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ORIGINAL ARTICLE

# Impact of COVID-19 on Thyroid Hormones

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**Abstract**

*Background*: **Hypothyroidism is a significant endocrine condition. It addresses roughly 30−40% of patients found in endocrinology.**

*Aim*: **Intended to examine thyroid hormones in COVID-19-positive patent patients.**

*Methods and patients*: **This cross-sectional study included 200 Assiut patients admitted to isolation hospitals with a confirmed COVID-19 infection.**

*Results*: **Were compared and assessed for each patient: In intensive care, 182 patients improved, while 18 died. The study lasted anywhere from one to twelve months. The average TSH level was 1.28 0.714 IU/ml, while the average free T4 level was 1.36 0.624 ng/dl and the average free T3 level was 2.29 0.783 pg/ml, respectively. 68% of patients had hypothyroidism, while 32% of patients had normal thyroid function. 9% of patients survived, and 91% were cured. Heart rate and RR are not significantly different between the groups. Comorbidities studied showed statistically significant differences between the groups. Massive contrast between bunches for D. dimer and ferritin, ALT, AST, egg whites, TSH and free T3.**

*Conclusion*: **Thyroid hormones are affected by COVID-19. In terms of TSH and free T3, survivors and deceased individuals differ significantly. Decline in TSH and FT3 more in perished patients than in enduring patients.**

*Keywords:* COVID, FT3, FT4, Thyroid dysfunction, TSH

## 1. Introduction

In clinical practice, the most common endocrine problem is a ruptured thyroid gland; however, approximately a portion of the population with a ruptured thyroid gland is still unknown. The prevalence of thyroid dysfunction in the population has varied across a number of studies.[1] The terms ‘nonthyroidal disease syndrome’ (NTIS) and ‘euthyroid disease syndrome’ (ESS) are used to describe variations in thyroid dysfunction levels. Thyroid hormones during severe illness.[2] The pandemic of the global novel coronavirus (SARS-CoV2) began in 2019.[3] Lung involvement on CT is characterized by typical features such as the presence of dense lesions in multiple lung lobes, ground-glass opacities with consolidation shadows, or heart-shaped shadows and the presence of ground glass opacities with consolidation shadows. Patients are transferred to the intensive care unit.[3] A single preliminary study evaluating thyroid capacity in patients with cor Patients with more severe symptoms have lower levels of FT3 and TSH in their blood.[4]

Accordingly; this study aim to study thyroid disorder in patient with COVID-19 infection with previous history normal thyroid stat.

## 2. Patients and methods

This cross-sectional study of a COVID-19-infected patient admitted to isolation hospitals in Assiut aims to illustrate the patient's thyroid hormone levels. The study included 200 COVID-19-infected individuals, including 106 women and 94 men, from November 2021 to October 2022.

Adults over the age of 18 who have already been diagnosed with COVID-19 will be required to receive a nasopharyngeal SARS-CoV-2 vaccine as

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part of this study. Included was the CORADs score, a covid radiological assessment of disease severity; while waiting, people under the age of 18, pregnant women, people waiting with insufficient information, people waiting with a positive clinical history of hypo- or hyperthyroidism, thyroiditis, lithium treatment, or any disease are all prohibited.

For each patient, the following procedures were carried out: anamnesis, medication, and a comprehensive clinical examination are all included. Research Center Examinations: Complete Blood Count (CBC), Fasting Glucose and HbA1c, Erythrocyte Sedimentation (ESR), Plastic Agglutination Diatest, D-dimer Level, Serum Ferritin (TOSOH AIA-Mechanized Immunoassay Analyzer) 360 JAPAN), thyroid profile, LDH level, LDH level. Swabs from the oropharynx and nasopharynx were also taken. Obligation including photographs; Chest x-ray An admission was one that lasted for more than 24 h. COVID-19 pneumonia was diagnosed with consistent chest CT imaging and nasopharyngeal swab real-time PCR (RTPCR) detection of SARS-CoV 2.

For each patient, informed consent was obtained. The morals board of trustees of Al-Azhar Assiut College Emergency clinic endorsed the review.

SPSS 14.0 for Windows (SPSS Inc., Chicago, IL, USA) and MedCalc 13 for Windows (MedCalc Programming bvba, Ostend, Belgium) were utilized for information assortment, arrangement and factual examination. All statistical comparisons had a two-sided significance level; a significant room difference with a *P* value of 0.05; in a highly significant difference with a *P* value of 0.001 piece; and a difference that is not statistically significant has a *P* value greater than 0.05.

## 3. Results

This cross-sectional study was conducted on 200 patients with confirmed COVID-19 infection admitted in isolation hospitals in Assuit government. Patients were evaluated and compared according to outcome: 182 patients were improved and 18 patients died in ICU.

Table 1 shows that age of the study population ranged from 25 to 87 years with mean BMI was 27.55 kg/m$^2$ and (53%) of them were females. The 54% of the patients were diabetics, 44% of the patients were hypertensive and 31% of the patients were smokers and shows Laboratory parameters in the studied cases.

Table 2 shows Kidney and liver parameters among the studied patients.

*Table 1. Demographic distribution, comorbidities, laboratory parameters of the studied patients.*

| Variable | All patients ($n$ = 200) |
|---|---|
| Age (years) | |
| Mean ± SD | 47.76 ± 15.86 |
| Range | 25—87 |
| BMI (kg/m$^2$) | 27.55 ± 2.64 |
| Sex | |
| Female | 106 (53%) |
| Male | 94 (47%) |
| Comorbidities | |
| DM | 108 (54%) |
| HTN | 88 (44%) |
| CKD | 34 (17%) |
| COPD | 45 (22.5%) |
| Laboratory parameters | |
| Hb (g/dl) | 12.27 ± 2.5 |
| TLC ($\times 10^3$/l) | 6.59 ± 0.778 |
| Neutrophil ($\times 10^3$/l) | 69.88 ± 4.78 |
| Lymphocytes count ($\times 10^3$/μl) | 0.954 ± 0.304 |
| PLT ($\times 10^3$/μl) | 268.99 ± 86.47 |
| RBS (mg/dl) | 247.15 ± 24.1 |
| HbA1c (%) | 7.16 ± 1.3 |
| D. dimer (ng/ml) | 170.1 ± 20.51 |
| Ferritin (μg/l) | 445 ± 51.58 |
| CRP (mg/l) | 51.19 ± 5.38 |
| ESR (mm/h) | 66.53 ± 5.03 |
| LDH (mg/dl) | 240.13 ± 40.22 |

*Table 2. Kidney and liver parameters among the studied patients.*

| Variable | All patients ($n$ = 200) |
|---|---|
| Serum creatinine (mg/dl) | 0.992 ± 0.196 |
| Urea (mg/dl) | 30.42 ± 9.48 |
| ALT (U/l) | 40.31 ± 9.7 |
| AST (U/l) | 37.31 ± 7.45 |
| Bilirubin (mg/dl) | 0.995 ± 0.2004 |
| Albumin (g/dl) | 3.8 ± 0.199 |

Table 3 shows that mean TSH was 1.28 ± 0.714 μIU/ml, mean free T3 was 2.29 ± 0.783 pg/ml, and mean free T4 was 1.36 ± 0.624 ng/dl the 32% of the patients had normal thyroid functions and 68% had low thyroid functions.

Table 4 shows that mortality rate was 9% while 91% of the patients were recovered.

*Table 3. Thyroid function distribution and value distribution among the studied patients.*

| Variable | All patients ($n$ = 200) |
|---|---|
| Thyroid function value distribution | |
| TSH (μIU/ml) | 1.28 ± 0.714 |
| Free $T_4$ (ng/dl) | 1.36 ± 0.624 |
| Free $T_3$ (pg/ml) | 2.29 ± 0.783 |
| Thyroid function distribution | |
| | $N$ (%) |
| Normal | 64 (32%) |
| Low | 136 (68%) |

*Table 4. Outcome distribution among the studied patients.*

| Variable | All patients (*n* = 200) *N* (%) |
|---|---|
| Living | 182 (91%) |
| Died | 18 (9%) |

Table 5 demonstrates a significant variation regarding pulse and RR between the groups, shows a highly significant variation regarding studied comorbidities between the groups and shows a significant variation regarding D. dimer and ferritin between the groups.

Table 6 shows a significant variation regarding ALT, AST, and albumin between the groups.

Table 7 shows a significant variation regarding TSH and free T3 between the groups.

*Table 6. Kidney and liver parameters.*

| Variable | Recovered (*n* = 182) | Died (*n* = 18) | t | *P* |
|---|---|---|---|---|
| Serum creatinine (mg/dl) | 0.985 ± 0.200 | 1.02 ± 0.139 | 0.725 | 0.470 |
| Urea (mg/dl) | 35.46 ± 8.3 | 38.9 ± 10.16 | 1.64 | 0.102 |
| ALT (U/l) | 39.87 ± 9.72 | 46.4 ± 7.32 | 2.77 | 0.006 |
| AST (U/l) | 37.13 ± 7.34 | 39.9 ± 8.81 | 2.58 | 0.011 |
| Bilirubin (mg/dl) | 0.998 ± 0.205 | 0.965 ± 0.113 | 0.672 | 0.502 |
| Albumin (g/dl) | 3.81 ± 0.198 | 3.69 ± 0.190 | 2.46 | 0.015 |

*Table 7. Thyroid functions distribution of the studied patients according to outcome.*

| Variable | Recovered (*n* = 182) | Died (*n* = 18) | t | *P* |
|---|---|---|---|---|
| TSH (μIU/ml) | 1.52 ± 0.841 | 0.534 ± 0.577 | 4.85 | 0.001 |
| Free $T_4$ (ng/dl) | 1.23 ± 0.522 | 1.41 ± 0.579 | 1.38 | 0.169 |
| Free $T_3$ (pg/ml) | 2.35 ± 0.812 | 1.81 ± 0.701 | 2.72 | 0.007 |

## 4. Discussion

WHO pronounced the worldwide pandemic of Coronavirus in Spring 2020.[5] Thyroid chemicals are emphatically engaged with the guideline of natural and versatile resistant responses.[6] Moreover, dysregulation of thyroid chemicals is in many cases portrayed in fundamentally badly hospitalized patients, serious treatment.[7]

Zhang *et al.*[8] concentrated on thyroid capability in 71 patients tainted with Coronavirus. 64% of patients with thyroid dysfunction and 56% of patients with COVID-19 had lower thyroid stimulating hormone (TSH) levels than controls, according to this retrospective study. In a similar vein, Chen *et al.*,[9] a retrospective study of 50 COVID-19 patients

*Table 5. Demographic and clinical distribution, comorbidities and laboratory parameters of the studied patients.*

| Variable | Recovered (*n* = 182) | Died (*n* = 18) | t | *P* |
|---|---|---|---|---|
| **Demographic and clinical distribution** | | | | |
| Age (years) | 47.64 ± 16.03 | 49.13 ± 14.64 | 0.379 | 0.705 |
| BMI (kg/m$^2$) | 27.12 ± 2.84 | 26.29 ± 2.39 | 1.2 | 0.232 |
| Sex | | | | |
| Female | 98 (53.8%) | 8 (44.4%) | 0.581 | 0.446 |
| Male | 84 (46.2%) | 10 (55.6%) | | |
| Pulse (beat/min) | 86.1 ± 5.76 | 90.45 ± 2.87 | 3.16 | 0.002 |
| RR (cycle/min) | 21.75 ± 5.45 | 25.21 ± 3.05 | 4.21 | <0.001 |
| SBP (mmHg) | 116.74 ± 5.95 | 115.1 ± 5.35 | 1.12 | 0.262 |
| DBP (mmHg) | 75.22 ± 6.19 | 76.25 ± 5.18 | 0.682 | 0.496 |
| **Comorbidities** | | | **$\chi 2$** | ***P*** |
| Smoking | 47 (25.8%) | 15 (83.3%) | 25 | <0.001 |
| DM | 90 (49.5%) | 18 (100%) | 17 | <0.001 |
| HTN | 70 (38.5%) | 18 (100%) | 25 | <0.001 |
| CKD | 26 (14.3%) | 8 (44.4%) | 11 | 0.001 |
| COPD | 28 (15.4%) | 17 (94.4%) | 59 | <0.001 |
| **Laboratory parameters** | | | **t** | ***P*** |
| Hb (g/dl) | 12.31 ± 2.54 | 11.7 ± 1.67 | 0.997 | 0.320 |
| TLC (×103/l) | 6.6 ± 0.757 | 6.5 ± 1.1 | 0.511 | 0.610 |
| Lymphocytes count (×103/μl) | 0.959 ± 0.310 | 0.884 ± 0.186 | 1.01 | 0.315 |
| Neutrophil (×103/μl) | 69.94 ± 4.68 | 69.0 ± 6.18 | 0.788 | 0.432 |
| PLT (×103/l) | 266.31 ± 87.86 | 306.5 ± 53.79 | 1.9 | 0.058 |
| RBS (mg/dl) | 246.89 ± 23.94 | 250.1 ± 25.97 | 0.539 | 0.591 |
| D. dimer (ng/ml) | 169.33 ± 20.45 | 180.3 ± 19.52 | 2.18 | 0.030 |
| Ferritin (μg/l) | 443.18 ± 50.64 | 470.6 ± 60.51 | 2.15 | 0.033 |
| CRP (mg/l) | 51.15 ± 5.41 | 51.7 ± 5.19 | 0.413 | 0.680 |
| ESR (mm/h) | 66.64 ± 4.96 | 65.1 ± 6.06 | 1.23 | 0.220 |
| LDH (mm/h) | 239.46 ± 40.23 | 249.4 ± 40.92 | 0.999 | 0.319 |

revealed low TSH and total triiodothyronine (TT3) levels, as well as a positive correlation between serum TSH and TT3 levels and the severity of the infection.

In any event, when the thyroid organ is known to communicate angiotensin-changing over catalyst 2 (ACE2) receptors, the relationship among hypothyroidism and results in patients with Coronavirus is hazy. It is consequently essential to comprehend the connection among hypothyroidism and Coronavirus-19.[10]

The essential goal of this study was to explore thyroid chemicals in patients with Coronavirus. This cross-sectional study included 200 patients admitted to public isolation hospitals in Assuit with a confirmed COVID-19 infection. Based on the results, patients were evaluated and compared: In the intensive care unit, 182 patients improved and 18 died. The span of the review shifted from 6 to a year.

In terms of the population; the study population had an average BMI of 27.55 kg/m$^2$, ranged in age from 25 to 87, and 53% of them were women. Teima *et al.*[11] report that 860 patients infected with COVID-19 were evaluated in this study substantiated our findings. The typical age of the patients contemplated was 46.1 ± 11.8 years and gone from 17 to 81 years, 45.8% of the patients examined were male and 54.2% were female. In addition, the study by Wu *et al.*[12] looked into 80 cases of COVID-19 in the Yancheng and Wuxi cities. Among them, 41 patients (51.25%) were ladies, with a middle period of 46.1 years. 27 patients (33.75%) were between the ages of 25 and 49, 19 patients (23.75%) were between the ages of 50 and 64, 15 patients (18.75%) were between the ages of 18 and 24, 10 patients (12.50%) were under the age of 18, and 9 patients (11.25%) were over the age of 65. Two patients were between the ages of 6 and 8 and six patients were between the ages of 11 and 13 out of the 10 patients under the age of 18; the age limit was 14 years old.

The ongoing review showed that concerning comorbidities; 54% of patients had diabetes, 44% had high blood pressure, and 31% smoked. 18.60% of respondents to the study by Razu *et al.*,[13] had hypertension, followed by diabetes (13.95%), asthma (11.62%), and allergies (11.62%). Fever (6.97%), cardiovascular diseases (6.97%), and additional issues in 2.32% of cases. While in the concentrate by Brigante *et al.*[14] seven patients (22.6%) had comorbidities like hypertension (2 patients, 6.5%), dyslipidemia (2 patients, 6.5%), gastritis (2 patients, 6.5%), and gentle melancholy (1 patient, 3.1%).

In terms of thyroid function, the current study demonstrated that; Free T4 was 1.36 0.624 ng/dl, free T3 was 2.29 0.783 pg/ml, and mean TSH was 1.28 0.714 IU/ml. 32% of patients had typical thyroid capability and 68% had low thyroid capability. When Colonnelo *et al.*[15] reported that 103 patients—57 women and 46 men—were included in the study, their findings were in line with ours. The TSH, FT3, and FT4 mean values were 0.27–4.2, 2–4.4, and 0.93–1.97, respectively. In the meta-examination by Chen *et al.*[16] the pervasiveness of thyroid-related chemical anomalies was higher in patients with extreme Coronavirus, and serum FT3 and TSH levels were lower than in patients with serious Coronavirus. With non-serious Coronavirus. However, there was no significant difference in FT4 levels. In the study by Baldelli *et al.*[17] thyroid function was evaluated in two groups of patients: patients in the intensive care unit (group B, $n = 23$) from 31 to 80 years old who were hospitalized with COVID-19 pneumonia (group A, $n = 23$). As a control group, a third group (group C) of 20 people between the ages of 37 and 80 with normal thyroid function (five women and fifteen men) was included.

In the current study, 9% of patients survived, and 91% recovered. The study by Al Saleh *et al.*[18] which found a mortality rate of 9.04% for 962 patients, provided support for our findings. Dessie and Zewotir[19] on the other hand, demonstrated that the analysis's findings revealed a combined death rate of 17.62% among COVID-19 hospitalized patients.

The ongoing review showed that as far as segment and clinical appropriation of patients concentrated on in light of results; In terms of heart rate, there is a significant difference between the groups. There was no relationship with segment factors. The study by Mikami *et al.*[20] which found that 858 out of 6493 patients, or 13.2%, died in their entire cohort, backed up our findings. 52/2785 outpatients, or 1.9%, and 806/3708 admitted patients, or 21.7%. Cox relative danger relapse demonstrating showed an expanded gamble of in-medical clinic mortality related with age north of 50 years, systolic circulatory strain under 90 mmHg, respiratory rate over 24 every moment minute.

The ongoing review showed that there is an exceptionally huge distinction between the gatherings as to the comorbidities examined. Our findings were supported by a number of studies: people with the risk factors mentioned in Garg *et al.*[21] had higher mortality rates and hospitalizations. Lippi *et al.*[22] discovered that people with COPD had a five-fold higher incidence of the disease Severe COVID-19 than people without this underlying condition. In addition, they demonstrated in a subsequent study by Henry & Lippi[23] that patients with end-stage renal disease (ESRD) are three times more likely than other patients to contract severe COVID-19 infections.

In the review we have in our grasp, there is a tremendous contrast between the gatherings as for D. dimer and ferritin. Albumin, ALT, and AST levels are significantly different between the groups. In contrast, there was a significant correlation between the two patient groups in terms of C-reactive protein, white blood cells, lymphocytes, hemoglobin, elevated creatinine, elevated liver enzyme, lactate dehydrogenase, ferritin, D-dimer, troponin, prothrombin time, and the internationally normalized ratio in the study by Mazaherpour *et al.*[24]

In terms of TSH and free T3, our findings demonstrated that there is a significant difference between the groups. A total of 127 patients were included in the Lang *et al.*[25] study, of which 116 were survivors and 11 were non-survivors. Non-survivors had lower levels of serum thyroid stimulating hormone (TSH) and free triiodothyronine (FT3) than survivors did, and non-survivors had a higher rate of low FT3 status at admission than survivors did. The survivors Univariate Cox relapse examination showed that FT3 level and low FT3 status were adversely and emphatically connected with the gamble of in-medical clinic passing, separately. Likewise, a multivariate Cox relapse examination uncovered that low FT3 status was related with an expanded gamble of in-emergency clinic demise in the wake of adapting to jumbling factors. In addition, COVID-19 patients with low FT3 status had a lower survival rate according to Kaplan–Meier curves.

### *4.1. Conclusion*

This study concludes that thyroid hormones are affected by COVID-19. TSH and free T3 levels differ significantly between survivors and deaths. TSH and FT3 were lower in deceased patients than in living ones).

## Presentation at a meeting

No.


## Source(s) of support

Nil.


## Authors' contribution

BA and AKB conceived and supervised the study; HAY and AKB were responsible for data collection. WA analysed and interpreted the data. All authors provided comments on the manuscript at various stages of development. All authors read and approved the final manuscript.

## Ethics information

Informed written consent was obtained from the patients. The study was done after approval from the Ethics Committee of the Faculty of Medicine, (approval code: MS 12-5-2023).

## Conflicts of interest

Nil.


## Acknowledgements

Nil.


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