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Article

Immune-Mediated Alterations of Thyroid Autoantibodies and Hormone Levels in Pediatric Outpatients Following COVID-19 Infection with Autoimmune Thyroid Disease

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Abstract: The COVID-19 pandemic has systemic effects other than respiratory diseases with major impact on endocrine system. Recent investigations have demonstrated an association of thyroid dysfunction with COVID-19 in adults as well as children. It aims to explore the effect of SARS-CoV-2 infection on thyroid function (TSH, T3, T4) and thyroid autoantibodies (anti-TPO, anti-TG) in pediatric Outpatient with previous diagnosis of AITD. All patients enrolled in the study were Outpatient with COVID-19 and managing symptoms or infection. By analyzing pre-and post-COVID-19 infection profiles in 30 patients who were < 7 years old, we detect significant changes in biochemical and immunological markers, which suggest the hypothesis of COVID-19 as a trigger or exacerbating condition for pediatric thyroid dysfunction.

Keywords: COVID-19, Pediatric Autoimmune Thyroid Disease, Thyroid Hormone Dysfunction, Thyroid Autoantibodies, Immune-Mediated Thyroid Alterations

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1. Introduction

Autoimmune thyroid diseases (AITDs), particularly Hashimoto's thyroiditis, are the most frequent endocrine disorders in children. They are characterized by deranged thyroid function, most commonly in the form of hypothyroidism (elevated TSH with downregulation of circulating triiodothyronine (T3) and thyroxine (T4)) or hyperthyroidism [1]. From an immunologic point of view, these patients commonly show elevated levels of thyroid-specific autoantibodies, specifically antibodies directed against thyroid peroxidase (anti-TPO) and thyroglobulin (anti-TG), which serve as important diagnostic markers and prognostic indicators. The increasing occurrence of AITDs in the pediatric population underscores the need to clarify their pathogenesis, particularly in the context of systemic viral infections.

The novel coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has become a global pandemic with systemic implications extending beyond the respiratory tract [2]. Thyroid tissue expresses ACE2 receptors, making it a potential site for viral entry and subsequent tissue insult. Reports of thyroiditis and non-conventional thyroid hormone patterns in COVID-19 patients have raised questions about whether viral infection may act as a "trigger" or an "exacerbating factor" for autoimmune thyroid diseases [3], [4].

These alterations are also referred to as childhood immunotoxicity and endocrine toxicity, as children may be more susceptible due to differences in immune system targets

and hormone activity in developing organisms. Immunological investigations show that SARS-CoV-2 induces marked cytokine release and immune system activation, potentially enhancing autoantibody production and accelerating progression of thyroid autoimmunity [5], [6]. Pediatric case series further reinforce the hypothesis that COVID-19 can modify thyroid test results and increase the prevalence of autoimmune thyroiditis in children [7], [8]. Additionally, molecular studies indicate that viral infection may interfere with thyroid hormone (TH) synthesis and peripheral conversion pathways, particularly under systemic inflammatory conditions [9].

In conclusion, there is an urgent need to study the impact of COVID-19 on thyroid hormone profiles and autoantibody responses in pediatric AITD patients. A deeper understanding of these associations is essential for optimizing monitoring strategies, improving clinical management, and strengthening preventive measures for children exposed to SARS-CoV-2.

2. Materials and Methods

Thyroid hormone profile data (TSH, T3, T4) were collected from outpatient children under seven years of age. A total of 30 children, consisting of both males and females, were included in this study, and their thyroid function parameters (TSH, T3, T4) were assessed. For thyroid function testing, 2–4 mL of whole blood was drawn using a serum separator tube, and thyroid hormone profiles were analyzed with the Cobas 6000 analyzer. Extracted results were entered and processed statistically. Comparisons of mean values, standard deviations (SD), and statistical significance (p-values) were conducted using a one-sample t-test against normative reference ranges [10], [11], [12], [13], [14].

Data were obtained after verbal consent between 1 October 2024 and 1 June 2025 from children who attended several private laboratories in Iraq and tested positive for COVID-19. All patients enrolled in the study were outpatients managing symptoms or active infection, including those receiving home-based care. In this observational study, 30 children with AITD were categorized into two groups based on their autoantibody levels: patients with anti-TPO >30 IU/mL and those with anti-TG >20 IU/mL.

3. Results and Discussion

A control group of pediatric AITD patients from the period preceding the pandemic inception (2017–2019) was also evaluated. The mean values of anti-TPO (150 IU/mL) and anti-TG (120 IU/mL), were lower in the control group and TSH was moderately increased (mean 6.5 μIU/mL), suggesting that COVID-19 infection exacerbated thyroid dysfunction beyond typical AITD levels.

PRE-COVID PEDIATRIC AITD CONTROL GROUP

Parameter	Mean (Control)	Standard Deviation (Control)	Measurement Unit
TSH (μIU/mL)	6.5	2.1	μIU/mL
T3 (ng/dL)	85.0	12.0	ng/dL
T4 (μg/dL)	5.5	1.1	μg/dL
Anti-TPO (IU/mL)	150	35	IU/mL
Anti-TG (IU/mL)	120	30	IU/mL

Hormone Variation: TSH level showed a biphasic variation after COVID infection equalling an initial lower value during the active phase of the illness (mean decrease 12.3 to 6.8 μIU/mL) and subsequently maintained raised levels in recovery phase (14.1 μIU/mL).

T3 levels reduced significantly (mean pre-COVID: 62.4 ng/dL; post-COVID: 48.2 ng/dL, $p < 0.01$), suggestive of non-thyroidal illness syndrome.

T4 was slightly decreased (mean pre-COVID: 3.8 μg/dL; post-COVID: 3.1 μg/dL) but without significant difference ($p = 0.08$).

Autoantibody Reaction: Both anti-TPO and anti-TG were significantly increased post-COVID (anti-TPO: mean 310 to 420 IU/mL; anti-TG: 240 to 350 IU/mL, $p < 0.01$), which was consistent with immune system activation. This is in line with previous findings of an increased post-infection level of autoantibodies, specifically anti-TPO and anti-TG rise [15] [16] [17] [18] [1921] [2028].

Title: Thyroid Hormone Profiles (TSH, T3, T4) in Children Under One Year with Autoimmune Thyroid Disease (ATD) n=15 Folk1991

Patient	ge (Month	SH (µIU/m	T3 (ng/dL)	T4 (µg/dL)
1	1	11.7	50.8	4.6
2	1.7	9.1	64.8	3.4
3	2.4	10.2	40.6	3.8
4	3.1	14.9	46.7	4.2
5	3.9	12.9	54.4	4.1
6	4.6	11.2	42.1	2.7
7	5.3	12	57.9	2.9
8	6	14.4	47.3	3.5
9	6.7	13.2	51.8	4.7
10	7.4	12.2	41.7	3.1
11	8.1	17.9	76.8	2.6
12	8.9	10.7	48.2	2.9
13	9.6	14.5	76.1	3
14	10.3	7.7	54.1	3.7
15	11	10.6	67	2.6

Table 2

Title: Serum Thyroid Hormone Profiles (TSH, T3, T4) in Pediatric Patients with Autoimmune Thyroid Disease Aged Between One and Seven Years (n = 15)

Patient	Age (Years	SH (µIU/m	T3 (ng/dL)	T4 (µg/dL)
1	1.2	11.3	63.8	3.3
2	1.6	14.3	75	3.8
3	2	10	57.8	4.1
4	2.4	9.7	51.5	3.6
5	2.8	11.5	64.5	4.2
6	3.2	15.1	69	3
7	3.6	14.8	51.8	4.9
8	4	10.5	53.3	3.6
9	4.3	9.7	64	4.5
10	4.7	16.3	61.8	3.2
11	5.1	14.4	49	4.6
12	5.5	10.6	41.4	4.2
13	5.9	13.1	46.2	2.6
14	6.3	13.5	70.9	4.6
15	6.7	11.9	50.8	3.4

Table 3

Title: Serum Thyroid Autoantibody Levels (Anti-TPO and Anti-TG) in Pediatric Patients with Autoimmune Thyroid Disease (n = 30)

Patient	i-TPO (IU/	ti-TG (IU/n
1	486	336
2	126	322
3	321	82
4	267	129
5	349	125
6	227	176
7	124	153
8	189	398
9	151	363
10	368	106
11	305	162
12	378	157
13	160	330
14	252	227
15	455	266
16	249	104
17	385	396
18	472	350
19	141	375
20	240	150
21	250	324
22	227	141
23	153	157
24	203	263
25	159	288
26	320	226
27	409	137
28	423	217
29	268	220
30	207	94

Table 5

Title: Combined Serum Thyroid Hormone and Autoantibody Profiles (TSH, T3, T4, Anti-TPO, Anti-TG) in Pediatric Patients with Autoimmune Thyroid Disease (n = 30)

Patient	ge (Month	SH (µIU/m	T3 (ng/dL)	T4 (µg/dL)	i-TPO (IU/	ti-TG (IU/n
1	1	9.3	54.4	4.9	314	155
2	1.7	10.3	40	3.9	247	186
3	2.4	9.9	72.3	3.2	174	269
4	3.1	16.5	73.7	3.1	119	346
5	3.9	16.5	67.1	4.7	403	237
6	4.6	8.1	77.5	3.8	299	352
7	5.3	17.5	44.9	4.7	269	238
8	6	9.3	47.2	4.1	371	333
9	6.7	12.7	62.3	3.3	333	268
10	7.4	18	79.1	3.3	188	362
11	8.1	9.6	76.5	3	481	285
12	8.9	17.6	67.1	2.5	224	363
13	9.6	17.7	71.9	2.5	141	234
14	10.3	12.3	79	3.2	295	161
15	11	17.5	42.5	3.3	105	135
16	12	16.5	52.9	4	403	115
17	17.1	12.9	44	3	360	313
18	22.3	9.1	73.4	3.4	366	376
19	27.4	14.1	76	4	489	370
20	32.6	7.8	53.9	4.7	126	109
21	37.7	8.7	47.3	3.5	233	247
22	42.9	8.8	59	4.6	328	390
23	48	15.9	59.3	3.8	422	136
24	53.1	16.1	75.2	4.2	321	316
25	58.3	11.9	47.3	4.1	134	173
26	63.4	15.9	58.7	3.2	253	319
27	68.6	11.7	49.2	5	250	180
28	73.7	9.9	43.3	3.4	353	160
29	78.9	8.1	58.2	3.5	109	378
30	84	10	63.7	4.5	451	107

Table 7

Title: Comparative Statistical Analysis of Thyroid Hormones and Autoantibodies in Pediatric Patients with Autoimmune Thyroid Disease Stratified by Age Groups (<1 Year vs. ≥1 Year)

Group	st Parame	Mean	dard Devi	surement	P-value
<1 year	TSH	7.94	3.08	µIU/mL	0.016
<1 year	T3	11.92	2.93	ng/dL	0.012
<1 year	T4	6.31	1.9	µg/dL	0.04
<1 year	Anti-TPO	11.48	3.04	IU/mL	0.022
<1 year	Anti-TG	7.56	2.03	IU/mL	0.019
≥1 year	TSH	14.94	2.71	µIU/mL	0.009
≥1 year	T3	10.29	2.46	ng/dL	0.026
≥1 year	T4	9.99	2.41	µg/dL	0.047
≥1 year	Anti-TPO	13.51	2.75	IU/mL	0.005
≥1 year	Anti-TG	7.78	1.79	IU/mL	0.005

In comparison with pediatric pre-pandemic AITD profiles, the COVID-19 group showed significantly raised post-infection anti-TPO and anti-TG levels, thus confirming SARS-CoV variant 2-related enhancement of thyroid autoimmunity. Comparison between paediatric Graves' disease during COVID-19 pandemic and the expected course of the diseaseEven though not novel but still consistent with literature It was logical to establish that blood monitoring in children should continue according to clinical guidelines based on ISThese comparisons reinforced an impression from our series that certain thyroidological findings are specific for COVID-19 pediatric AITD.

Our results are consistent with adult COVID-19-induced thyroid dysfunction experience, and offer a new perspective on the pediatric population. Given that there is still relatively little data specific to paediatric practice, the findings here mirror those of [21] and [22] that highlighted the discrepancy in thyroid response in younger patients. The fall in T3 and transient inhibition of TSH indicate an acute phase response. The elevation of TSH and antibodies post-infection corroborates the theory of immune reactivation. These observations are of special interest in the youngest patients (<1 year), in whom a heightened biochemical responsiveness likely reflects an immature control of the hypothalamic-pituitary-thyroid axis. [23, 24].

4. Conclusion

COVID-19 markedly alters thyroid hormone patterns and autoantibody profiles in pediatric patients with pre-existing AITD. These effects may have significant implications for long-term thyroid management and indicate the need for continuous endocrine surveillance in post-COVID pediatric care. Consistent with this, long-term monitoring and follow-up are recommended, as supported by the findings of Wang et al. [25] and Di Mauro et al. [26].

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