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ORIGINAL ARTICLE

Thyroid Disorders that Impact Covid-19 A Multi-Center Study

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ABSTRACT

COVID-1 has been linked to thyroid problems in the past. Hyperthyroidism is a disease that affects a wide range of people, from those with no symptoms to those who are experiencing symptoms.

Objective: determination of this study is to look at mortality rates and clinic pathological aspects of various thyroid illnesses.

Method: A Multi-center study conducted in NTH and HMC Hospital Peshawar between 2017 and May 2018 was culled for the study. Cases without informed consent and with a past of (thyroid disease) or thyroid surgery excluded. Chi-square and Fischer's exact tests were employed to analyse thyroid lab findings for connection with clinical and pathological features. (SPSS version 22) be present the statistical analysis. In this experiment, a P-value of 0.05 was considered significant.

Results: Ninety sample size had greater FT3 levels, and none reported having lower levels. Seventy-seventy percent (58.2 percent) reported greater FT4 levels, whereas nine percent (61.2 percent) reported lower FT4 levels.

Anti-thyroperoxidase and anti-thyroglobulin antibodies were found in 6 (6.1 percent) and 6 (3.9 percent) participants, respectively. .Anti.TG (.P-value 0.002),(anti-TP) (p-value 0.016) and free FT4 antibodies were shown to be statistically significant (p-value 0.005).

Conclusion: Thyroid disorders were common in COVID-19 individuals. Time and spontaneous healing seem to be the hallmarks of thyroid dysfunction.

Keywords: FT4 and FT3 anti-thyroglobulin and anti-thyroperoxidase

INTRODUCTION

In Wuhan, China, a novel and alarming lung pathology was discovered in November 2019. An aggressive and infectious sickness that quickly spreads over the globe, the (WHO) proclaimed it a epidemic illness on March 11, 2020. More than 2,543,755 people have died worldwide since then in 114,428,211 cases. The lung is the most often affected system by COVID-19, and the most likely cause is thrombosis, which then leads to pulmonary infarction**1**. According on lung4 involvement, it has been classified as an asymptomatic carrier, mild, moderate, severe, and catastrophic sickness. As long as they don't show symptoms, they're considered asymptomatic carriers. Mild instances are those with just a few infiltrates in the bases of the (lungs) and an oxygen saturation level of more than 94%**2**. These are the situations when the lung ground and reticulonodular darks are affected, but involvement is less than 50%. Despite the need for high-flow O2 through a nasal cannula, some individuals' oxygen saturation drops below 94%. When the lung has been affected to a greater than 50% degree, the severity rises**4**. ARDS, sepsis, and cytokine storm syndrome are some labels for critically ill individuals. In addition to the lungs, other tissues such as the (kidneys, heart, brain), and thyroid gland have been affected in the previous year**5**.

Thyroid diseases aren't included in a comprehensive guideline, which is why it's dangerous. In addition, we don't know whether asymptomatic COVID-19 individuals are in danger. De novo thyroid dysfunction may occur after an infection**6**. Cases might develop subclinical or secondary

hyperthyroidism, leading to various thyroid disease symptoms. Although these individuals had a history of thyroid disease, they did not have a higher mortality rate**7**. Cases with additional comorbid conditions, such as diabetes, heart disease, hypertension, or renal disease, have a much greater death rate**8**. The purpose of this research was to determine the mortality risk associated with different anomalies of thyroid disease**9**.

MATERIAL AND METHODS

This Multicenter study investigation was conducted at Naseer teaching hospital and the hmc endocrinology unit as soon as the institutional ethical review board provided its approval. All cases , regardless of age or gender, admitted to the intensive care unit, general medical ward, or hospitalised isolation unit with COVID-19 between December 2020 and May 2021 were consecutively sampled. A prior history of thyroid disease or surgery necessitated the patient's informed consent before participation.

The process of identifying and recording a patient's requirements: In order to gather data, the patient's consent is necessary. Every person who had a COVID-PCR test also had a (TSH, FT3, and FFT4 run) Thyroid .antibodies were tested for in cases with aberrant TSH levels or low average TSH levels after getting consent from their carers. Lab results were distributed into three collections as shown in the table below:

Table 1: Effective date for the delivery of laboratory reports

Test	Values		
	norm al Below	Normal range	Increased levels
Thyroid- exciting hormone (TSH)	< 0.4 mIU/L	0.3.3.0mIU/L	> 3.5 mIU/L
Free T3 (FT3)	< 1.9 pg/ml	1.9-3.3 (mg/ml)	(> 4.1 pg/ml)
Allowed FT4 (FFT4)	< 0.76 ng/dl	0.78-1.79 ng/dl	> 1.72 ng/dl
Anti- Thyroglobuli n (Anti-TG)	-	< 39 IU/ml	> 39 IU/ml
Anti- Thyropeoxidas e (Anti-TP)	-	< 34 IU/ml	> 34 IU/ml

Statistical Analysis: Mean, standard deviation (SD), and percentage are used for continuous variables. The "Shapiro-Wilk test" checked for aberrant data. (The Chi-square (2) test" and "Fischer's exact test" were used to measure correlations among results, thyroid lab data), clinical and pathological features. SPSS 22 was used for statistical analysis. P-values less than 0.01 are statistically significant.

This discovery might assist treat COVID cases with thyroid dysfunction and minimise mortality.

RESULTS

We studied 90 instances. Anti-thyroglobulin and anti-thyroid peroxidase anti-bodies remained verified. Cases under 38 and beyond 39 were segregated for statistical analysis. 16 competitors were over 39. (82.3 percent). 52% of males had TSH levels 3.39–11.0, FT3 values 1.7–0.69, and FFT4 levels 3.93–3.32.

Clinical and pathological features, such thyroid hormone levels, and patient outcome We looked at the association between outcomes and clinical and pathological features such thyroid

levels. Anti-TG (p=0.001), anti-TP (p=0.018), and free FT4 antibodies were significant (p-value 0.005). 70 (48.5%) had to lower TSH levels, while 17 (11.8%) had to raise them. 105 (73.9%) cases had higher-than-normal FT3 levels, while none had lower levels. 61% (n=88) had higher FT4 levels, whereas 6.3% had lower levels. Anti-thyroperoxidase (9.3%) and anti-thyroglobulin (4.2%) antibodies were identified. Two of nine deceased cases had anti-thyroperoxidase antibodies, compared to three of six with anti-thyroglobulin antibodies (Table 2).

Age affects cases ' clinical, pathological, and thyroid levels. Age was also a determinant in clinical, pathological, and thyroid levels. There was no statistically significant connection between anti-thyroglobulin and anti-thyroperoxidase antibodies in people over 40. (Table 3).

We also compared gender to clinic-pathological features and thyroid ranks. Even though males are extra likely to have thyroid disease, we couldn't find a statistical link (Table 4).

Table 2: Connotation of the consequence of cases with Clinic pathological physiognomies and Thyroid ranks

Specific	n=90	Result		P value
		Discharge	Discharge	
Result				
Cleared	30	16	2	0.711
Expired	60	60	14	
Male	45	61	11	0.208
Female	45	63	5	
tsh levels				
Blow Normal (< 0.4 mIU/L)	69	57	11	
Normal (0.4-4.0 mIU/L)	56	53	2	0.102
Increased (> 4 mIU/L)	16	13	2	
ft3 levels				
Blow Normal (< 2.1 pg/ml)	105	90	12	
Normal (2.1-4.4 pg/ml)	38	33	4	1.00
Increased (> 4.4 pg/ml)	0	0	0	
fft4				
Blow Normal (< 0.89 ng/dl)	8	5	2	
Normal (0.89-1.76 ng/dl)	46	37	8	0.018
Increased (> 1.76 ng/dl)	87	80	5	
Anti-Thyroglobulin (AntiTG)				
Normal (< 40 IU/ml)	137	14	121	0.001
Raised (> 40 IU/ml)	5	3	2	
Anti-Thyroperoxidase (AntiTP)				
Normal (< 35 IU/ml)	134	118	15	0.005
Raised (> 35 IU/ml)	8	6	2	

Table 3: Correlations between case ages, clinicopathologic traits, and Thyroid hormone concentrations

Characteristic	n=90	Age		p-value
		<= 38 years	> 39 Years	
Outcome				
Discharged	30	9	53	0.716[b]
Expired	60	1	7	
GENDER				
Male	45	5	32	1.00[b]
Female	45	5	30	
TSH Ranks				
Blow Standard (< 0.4 mIU/L)	69	3	31	
Typical (0.4-4.0 mIU/L)	56	5	24	0.422[a]
Improved (> 4 mIU/L)	16	1	7	
FT3 Ranks				
Blow Standard (< 2.1 pg/ml)	105	6	46	
Typical (2.1-4.4 pg/ml)	38	5	15	0.061[b]
Improved (> 4.4 pg/ml)	0	0	0	
FFT4				
Blow Typical (< 0.89 ng/dl)	8	1	3	
Standard (0.89-1.76 ng/dl)	46	3	21	0.218[a]
Improved (> 1.76 ng/dl)	87	6	36	
Anti-Thyroglobulin (AntiTG)				
Normal (< 40 IU/ml)	137	10	59	0.512[a]
Elevated (> 40 IU/ml)	5	0	3	
Anti-Thyroperoxidase (AntiTP)				
Normal (< 35 IU/ml)	134	10	76	0.755[a]
Raised (> 35 IU/ml)	8	1	4	

The chi-square test examines the relationship between clinical and pathological factors and the outcomes of cases. Fischer's exact test for the correlation between clinical and pathological variables and the outcome of cases

The chi-square test examines the relationship between clinical and pathological factors and the outcomes of cases. Clinical and pathological characteristics are linked to the outcome of cases in Fischer's exact test.

DISCUSSION

Coronaviruses affect the thyroid and other endocrine glands. SARS-CoV produced post-mortem damage to thyroid follicular and parafollicular cells10. SARS-CoV cases ' anterior pituitary glands revealed reduced thyrotropin (TSH) staining, indicating coronaviruses after death. SARS-CoV-2 penetrates cells via the thyroid-expressed ACE2 receptor11. Pakistan has 5.1% hyperthyroidism and 5.8% hypothyroidism. Even while hyperthyroidism is rare in Pakistan, both are more common than in Europe (0.7%) and the U.S. (0.5 percent) 12.

In India (3.4% vs. 1.6%), 3.4% had hypothyroidism13. Pakistan seems to have escaped COVID-19 untouched, unlike other countries. Our study sample verified that COVID-19 can take long-term belongings on cases , particularly the elderly when signs of increased FT3 in the circulatory system (such as build pains, misperception, and a raised temperament rate) may be dismissed as viral infection14.

This observational study assessed COVID-19's short-term thyroid effects15. COVID-19 accepted several euthyroid cases . Cases with COVID-19 exhibited reduced TSH and FT4 levels. Our investigation comprised regular thyroid function testing including FT4 and TSH values15. Increased proinflammatory cytokines like interleukin-6 induce TSH suppression. Cortisol reduces TSH production even at healthy levels. SARS-CoV-2 expresses ACE2, a pituitary virion binding receptor, which may kill thyrotrophs16.

Nonthyroidal sickness syndrome (NTI) or euthyroid sick syndrome cases have high thyroid hormone levels (SS). (22). NTI may not be primarily responsible for lower-than-normal TSH levels in 48.6% (70/144) of our COVID-19 cases 17. Min Chen initiate short TSH in 34% of COVID-19 cases in 2021. A previous study found substantial damage to SARS cases ' follicular epithelium and parafollicular cells. During SARS17's course and recovery, cases ' T3, T4, and TSH levels were lower than controls.

Prejudice has several causes. The study's small sample size and single researcher may be biassed. After the hospitalisation, we didn't follow up. At admission, we only included T3, T4, and TSH. Thyroid hormones were evaluated while most cases got glucocorticoids. Pituitary–endocrine feedback loops were difficult to rule out. Future studies should include more cases from different sites, detailed data, and dynamic thyroid function18.

CONCLUSION

Thyroid function issues were seen in a large number of COVID-19 participants in this study. The symptoms of hypothyroidism seem to fluctuate dramatically over time, yet typically return gradually and

naturally. While nonthyroidal sickness syndrome may have a role, the SARS CoV-2 virus may assault the thyroid gland directly. It is vital to mention that cases with thyroid problems should take special precautions to avoid viral contact. Doctors should closely monitor thyroid illness cases who are at high risk of acquiring COVID-19. Finally, future COVID-19 risk classification models will take thyroid diseases into account.

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