect of Thyroxine Hormone on Cardiac Patients After Therapy for Infection with The Corona Virus

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Abstract

The clinical investigation focused on the effect of thyroxine hormone on heart patients following coronavirus therapy, with 45 samples obtained from heart patients and 45 samples as a control group. The study found a substantial rise in thyroxine hormone, free thyroxine hormone, thyroid-stimulating hormone, Thyroglobulin AB, and magnesium levels (P = 0.001, P = 0.001, P = 0.001, P = 0.001, P = 0.001), respectively, in cardiac patients treated for coronavirus were compared to the control group. The study also discovered a strong link between thyroxine hormone and cardiac patients, as well as the impact of hormone levels following therapy. Accordingly, patients need medical follow-up for thyroxine levels to maintain their lives..

Keywords: Thyroxine Hormone, free Thyroxine Hormone, Cardiac Patients, and Corona Virus

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دراسة سريرية حول تأثير هرمون الثيروكسين على مرضى القلب بعد العلاج من الإصابة بفيروس كورونا

زينة عبد المنعم الجوادي [1]

المستخلص

وركزت الدراسة السريرية على تأثير هرمون الثايروكسين على مرضى القلب بعد علاج فيروس كورونا، حيث تم الحصول على 45 عينة من مرضى القلب و45 عينة كمجموعة تحكم. ووجدت الدراسة ارتفاعًا كبيرًا في مستويات هرمون الثايروكسين، وهرمون الثايروكسين الحر، والهرمون المحفز للغدة الدرقية، والثايروكلوبيولين AB، والمغنيسيوم عند مستوى احتمالية (P = 0.001، P = 0.001، P = 0.001، P = 0.001، P = 0.001)، على التوالي، في مرضى القلب الذين عولجوا من فيروس كورونا مقارنة بالمجموعة الضابطة. كما اكتشفت الدراسة وجود ارتباط قوي بين هرمون الثايروكسين ومرضى القلب، وكذلك تأثير مستويات الهرمون بعد العلاج. وعليه، يحتاج المرضى إلى متابعة طبية لمستويات الثايروكسين للحفاظ على حياتهم.

الكلمات المفتاحية: هورمون الثايروكسين، هورمون الثايروكسين الحر، مرضى القلب، فايروس كورونا

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Introduction

The cardiovascular system functions as a pump to continuously circulate blood through the body. The heart is the hub of the bloodstream, which includes a system of vessels for blood such as capillaries, veins, and arteries that convey blood to and from all parts of the body [1].

Thyroid gland hormones are unable to dissolve inwater, and greater than 99 percent of the levels of triiodothyronine (T3) and thyroxine (T4) in bloodstreams are linked to proteins that are carriers. Thyroxine is the major carrier of hormones from the thyroid, which is attached to globulin, a glycoprotein produced in the liver. There are also two significant carriers: transferrin and albumin. Carrier proteins maintain a constant pool of thyroid hormones, from which active and

unbound hormones released for target cell absorption [2, 3]. Thyroid hormones regulate the expression of genes in the heart and contribute to a variety of cardiac symptom, hyperthyroidism causes cardiac enlargement, this cardiac expansion is mostly caused by the additional labor required by the heart due to the higher hemodynamic stress [4, 5].

The thyroid gland directly influences the function of the cardiovascular system and blood arteries, hyper or hypo activity of it can create a number of issues [6].

Coronavirus disease 2019 (COVID-19) is an illness caused by a novel virus termed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; previously referred to as 2019-nCoV), which was discovered after an epidemic of respiratory sickness in Wuhan, in the Chinese province of Hubei [7], Since the start of the worldwide epidemic until the eleventh of November 2021, 251,266,207 documented cases of COVID-19 have been detected registered to the World Health Organization (WHO), with 5,070,244 fatalities, and the virus is still spreading [8]. SARS-CoV-2 can produce a range of clinical signs and symptoms, respiratory failure, and multiple organ failure by injuring the body directly and indirectly: The direct effect is caused by the virus's harmful effect on the target cell, whereas the indirect effect is caused by aberrant inflammatory immune responses involving cytokines, complement, and coagulation systems, thyroid hormones control both adaptive and innate immune responses via both genetic and nongenomic mechanisms [9]. as Figure (1).

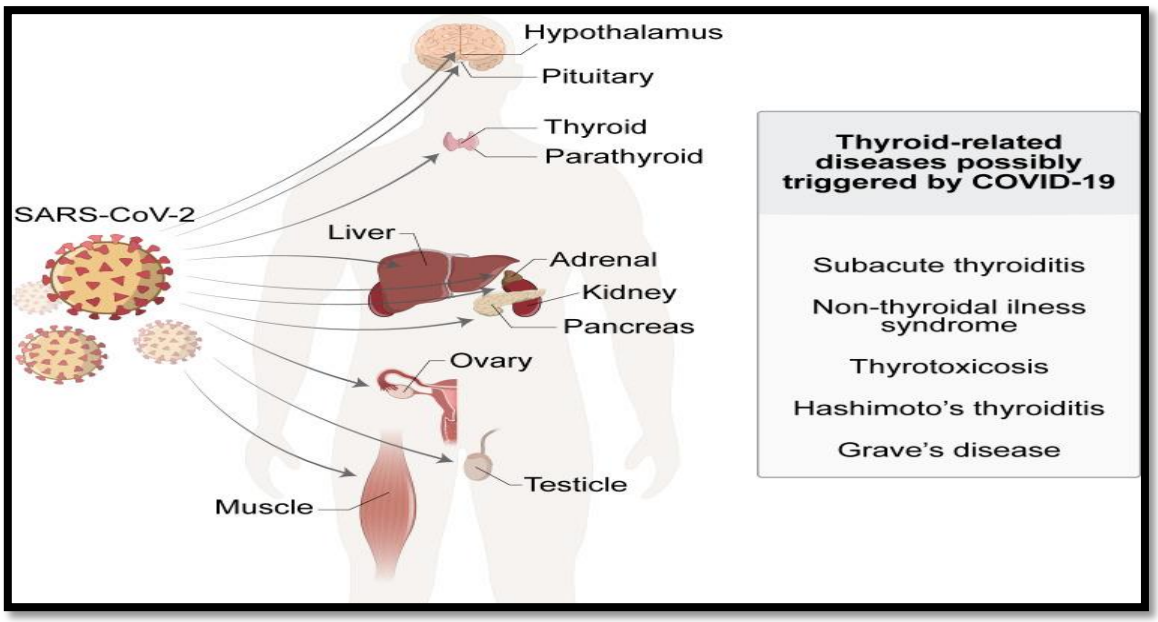

Figure (1): SARS-Cov-2 infection affects endocrine disorders, especially thyroid-related diseases

Thyroid hormones promote cytokine synthesis and release, resulting in a "cytokine storm," which is commonly associated with systemic infections caused by viruses [10].

Thus, more research is essential to better understand the causes and effects of thyroxin dysfunction caused by COVID-19 at both the biochemical and physiological levels after treatment.

Methods And Materials

Samples were collected from Ibn Sina Hospitals,

Iraq for the period from August 31, 2024 to June 1, 2025, according to the criteria of the Declaration of Helsinki.

Patients Group:

In this investigation, forty-five individuals of all patients in Ibn Sina Hospital. Their ages varied from 20 to 73 years.

Control Group:

consisted of (45) men and women aged 22 to 74 years.

Blood Collection:

The levels of thyroid hormones and other indicators were measured in both groups. Blood was taken in a test tube (5 ml), centrifuged to extract the serum, and kept in a deep freezer at -20°C for future analysis.

Materials and Procedures:

This study used a specific kit (Bio Merieux kits) for each of the Minividas-France hormonal variables: T4 (total thyroxine), fT4 (free thyroxine), TSH (thyroid stimulating hormone), Thyroglobulin AB (thyroglobulin antibodies), and Mg^{+2} (magnesium).

Statistical Analysis:

The data were examined with SPSS software. The T-test, Duncan test, and Pearson's correlation coefficient test were used to examine the variables among the total amount of control and patients based on occupation at $p \leq 0.05$, $p \leq 0.01$, and $p < 0.001$, respectively [11].

Ethical Approval:

The study was conducted under all applicable national legislation, institutional policy, and the Helsinki Declaration ideals, and was approved by the author's institutional review board No. 46692 at December 29, 2021.

Results and Discussion

Comparison of the level of clinical variables of Cardiac Patients After Therapy for Infection with The Corona Virus compared to the control group:

Table (1) indicates a decline in significance for T4 and fT4 at $p \leq 0.001$ in cardiac patients after therapy for infection with the corona virus compared to the control group. T4 levels in cardiac patients with COVID-19 may fall following therapy. This is determined by a number of factors, including the severity of COVID-19, the existence of other disorders such as thyroid disease, and any other medications that may alter thyroid hormone levels [12]. Overall, the findings suggested that thyroid dysfunction might emerge during the convalescent period of COVID-19. Although the cellular and molecular processes are not completely understood, research indicates that the "cytokine storm" plays an essential role in this environment. It is also worth mentioning that T4 can stimulate human platelets, resulting in pathological thrombosis, which is a consequence of viral infection.[13,14]

Also, the results in the same table shower significance low level of Mg+2 at $p \leq 0.001$ in cardiac patients after therapy for infection with the corona virus compared to the control group, Low levels of magnesium may harm cell membranes and promote inflammation, leading to high C-reactive protein,, cytokine release, NF-κB activation, and platelet dysfunction, which can contribute to thrombosis, although magnesium is vital for cardiovascular wellness and health, a suitable amount has yet to be determined [15,16].

Thus, magnesium shortage in the human body can raise the danger of an inflammatory "cytokine storm" as well as the possibility of endothelial damage and the coagulation cascade, which is followed by the phenomena of disseminated intravascular coagulation [17]. Rapid magnesium mobilization treatment can also affect the heart [18].

The results also demonstrated a substantial rise in the level of TSH and Thyroglobulin AB at a P≤0.001, this increase was largely attributable to elevated levels of “bad" cholesterol (LDL-C) in cardiac patient.[19]

Table (1): Level of clinical variables of Cardiac Patients After Therapy for Infection with The Corona Virus compared to the control group

Clinical Parameters	Control Group (Mean±SD)	Cardiac Patients After Therapy (Mean±SD)	P-value
T4 (nmol/ml)	105.02 ±13.7	80.88 ±21.9	≤0.001
fT4 (pmol/ml)	17.28 ±0.77	11.37±0.1	≤0.001
TSH (µIU/ml)	2.7 ±0.03	9.09 ±3.4	≤0.001
Thyroglobulin AB(IU/ml)	79.92±10.89	1301.6 ±18.8	≤0.001
Mg^{+2}(mg/dl)	2.00±0.07	0.95±0.5	≤0.001

The correlation between thyroxine hormone and other parameters effect on cardiac patients after therapy:

Table (2) indicates a substantial negative connection (P≤0.001) between T4 and TSH, as well as a significant positive correlation (P≤0.001) between T4 and TSH, Thyroglobulin Antibody, and Mg. The cause might be inflammation of the gland in the hormone receptor, which causes heart failure. When infected with Corona, the disease can persist even after therapy for a long time before returning to normal.

Table (2): The correlation between thyroxine hormone and other parameters effect on cardiac patients after therapy

Clinical Pparameters	Effect of T4 on Cardiac Patients After Therapy	
	R-value	P-value
fT4 (pmol/ml)	0.611	≤0.001
TSH (µIU/ml)	-0.522	≤0.001
Thyroglobulin AB(IU/ml)	0.512	≤0.001

Mg^{+2}(mg/dl)	0.423	≤0.001

The Proportion of Persons with and Without Thyroid Illness Who Have Cardiac Disease:

Figure (2) shows that 81% of heart patients in the study had thyroid disease at some point in the past before contracting the Corona virus and its treatment, but only 19% did not have it when they were infected with the virus. This is a modest number compared to patients with thyroid disease. Here, the relationship between thyroid dysfunction and heart disease becomes clear.

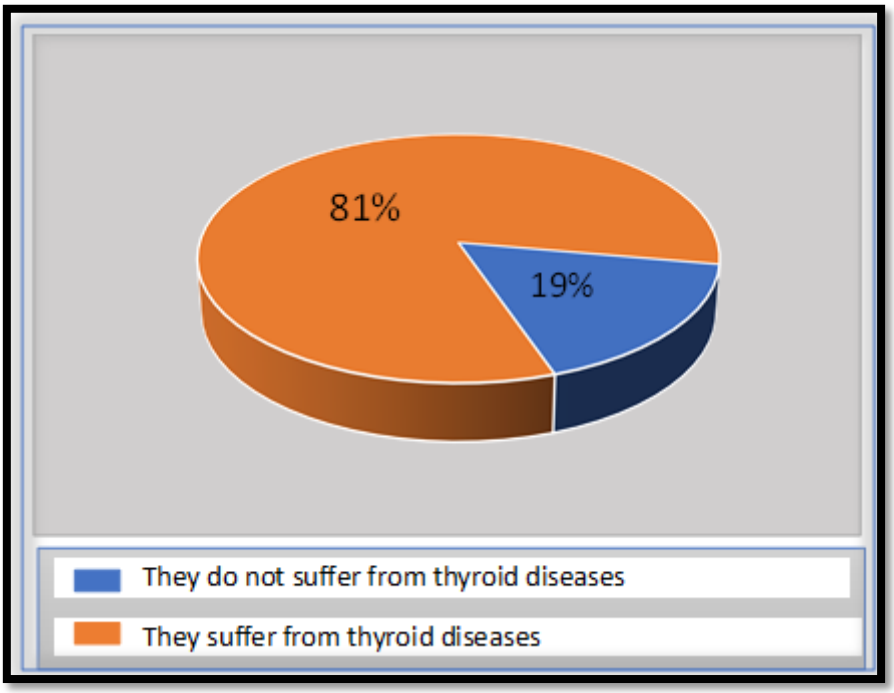

Figure (2): The proportion of persons with and without thyroid illness who have cardiac disease

Conclusions

According to the study, it is critical that physicians observe these alterations in thyroxine levels in heart patients infected with COVID-19 following treatment for the virus and take the appropriate steps to maintain stable thyroxine levels in heart patients in order to avoid complications that could result in death.

Recommendations

The prospect of administering a thyroxine test to heart patients promptly after infection with the Corona virus, or even only a suspicion of infection, to regulate the condition.

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