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Thyroid dysfunction in patients with confirmed COVID 19 and short-term outcomes

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Abstract
Background: Coronavirus disease (COVID-19) is a pandemic of the new millennium that has presented unprecedented public health challenges. Thyroid dysfunction caused by Covid-19 was transient, with nearly all cases regaining normal thyroid function within three months of infection. The aim of this work was to assess thyroid function in cases suffering from confirmed COVID 19 to determine if this infection is associated with abnormalities in thyroid function.
Methods: This prospective research was conducted on 50 cases suffer from pneumonia caused by COVID 19 infection, all cases were adults, underwent clinical, laboratory assessment and chest computerized chest tomography, COVID 19 diagnosis was confirmed by reverse transcription-polymrase chain reaction.
Results: There was a significant increase in TSH, fT3 & fT4 after three months of follow up. TSH (µIU/mL), Free T4 (ng/dL) and Free T3 (pg/mL) were (3.04 ± 0.923, 2.14 ± 0.591, 2.6 ± 0.69 respectively) for 45 Followed up patients.
Conclusions: The current study showed that hypothyroidism seems to be associated with a worse prognosis. Our investigation demonstrated that abnormal thyroid functions were prevalent between cases with COVID-19, with a prevalence of up to 64%. The thyroid functions restored to normal levels after 2 months of COVID-recovery.

Keywords: Thyroid dysfunction, confirmed COVID 19, short-term outcomes, severe acute respiratory syndrome

Introduction
A new millennium pandemic, coronavirus disease 2019 (COVID-19) presents an unprecedented threat to global health [1]. This new βcoronavirus with an envelope has been called SARS-CoV-2, and it is the one responsible for the severe acute respiratory syndrome [2].

761,700 people have lost their lives to COVID-19 so far, while the number of SARSCoV-2 infections has surpassed 21,294,845, as of August 16, 2020 [3]. A virus that is related to SARS-CoV-1 is responsible for the respiratory disease known as severe acute respiratory syndrome (SARS) [2, 4, 5]. Consistent with its predecessor, SARS-CoV-1, SARS-CoV-2 infects human tissues by penetrating cells through the ACE2 receptor [4, 5].

Coronavirus infections can range from mild, asymptomatic infections like the common cold to life-threatening lung complications [6]. Cases at high risk (e.g., elderly age, male gender, chronic hypertension and other cardiovascular comorbidities, diabetes) are more likely to experience multi-organ dysfunction due to SARS-CoV-2 infection, which can induce inflammation both in the lungs and throughout the body [7, 8]. Some of the most prevalent serious consequences of COVID-19 include respiratory failure, acute cardiac damage, sepsis, acute respiratory distress syndrome (ARDS), and heart failure [7]. There are two types of damage that have been associated with the wide range of symptoms and organ failure in COVID-19 and SARS: direct damage, which occurs when the virus infects the cells directly, and indirect injury, which occurs when the immune system reacts abnormally to the virus, most likely affecting the coagulation, cytokine, and complement systems [9, 10].

Clinical management recommendations from the World Health Organization do not include testing thyroid function for COVID-19 [11].

Similarly, Leow *et al.* [12] discovered that four SARS cases, accounting for 6.7% of the total,

were found to be biochemically hypothyroid three months after their recovery. Out of them, three had central hypothyroidism and one had primary hypothyroidism as a result of newly-onset chronic lymphocytic thyroiditis. Permanent T4 medication was necessary for the case of primary hypothyroidism, in contrast to the three cases whose central hypothyroidism spontaneously resolved after three to nine months [12].

This investigation aimed to assess thyroid function in cases with confirmed COVID 19 to determine if this infection is associated with abnormalities in thyroid function.

Patients and Methods

The 50 cases who participated in this prospective trial were all under the age of 18, with COVID 19 infection underwent through clinical and laboratory assessment and chest computerized chest tomography (CT), Covid 19 diagnosis was confirmed by reverse transcription-polymrase chain reaction (RT- PCR) using nasal and pharyngeal swap.

The study lasted from April to June 2022 after receiving approval from the Ethical Committee at Tanta University Hospitals in Tanta, Egypt. Prompt written consent was demanded from cases relatives.

Any case who met the following criteria could not be admitted to our hospital: pregnant, suspected or confirmed case of COVID-19, history of thyroid, pituitary, or hypothalamic disorders, current use of anti-thyroid drugs or thyroid hormone replacement, use of systemic steroids, amiodarone, or radioiodine within the previous six months, or prior administration of glucocorticoids.

All cases were exposed to complete history taking and clinical examination and laboratory investigations [Complete blood count, thyroid-stimulating hormone (TSH), totaltriiodothyronine (TT3), free triiodothyronine (FT3), serum total thyroxine (TT4), serum free thyroxine (FT4), C-reactive protein (CRP), lactate dehydrogenase (LDH), D-dimer, serum ferritin, kidney function tests (serum creatinine, blood urea and urine analysis), liver function tests (Serum aspartate and alanine aminotransferases (AST and ALT), serum albumin, serum bilirubin, serum gamma glutamyl transferase (GGT), prothrombin time and international normalized ratio (INR))] and radiological investigations (computerized CT). TSH, FT3, and FT4 were administered as follow-up tests after three months.

Examination of thyroid gland

Neck: examine for size, form, and scars from prior surgeries on both the front and side of the neck. Have the case swallow some water and watch for any movement of the glands. Examine the thyroid gland, check the cervical lymph nodes (submental, submandibular, pre-auricular, anterior cervical, supraclavicular, posterior cervical, post-auricular, and occipital), sense any deviation of the trachea, and palpate it. To check for possible ectopic glandular tissue, percussion down the sternum may reveal retrosternal dullness; while auscultating the gland, have the case hold their breath as you listen for bruit. Examine the face and head for reddening or a 'peaches and cream' look, check for thinning hair or alopecia on the scalp, and look for signs of eye and periorbital swelling, such as lateral eyebrow loss, pale conjunctiva, periorbital oedema, and proptosis (one or both sides). evaluate eye movement downwards, check for lid lag, evaluate convergence, and evaluate extraocular muscle activity. Hands: (check for thyroid acropachy or onycholysis under the nails and fingers, palmar erythema under the palms), slight tremor under the skin, temperature under the skin of both hands, palpation of the radial pulse.

Statistical analysis

For the statistical analysis, we utilized SPSS v26 (IBM Inc., Chicago, IL, USA). The normality of the data was checked using histograms and the Shapiro-Wilks test. To compare quantitative parametric data, which were shown as mean and standard deviation (SD), the paired T test (or repeated measures ANOVA) was utilized. When comparing quantitative non-parametric data, we utilized the Wilcoxon test. The data was presented as the median and interquartile range (IQR). Data presented as percentages and frequencies were utilized to compare qualitative variables using the Chi-square test. For statistical significance, a two-tailed P value below 0.05 was used.

Results

Demographic distribution, comorbidities, symptoms distribution and mortality rate distribution were enumerated in this table. Table 1

Table 1: Demographic distribution, comorbidities, symptoms distribution and mortality rate distribution among the studied cases

		N=50
Age (years)		48.75 ± 16.42
BMI (kg/m^2)		26.83 ± 2.71
Sex	Male	28 (56%)
	Female	22 (44%)
Comorbidities	Smoking	18 (36%)
	DM	27 (54%)
	HTN	21 (42%)
	CKD	16 (32%)
	COPD	23 (46%)
Symptoms distribution	Fever	41 (82%)
	Cough	33 (82%)
	Fatigue	26 (52%)
	Shortness of breath	18 (52%)
	Diarrhea	9 (18%)
	Nausea	5 (18%)
	Vomiting	4 (8%)
	Headache	6 (12%)
Mortality rate	Recovered	45 (90%)
	Died	5 (10%)

Data are presented as mean ± SD or frequency (%). BMI: Body mass index, DM: Diabetes mellitus, HTN: hypertension, CKD: Chronic kidney disease, COPD: Chronic obstructive pulmonary disease.

Laboratory parameters and distribution of USG thyroid in cases with COVID-19 were enumerated in this table. Table 2

Table 2: Laboratory parameters and distribution of USG thyroid in cases with COVID-19

	N=50
Hb (g/dL)	12.27 ± 2.5
TLC (x103/L)	6.59 ± 0.778
Neutrophil (x103/L)	69.88 ± 4.78

Lymphocytes count (x103/µL)	0.954 ± 0.304
PLT (x103/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
Kidney and liver parameters	
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
Thyroid function value distribution	
TSH (µIU/mL)	1.28 ± 0.714
Free T4 (ng/dL)	1.36 ± 0.624
Free T3 (pg/mL)	2.29 ± 0.783
Anti-TPO	10 (20%)
Thyroid function status distribution	
Normal thyroid functions	18 (36%)
Low thyroid functions	32 (64%)
US finding	
Increased thyroid volume	20 (40%)
Increased vascularity	15 (30%)
Increased echogenicity	18 (36%)
Nodules or cysts	7 (14%)

Data are presented as mean ± SD or frequency (%). Hb: hemoglobin, TLC: total leucocytic count, PLT: platelet count, RBS: Random blood sugar, HbA1c: glycated hemoglobin, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, LDH: Lactate dehydrogenase, ALT: alanine transaminase, AST: Aspartate aminotransferase, TSH: thyroid stimulating hormone, FT3: Free triiodothyronine, TT4: serum total thyroxine, US: Ultrasound.

The levels of TSH and fT4 increased significantly following three months of follow-up. Between the two sets of data, there was no statistically significant change in fT3. Table 3

Table 3: Thyroid function after 3 months of follow up among the studied cases

	Baseline (n=50)	**Follow up (n=45)**	**P**
TSH (µIU/mL)	1.28 ± 0.714	3.04 ± 0.923	<0.0001*
Free T_4 (ng/dL)	1.36 ± 0.624	2.14 ± 0.591	<0.001*
Free T_3 (pg/mL)	2.5 ± 0.91	2.6 ± 0.69	0.551

Data are presented as mean ± SD. * significant p value <0.05, TSH: thyroid stimulating hormone, FT3: Free triiodothyronine, TT4: serum total thyroxine.

Pulse, RR, smoking, DM, HTN, TSH, Free T3 (pg/mL) and anti-TPO +ve were a significant difference between both groups. Age, sex, BMI, SBP, DBP, CKD, COPD and free T4 didn't differ significantly among both categories. Table 4

Table 4: Demographic, clinical, comorbidities and thyroid functions distribution of the studied cases according to mortality

		Recovered (n=45)	**Died (n=5)**	**Test**	**P**
Age (years)		48.64 ± 16.03	50.13 ± 14.52	.199	0.843
Sex	Female	25 (55.6%)	3 (60%)	.036	0.849
	Male	20 (44.4%)	2 (40%)		
BMI (kg/m^2)		26.29 ± 2.39	27.12 ± 2.84	1.2	0.232
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	.682	0.496
Thyroid functions distribution					
TSH (µIU/mL)		3.04 ± 0.923	0.2± 0.01	3.477	0.001*
Free T4 (ng/dL)		2.14 ± 0.591	0.9± 0.05	1.38	0.169
Free T3 (pg/mL)		2.6 ± 0.69	1.6± 0.03	2.046	0.046*
Anti-TPO +Ve		15 (33.3%)	5 (100%)	8.33	0.004*
Comorbidities					
		Dysfunction (n=32)	**Dysfunction (n=32)**	**χ^2**	**P**
Smoking		13 (28.9%)	5 (100%)	9.88	0.002*
DM		22 (48.9%)	5 (100%)	4.73	0.030*

HTN	16 (35.6%)	5 (100%)	7.67	0.006*
CKD	13 (28.9%)	3 (60%)	2.01	0.157
COPD	19 (42.2%)	4 (80%)	2.59	0.108

Data are presented as mean ± SD or frequency (%). BMI: Body mass index, DM: Diabetes mellitus, HTN: hypertension, CKD: Chronic kidney disease, COPD: Chronic obstructive pulmonary disease, RR: Respiratory rate, SBP: Spontaneous bacterial peritonitis, DBP: diastolic blood pressure, TSH: Thyroid stimulating hormone, TPO-Ab: Anti–thyroid peroxidase antibody.

Laboratory parameters and comorbidities were insignificantly different between cases with dysfunction thyroid and normal thyroid cases. Table 5

Table 5: Laboratory parameters and comorbidities of the studied cases according to thyroid Dysfunction

	Dysfunction (n=32)	**Normal (n=18)**	**t**	**p**
Hb (g/dL)	11.55 ± 1.64	12.18 ± 1.43	1.45	0.154
TLC (x103/L)	8.12 ± 2.32	6.87 ± 3.02	1.64	0.107
PLT (x103/L)	273.44 ± 35.92	268.18 ± 37.87	.504	0.617
RBS (mg/dL)	126.14 ± 19.61	118.57 ± 16.84	1.38	0.175
D. dimer (ng/mL)	188.75 ± 25.71	179.22 ± 22.11	1.32	0.193
Ferritin (µg/L)	486.97 ±65.82	425.75 ± 47.31	1.54	0.130
CRP (mg/L)	57.23 ± 29.18	48.77 ± 34.41	.922	0.361
ESR (mm/h)	63.14 ± 19.26	59.37 ± 18.1	.679	0.501
LDH (mm/h)	241.63 ± 85.25	219.58 ± 93.66	.847	0.401
Kidney and liver parameters				
Serum creatinine (mg/dL)	0.854 ± 0.154	0.792 ± 0.163	1.34	0.187
Urea (mg/dL)	23.25 ± 5.27	21.32 ± 5.64	1.21	0.231
ALT (U/L)	31.65 ± 8.56	33.38 ± 10.6	.629	0.532
AST (U/L)	30.09 ± 10.01	28.36 ± 9.11	.601	0.550
Bilirubin (mg/dl)	0.963 ± 0.172	0.919 ± 0.142	.922	0.361
Albumin (g/dl)	3.78 ± 0.213	3.86 ± 0.233	1.23	0.224
Comorbidities				
	Dysfunction (n=32)	**Dysfunction (n=32)**	**χ^2**	**P**
Smoking	12 (37.5%)	6 (33.3%)	.087	0.768
DM	20 (62.5%)	7 (38.9%)	2.59	0.108
HTN	14 (43.8%)	7 (38.9%)	.112	0.738
CKD	12 (37.5%)	4 (22.2%)	1.24	0.267
COPD	16 (50%)	7 (38.9%)	.573	0.449

Data are presented as mean ± SD or frequency (%). Diabetes mellitus (DM), hypertension (HTN), chronic kidney disease (CKD), chronic obstructive pulmonary disease (COPD). Hb: hemoglobin, TLC: total leucocytic count, PLT; platelet count, RBS: Random blood sugar, CRP: erythrocyte sedimentation rate, ESR: erythrocyte sedimentation rate, LDH: Lactate dehydrogenase, ALT: alanine transaminase, AST: Aspartate aminotransferase.

Discussion

One of the most significant public health concerns of the new century is the 2019 coronavirus disease pandemic (COVID-19) [13]. Hormones and immunomodulatory signaling molecules have a complicated association with thyroid and viral infections [14]. In some instances, viruses and their associated inflammatory-immune responses have been observed to permanently impact thyroid function, which is particularly noteworthy [15].

The present investigation demonstrated a significant increase in TSH, fT4, and fT3 after three months of follow up.

In concordance with the current study Chen *et al*. [16] showed that hormonal anomalies diminished after COVID-19 recovery, suggesting that the virus may cause short-lived, acute effects on the HPT axis. These hormonal changes returned to normal following recovery from COVID-19, according to the investigation, even in the absence of thyroid replacement treatment.

Also, in agreement with the current study Yanachkova *et al*. [17] demonstrated that 61.1% of cases had thyroid dysfunction two months after contracting COVID-19. Of those, 78.3% had subclinical hypothyroidism, 13% had preclinical hyperthyroidism, and 8.7% had overt hypothyroidism. Subclinical hypothyroidism persisted in 28.3% of the subjects who were monitored. Furthermore, except for cases without hormone replacement treatment or those with mild hypothyroidism, the TSH level was significantly lower in all cases when compared to the second month after the first COVID-19 infection ($p < 0.001$). Regarding thyroid functions distribution of the studied cases according to outcome, it was revealed that there is a significant association between mortality with decreased TSH and free T3.

In agreement with the current study Patel *et al*. [18] found significant association between mortality with abnormal TSH and free T3.

Also, in concordance with the Present investigation Deng *et al*.[19] revealed that The levels of FT3 and TSH were lower in the non-survival category of cases compared to the survival category (3.24 ± 0.42 vs. 4.19 ± 0.08, $p < 0.05$ and 0.69 ± 0.19 vs. 2.32 ± 0.2, $p < 0.05$). Just like that, the FT3 levels of the severe category's cases were lower than those of the non-severe category (3.67 ± 0.14 vs. 4.33 ± 0.09, $p < 0.05$). Regarding FT4, however, there was no discernible variation.

As well, Zhang *et al*. [20] observed that COVID-19 cases with TD were more likely to acquire a severe form of the disease. Among cases admitted to the hospital, the death rate was significantly greater for those with TD (20% vs. 0%, $P = 0.002$). It was more common for cases with TD to have a

hospital stay of more than 28 days (80% vs. 56.52%, P = 0.048) compared to those without TD.

As stated similarly by Beltrão *et al*. [21] The area under the receiver operating characteristic (ROC) curve can be used to predict COVID-19 fatality by serum FT3, and ESS has been correlated with a 7.057.05 OR of fatality.

Moreover, Dutta *et al*. [22] determined that FT3 was thought to serve as a standalone measure of severity in COVID-19 cases. Furthermore, serum TSH levels were shown to be lower in the non-survivors compared to the survivors.

Chen*et al*., (144) an investigation of fifty cases found that a lower TSH may be connected to a more severe case of COVID-19.

The systematic review and meta-analysis by Bhattacharyya *et al*. [23] discovered that the COVID-19 mortality rate was 1.57 (9.91, 2.72) with a combined HR of TSH level. This virus is associated with thyroid dysfunction, since the data demonstrate that the incidence of abnormal TFT was 3.77 times higher in severe COVID-19 cases compared to mild to moderate cases.

In contrast to the Present investigation Pereira *et al*. [24] found that hypothyroidism cases tended to have reduced rates of in-hospital mortality and a decreased need for mechanical breathing. Thus, it appears that hypothyroidism is not connected to a worse prognosis. The disagreement may be due to the difference in sample size and severity. The present study reported that 14% of cases had nodules or cysts, 40% had increased thyroid volume and 36% had increased echogenicity. The increased thyroid volume and hypoechoic areas are thought to be as a result of thyroid gland inflammation; the increased vascularity was thought to be caused by increased blood flow to the thyroid gland while the increased echogenicity was thought to be caused by increased deposition of calcium in the thyroid gland. Nodules or cysts can also be seen in the thyroid gland of cases suffering from COVID-19, but they are not as common as the other findings.

In agreement with our finding Fung *et al*. [25] found that out of thirty-two cases (59.3%) had a noticeably larger thyroid, as measured by a rise of 15% or more, whereas just one case (1.9%) had a noticeably smaller thyroid, as measured by a decrease of 17%.

Similarly, our results in consistent with Lui *et al*. [26] who disclosed that 11 cases (13.9%) exhibited ultrasonographic alterations indicative of thyroiditis. Of these, five exhibited heterogeneous echogenicity, six exhibited abnormal vascularity (two increased and 4 decreased), and three exhibited micro-nodulation. Hypoechoic thyroid parenchyma was not observed in any of the cases. A thyroid inferno pattern was not observed in any of the cases who exhibited increased thyroid parenchymal vascularity. In total, fifteen cases were diagnosed with macro-nodules in the thyroid.

Conclusions

The current study showed that hypothyroidism seems to be associated with a worse prognosis. Our investigation demonstrated that abnormal thyroid functions were prevalent between cases with COVID-19, with a prevalence of up to 64%. The thyroid functions restored to normal levels after 2 months of COVID-recovery.

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Conflict of Interest: Nil

References

1. Team CC-R, Bialek S, Bowen V, Chow N, *et al*. Geographic differences in COVID-19 cases, deaths, and incidence-United States, February 12–April 7, 2020. MMWR Morb Mortal Wkly Rep. 2020;69:465-71.
2. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, *et al*. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020;382:1708-20.
3. Durmuş V. Is the country-level income an important factor to consider for COVID-19 control? An analysis of selected 100 countries. Int J Health Gov. 2021;26:100-13.
4. Hoffmann M, Kleine-Weber H, Schroeder S, Krüger N, Herrler T, Erichsen S, *et al*. SARS-CoV-2 cell entry depends on ACE2 and Tmprss2 and is blocked by a clinically proven protease inhibitor. Cell. 2020;181:271-80.e8.
5. Ziegler CGK, Allon SJ, Nyquist SK, Mbano IM, Miao VN, Tzouanas CN, *et al*. SARS-CoV-2 receptor ACE2 is an interferon-stimulated gene in human airway epithelial cells and is detected in specific cell subsets across tissues. Cell. 2020;181:1016-35.e19.
6. Yang X, Yu Y, Xu J, Shu H, Xia J, Liu H, *et al*. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med. 2020;8:475-81.
7. Chen T, Wu D, Chen H, Yan W, Yang D, Chen G, *et al*. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. BMJ. 2020;368.
8. Marazuela M, Giustina A, Puig-Domingo M. Endocrine and metabolic aspects of the COVID-19 pandemic. Rev Endocr Metab Disord. 2020;21:495-507.
9. Zhu J. SARS-CoV-2 induces pulmonary injury from basic to clinical research. World J Cardiovasc Dis. 2022;12:168-90.
10. Guo Y, Korteweg C, McNutt MA, Gu J. Pathogenetic mechanisms of severe acute respiratory syndrome. Virus Res. 2008;133:4-12.
11. World Health Organization. Clinical management of severe acute respiratory infection (SARI) when COVID-19 disease is suspected: interim guidance, 13 March 2020. World Health Organization; 2020.
12. Leow MK, Kwek DS, Ng AW, Ong KC, Kaw GJ, Lee LS. Hypocortisolism in survivors of severe acute respiratory syndrome (SARS). Clin Endocrinol (Oxf). 2005;63:197-202.
13. Saberi-Movahed F, Mohammadifard M, Mehrpooya A, Rezaei-Ravari M, Berahmand K, Rostami M, *et al*. Decoding clinical biomarker space of COVID-19: exploring matrix factorization-based feature selection methods. medRxiv. 2021;7:21259699.
14. De Vito P, Incerpi S, Pedersen JZ, Luly P, Davis FB, Davis PJ. Thyroid hormones as modulators of immune activities at the cellular level. Thyroid. 2011;21:879-90.
15. Tomer Y, Davies TF. Infection, thyroid disease, and autoimmunity. Endocr Rev. 1993;14:107-20.
16. Chen M, Zhou W, Xu W. Thyroid function analysis in 50 patients with COVID-19: a retrospective study. Thyroid. 2021;31:8-11.
17. Yanachkova V, Stankova T, Staynova R. Thyroid dysfunction as a long-term post-COVID-19

complication in mild-to-moderate COVID-19. Biotechnol Biotechnol Equip. 2023;37:194-202.
18. Patel D, Naik D, Kamalanathan S, Tamilarasu K, Sahoo J, Roy A, *et al*. Thyroid function abnormalities and outcomes in hospitalized patients with COVID-19 infection: a cross-sectional study. Horm Metab Res. 2023;55:169-75.
19. Deng J, Zhang S, Peng F, Zhang Q, Li Y, Zhong Y. The association between FT3 with the outcome and inflammation/coagulopathy/fibrinolysis of COVID-19. Front Endocrinol (Lausanne). 2022;13:877010.
20. Zhang Y, Lin F, Tu W, Zhang J, Choudhry AA, Ahmed O, *et al*. Thyroid dysfunction may be associated with poor outcomes in patients with COVID-19. Mol Cell Endocrinol. 2021;521:111097.
21. Beltrão FEL, Beltrão DCA, Carvalhal G, Beltrão FEL, Brito ADS, Capistrano K, *et al*. Thyroid hormone levels during hospital admission inform disease severity and mortality in COVID-19 patients. Thyroid. 2021;31:1639-49.
22. Dutta A, Jevalikar G, Sharma R, Farooqui KJ, Mahendru S, Dewan A, *et al*. Low FT3 is an independent marker of disease severity in patients hospitalized for COVID-19. Endocr Connect. 2021;10:1455-62.
23. Bhattacharyya A, Seth A, Srivastava N, Imeokparia M, Rai S. Coronavirus (COVID-19): a systematic review and meta-analysis to evaluate the significance of demographics and comorbidities. Res Sq. 2021;10:144684.
24. Pereira DN, Silveira LFG, Guimarães MMM, Polanczyk CA, Nunes AGS, Costa ASM, *et al*. Hypothyroidism does not lead to worse prognosis in COVID-19: findings from the Brazilian COVID-19 registry. Int J Infect Dis. 2022;116:319-27.
25. Fung MHM, Lui DTW, Chiu KWH, Lee SH, Lee CH, Chow WS, *et al*. A prospective follow-up of thyroid volume and thyroiditis features on ultrasonography among survivors of predominantly mild to moderate COVID-19. PeerJ. 2023;11.
26. Lui DTW, Fung MMH, Chiu KWH, Lee CH, Chow WS, Lee ACH, *et al*. Higher SARS-CoV-2 viral loads correlated with smaller thyroid volumes on ultrasound among male COVID-19 survivors. Endocrine. 2021;74:205-14.

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