ORIGINAL ARTICLE

ASSOCIATION OF THYROID FUNCTION TEST WITH POST-COVID-19 PCR-NEGATIVE PATIENTS

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Background: Corona virus diseases-19 (COVID-19) infection has shown many complications in all organ systems, like thyroid gland, during the acute phase and in the post-COVID-19 period. The aim of this study was to find the risk of hypo-functioning thyroid gland during post COVID-19 period as a chronic complication for possible follow up and management implications. **Methods:** This combined retrospective-prospective study was conducted over a period of nine months at tertiary care hospitals, Ayub Medical Complex Abbottabad and King Abdullah Hospital, Mansehra. Through non-probability convenient sampling technique, data was collected from 160 records at the hospitals. Confirmed cases of COVID-19, after their consent, were measured for thyroid hormones at two, four and eight weeks. Data was analyzed using Chi-square test. *p*-value less than 0.05 was considered as significant. **Results:** Out of 160 study participants, majority 102 (63.75%) were males and 58 (36.25%) were females with mean age of study population of 47.41±14.29 years. The frequency of hypothyroidism decreased over eight weeks from 8 (38.09%) to 6 (28.57%). While subclinical hypothyroidism decreased from 14 (36.89%) to 12 (30.76%). There was a very highly significant (*p*= 0.0001) association between proportions of hypothyroidism, Euthyroid Sick Syndrome and Subclinical hypothyroidism, and proportions of euthyroid patients in post COVID-19 period over eight weeks. **Conclusion:** Subclinical hypothyroidism, overt hypothyroidism, Euthyroid Sick Syndrome, and thyrotoxicosis are manifestations of hypo functioning thyroid gland. There is a need to monitor patients over a period of as short as two weeks to two months and beyond for possible detection of these complications of COVID-19 for appropriate management.

Keywords: COVID-19; Hypothyroidism; Subclinical hypothyroidism; Thyroid functions; Thyroid hormone

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INTRODUCTION

In December 2019, an outbreak of beta coronavirus known as "Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2)" occurred and invaded the world within no time. Patients affected by COVID-19 are mostly asymptomatic having mild cough or flu-like symptoms. Around 14% of the infected people get severe disease out of which almost 5% develop critical illness needing Intensive Care Unit ICU.[1] There is no absolute treatment till now and only limited supportive and symptomatic therapy is available. Complication of COVID-19 comprises of a spectrum diseases and severe complicated cases lead to death due to acute respiratory distress syndrome (ARDS) and respiratory failure.[2,3]

SARS COV2 infection is usually associated with subclinical involvement of systems. Subclinical diseases are usually without symptoms or may have some minor symptoms. Subclinical thyroid involvement is a condition in which there is lymphocytic infiltration and thyroid stimulating hormone (TSH) concentrations are low or undetectable but free thyroxin (T4) and triiodothyronine (T3) concentrations are normal are usually normal or low it is usually caused by viral infection or a post viral inflammatory process. The common viruses involved are coxsackievirus, enterovirus, measles, mumps and adenovirus, however, the prevalence is increasing with the novel coronaviruses. Subacute thyroiditis is usually self-limiting disease and need no treatment but, in some cases, can lead to hypothyroidism.[8–10]

COVID-19 associated sub-acute thyroiditis is a complication of viral infection where patient presents with tachycardia without progression of COVID-19 illness[11]. Sub-acute thyroiditis is a rare type of thyroiditis that causes pain and discomfort in the thyroid. It is an acute inflammatory disease of the thyroid, often caused by a virus. Symptoms include fever, thyroid tenderness, and a triphasic clinical course of hyperthyroidism, hypothyroidism, and return to normal thyroid function complications. Clinical investigations usually show different thyroid function tests which may be decreased thyroid stimulating hormone (TSH) with elevated serum T3 and T4 level in the initial period followed by hypothyroidism [12,13] There is no evidence of thyroid function characteristics and the use of thyroid hormone for clinical outcomes prediction in patients with severe

COVID-19 yet; however, hypothyroidism has been linked to increased mortality in patients with chronic renal failure, sepsis and acute myocardial infarction.[14–16] The study aims to assess thyroid function tests in post-COVID-19 PCR-negative patients at 2 weeks, 4 weeks, and 8 weeks of recovery and to identify hypothyroidism and other forms of thyroid hormone abnormalities

MATERIAL AND METHODS

This combined retrospective-prospective study conducted over a period of nine months (From 26-02-2022 to 8-11-2022) at tertiary care hospitals, Ayub Medical Complex Abbottabad and King Abdullah Hospital, Mansehra. The study protocol was approved by Khyber Medical University-Advanced Study Research Board (ASRB- KMU-ASRB001621/FH/KGMC), Dated:24-02-2022).

Through non-probability convenient sampling technique, data was collected from 160 records at the hospitals. All patients who had recovered from COVID-19 and were PCR negative for at least 2 weeks, had recovered from mild to severe disease were enrolled in the study. Those with a positive medical history for hypo- or hyperthyroidism, thyroiditis or on treatment with lithium and/or amiodarone and pregnant women were excluded. Informed written consent was obtained from the patients before enrolling them in the study.

For blood tests, samples of venous blood (2ml) were collected from each patient under aseptic conditions and were transported to the lab for testing. Sampling was done on 2nd week, 4th week and 8th week post-COVID-19. The protein analyzer using thyroid functions test kit (Bio-compare USA) was used to measure thyroid hormones freeT3, freeT4 and TSH according to the manufacturer instructions. Data was recorded on a data collection *proforma.*

Statistical analysis was done using (SPSS) version 24.0. Descriptive variables like age and gender were calculated as mean, standard deviations, and frequencies. Frequency of post-COVID-19 patients having hypothyroidism at two, four and eight week was calculated. Patients were labelled hypothyroid based on reference values as T3 - 0.87 - 1.78 nmol/L, T4 - 6.09-12.23 ug/dL and TSH- 0.34 - 5.60 m IU/L. Proportions were compared by the Chi-Square test of independence/Fisher's Exact test, with Fisher correction where Chi-square assumptions were not met by the data. The *p*-value of ≤0.05 was considered statistically significant. Mean values of thyroid hormones T3, T4 and TSH will be compared between hypothyroid and normal post-COVID19 patients through unpaired t-test. *p*-value of less than 0.05 was considered as significant.

RESULTS

Out of 160 study participants, majority 102 (63.75%) were male. The mean age of study population was 47.41±14.29 years, with lightly higher mean age of males 47.64±15.25 than females 47.00±12.52 years, Table 1. The mean TSH level was higher at two weeks (5.62±14.58) which became progressively lower at four (4.30±10.05) and eight weeks (3.63±5.6 7) as shown in Table-2. The mean fT3 was lower at two weeks, which became slightly higher at four weeks followed by a slight fall again at eight weeks. A similar pattern was seen in fT4 levels as shown in table 3. The predominant abnormality at two, four and eight weeks was low free T3 (fT3) in 10(6.1%), low TSH in 19 (11.87%) and low fT3 10 (6.25%) respectively, Table 3. The collective interpretation of T3, T4 and TSH, case wise at week 2, 4 and 8 are show in figure 1,2, and 3. Among total, 29 cases have different interpretations of impaired thyroid hormones at two-, four- and eight-week, Table 4. The frequency of hypothyroidism decreased with passage of time over eight weeks from 8 (38.09%) to 6 (28.57%). The frequency of subclinical hypothyroidism showed a decreasing trend over period of eight weeks from 14 (36.89%) to 12 (30.76%) as shown in Table-5.

To compare the proportions of hypothyroidism, ESS and subclinical hypothyroidism at two, four and eight weeks, Chi-Square test of independence was used to find association between being found with hypo-functioning thyroid with passage of time after COVID-19, no association was found at. The X^2 result at (df 4, N = 61) = 1.05, $p = 0.09$. as shown in Table 6. To find the association between hypo-functioning thyroid with post COVID-19 status, those found with hypo-functioning thyroid and euthyroid, Chi-square test of independence was used. Strong association at X^2 result at (df2, N = 62) = 38.09, $p = 0.0001$ was found. Hence, the null hypothesis is rejected and the association of post COVID-19 infection with SAT and its triphasic interpretations is accepted at $p = 0.0001$, as shown in Table 7.

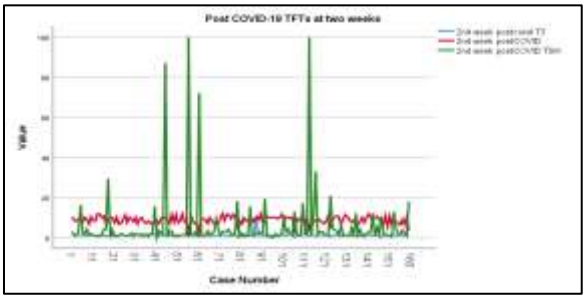

Figure-1: Free T3, free T4 and TSH at two weeks post COVID-19

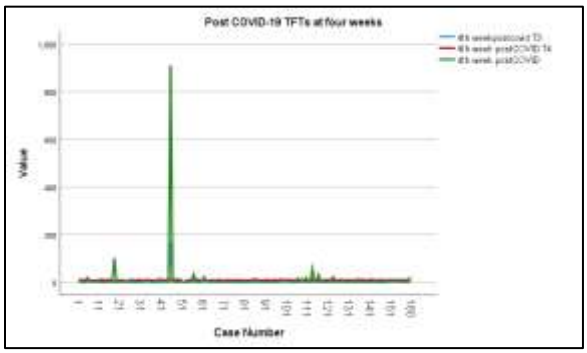

Figure-2: Free T3, free T4 and TSH at four weeks post COVID-19

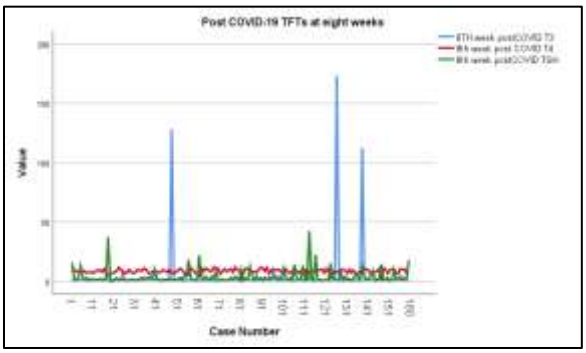

Figure-3: Free T3, free T4 and TSH at two weeks post COVID-19

Table-1*:* Mean, minimum, maximum and range of study participants. (n=160)

	Total participants	Males	Females
Frequency (%)	160 (100%)	102 (63.75%)	58 (36.25%)
Mean Age (years ± SD)	47.41 ± 14.29	47.64 ± 15.25	47.00 ± 12.52
Range	66	49	66
Minimum (years)	19	26	19
Maximum (Years)	85	75	85

Table-2: descriptive parameters at two, four and eight weeks (n=160)

Descriptive statistics of parameters at Two Weeks			
Parameter	fT3	fT4	TSH
Mean ± SD	1.171 ± 0.71	8.74 ± 1.91	5.62 ± 14.58
Median (IQR)	1.08 (0.96-1.29)	9 (7.41 – 10)	1.70(0.98 - 3.33)
Range (Minimum-maximum)	(0.04 - 9.40)	(0.92-12.02)	(0.001 – 100)
Descriptive statistics of parameters at **Four** Weeks			
Mean ± SD	1.32 ± 1.41	8.02 ± 2.25	4.30 ± 10.05
Median (IQR)	1.05(0.95-1.20)	8.22(7.27–9.42)	1.67(1.02 – 2.94)
Range (Minimum-maximum)	(0.20 – 10.5)	(0.51-12.21)	(0.36 –100)
Descriptive statistics of parameters at **Eight** Weeks			
Mean ± SD	1.11±0.52	8.41 ± 1.86	3.63 ± 5.6 7
Median (IQR)	1.01(0.94 -1.24)	8.42(7.25 -9.87)	1.80(1.14-3.30)
Range (Minimum-maximum)	(0.17-6.71)	(1.91-12.07)	(0.08-42.37)

Table-3: Frequency of Thyroid hormone levels at two weeks post COVID19

Thyroid Hormone	Week two		Week four		Week eight	
	Low	High	Low	High	Low	High
fT3	10 (6.1%)	1 (0.62%)	7 (4.37%)	1 (0.62%)	10 (6.25%)	0 (0.00%)
fT4	1 (0.62%)	0 (0.00%)	7 (4.37%)	0 (0.00%)	6 (3.73%)	0 (0.00%)
TSH	4 (2.5%)	21 (13.1%)	19 (11.87%)	21 (13.1%)	2 (1.25%)	20 (12.5%)

Low (<0.78nmol/L) High (> 1.78nmol/L)

Table-5: Frequencies of different interpretations of Thyroid functions over 2, 4 and 8 weeks

	Two weeks	Four weeks	Eight weeks
Hypothyroidism	8(38.09%)	7(3.33%)	6 (28.57%)
Males	7(41.17%))	5(29.41%%)	5(29.41%)
Females	1(25%)	2(50%)	1(25%)
Subclinical (39) Hypothyroidism	14(35.89%)	13(33.33%)	12(30.76%)
Males	11(35.48%)	11(35.48%)	9(23.97%)
Females	3(37.5%)	2(25%)	3(37.5%)
Euthyroid Sick Syndrome (ESS)	1(50%)	0	1(50%)
Males	0	0	0
Females	1(50%)	0	1(50%)
Sub Clinical (6) Hyperthyroidism	4(66.66%)	1(16.66%)	1(16.66%)
Males	2 (100%)	0	0
Females	2 (50%)	1 (25%)	1(25%)

Table-6: Chi-Square test of trend at 2, 4 and 8 weeks

	2 weeks	4 weeks	8 weeks	Total	X^2 df (2)	*p*
Hypothyroidism	8	7	6	21	1.05	< 0. 090
Subclinical Hypothyroidism	14	12	13	39		
ESS	1	0	1	2		
Total	23	19	20	62		

Table-7: Chi-Square test of independence of hypothyroidism, SC-hypothyroidism, and ESS

	Yes	No	Total	X^2 df (2)	*p*
Hypothyroidism	22	138	160	38.09	**< 0. 0001**
Subclinical Hypothyroidism	40	120	160		
ESS	2	158	160		
Total	64	416	480		

DISCUSSION

In the diagnostic or after treatment, thyroid hormone abnormalities and its different interpretations should be considered as a possible manifestation of COVID-19, particularly in hospitalized patients. The subclinical hypothyroidism and hypothyroidism prevalence is expected to be high in early days after COVID-19 with gradual recovery in some cases. This study focused on interpreting thyroid abnormalities as post COVID-19 manifestation at different weeks.

This study found subclinical hypothyroidism and hypothyroidism that might be possible sequalae of SAT (sub-acute thyroiditis). SAT causing thyrotoxicosis leading to subclinical hypothyroidism was reported predominantly in females between two to eight weeks, closer to this study. With 50% recovering at six weeks and 50% resulting in hypothyroidism.[17] Similar female predominance was reported by a systematic review with 20 (74.1%) female patients with SAT. The onset of SAT after COVID-19 infection ranged from 16–31 days, closer to the onset in this study.[18] An Indian study by Mondal S *et al* reported 11(6.8%) patients with post-COVID-19 SAT. Patients with painless thyroid gland had more severe thyrotoxic phase with higher levels of inflammatory markers as compared to painfull thyroid gland with SAT. At six months follow-up 9(82%) achieved euthyroid status with one subclinical and one overt hypothyroidism.[19]

At two weeks one case of ESS (Euthyroid sick syndrome) was reported and one case at eight weeks in this study. In contrast to this study an Indian study conducted in inpatients at a Delhi hospital reported 88/185 (53.7%) patients with ESS.[20] Persistent low T3 levels may affect prognosis in terms of clinical recovery, especially among critically sick hospitalized patients.[21] Similarly, higher mortality has been reported with low T3 levels, especially among elderly patients at 28-days of hospitalization (independent association, p= 0.001). The hospital expenses in the ICU were higher (p =0.001) for patients with ESS.[22] Cytokine storm during illness is considered a major determinant of ESS as these cytokines affect a variety of genes involved in regulating TH metabolism.[23] This difference can be partly attributed to more patients with mild disease in this study. Low fT3 levels suggest the need to monitor thyroid functions in patients with elevated inflammatory markers (IL-6) and severe COVID-19 infection.

The subclinical hypothyroidism was predominant presentation followed by hypothyroidism at two, four and eight weeks with slight fall in both conditions over period of eight weeks. The hypothyroidism was reported in 14 (8.53%) patients out of which nine had overt hypothyroidism and five had subclinical hypothyroidism.[20] This is closer to this study. A retrospective study reported 15 (5.2%) with hypothyroidism (13 subclinical hypothyroidism) with only 2 (0.69%) cases of overt hypothyroidism.[24] The systematic review by Trimboli P *et al*, reported subclinical hypothyroidism in 4 (14.8%) with only one patient with overt hypothyroidism at follow up.[18] The hypothyroidism in overt and subclinical form in COVID-19 patients may appear during triphasic course of SAT or stand-alone diagnosis without SAT. Majority of hypothyroid patients becomes euthyroid in viral and post viral illness.[18]

A study by Burekovic A *et al*, reported a significant difference in the number of patients with hypothyroidism and subclinical hypothyroidism after onset of COVID-19 pandemic when compared to pre-prevalence over one year. In contrast to the present study, the majority of patients were female with hypothyroidism and sub clinical hypothyroidism. The time of onset was comparatively later than present study, was average eight weeks after COVID-19 infection for subclinical hypothyroidism and hypothyroidism.[25] The low prevalence in the present study is partly explained by the early time of two weeks post COVID-19 for screening thyroid functions. The thyroxine dose required in these patients was not different from those without COVID-19 infection.

In this study 4(2.5%) patients had thyrotoxicosis at two weeks and 2 (1.25%) at four and

eight weeks. Three out of four recovered by four weeks and became euthyroid but one patient had thyrotoxicosis over the eight weeks period. All patients with thyrotoxicosis were female. Thyroiditis was reported in 9(5.5%) patients with high thyroxine and none of them reported neck pain.[20] Some of the studies reported low prevalence of thyrotoxicosis while others reported none. Paradoxically a retrospective study reported 58(20.2%) thyrotoxicosis, out of which 31 (10.80%) cases were of overt thyrotoxicosis and the rest subclinical thyrotoxicosis cases.[24] Subclinical thyrotoxicosis was reported in 12 (9.5%) of patients which was not significantly different from general population but still at the high end of general population range[26]. Subclinical hyperthyroid dysfunction was reported in 27 (36.98%) patients and subclinical hypothyroidism in 13 (17.8%) patients.[21]

A research study suggested higher incidence of thyrotoxicosis among COVID-19 patients than general population.[27] Though this finding was not supported but showed the thyrotoxicosis to be at higher end of normal as compared to general population[26]. The findings supporting the destructive thyroiditis as cause of thyrotoxicosis is supported by the mild impairment of functions and spontaneous recovery in most of the cases as in this study[24]. Another feature supporting destructive thyroiditis as cause of thyrotoxicosis is suggested by its association with high IL-6 levels that either initiate or trigger thyroiditis in cytokine storm.[28, 29]

CONCLUSION

COVID-19 infection adversely affects thyroid functions causing subclinical hypothyroidism, overt hypothyroidism, Euthyroid sick syndrome and thyrotoxicosis with hypo functioning thyroid as predominant pattern. Patients may need monitoring over a period as short as two weeks to two months and beyond for possible detection of these conditions for appropriate management and follow up.

Limitations

The thyroid hormones monitoring was not supplemented by laboratory data of inflammatory markers, management data and long term follow up. The measurement of inflammatory markers was limited by financial limitations.

Recommendations

The researcher recommends the longitudinal follow up over longer period to determine the outcome of different interpretations of thyroid functions and long term sequalae.

Conflict of interest

The researcher declared that the study was conducted in the absence of any financial or commercial interest that could be stated as a potential conflict of interest.

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