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PAEDIATRIC AUTOIMMUNE THYROIDITIS TRIGGERED BY PRIOR SARS-COV-2 EXPOSURE: A CASE WITH TRANSIENT HASHITOXICOSIS

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ABSTRACT

Introduction: Thyroid dysfunctions in the paediatric population can present with atypical and nonspecific systemic manifestations, making diagnosis particularly challenging. Viral infections, including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), have increasingly been implicated in the onset or exacerbation of autoimmune thyroid diseases both in children and adults. This report describes a case of autoimmune thyroiditis with a transient hyperthyroid phase (hashitoxicosis) following prior exposure to SARS-CoV-2, highlighting the possible immune-mediated mechanisms of viral-induced endocrine autoimmunity that may extend to paediatric populations.

Case report: A previously healthy 13-year-old boy presented with fever, asthenia, bicytopenia, and hepatic cytolysis. Extensive infectious workup, including tests for hepatitis viruses, Epstein–Barr virus, cytomegalovirus, *Leptospira spp.*, and *Coxiella burnetii*, was negative, except for rhinovirus detection by reverse transcription-polymerase chain reaction. Thyroid function testing revealed elevated thyroid-stimulating hormone (11.9 µIU/ml) and positive anti-thyroid antibodies (anti-thyroid peroxidase >1000 IU/ml, anti- thyroglobulin, 2108 IU/ml). Anti–SARS-CoV-2 neutralizing antibodies were markedly increased, consistent with previous infection. Thyroid ultrasound showed diffuse inhomogeneous echotexture with a pseudonodular pattern and increased vascularity, consistent with autoimmune thyroiditis. Corticosteroid therapy led to fever resolution and normalization of laboratory parameters, followed by a transient hyperthyroid phase (hashitoxicosis) and subsequent progression to subclinical hypothyroidism.

Conclusion: This case underscores the importance of thyroid function evaluation in paediatric or adult patients presenting with unexplained systemic inflammation or cytolysis following viral infections. The overlapping effects of SARS-CoV-2 and other respiratory viruses may unmask or accelerate autoimmune thyroid disorders.

KEYWORDS

Autoimmune thyroiditis, hashitoxicosis, SARS-CoV-2

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LEARNING POINTS

- Post-viral thyroid dysfunction, including autoimmune thyroiditis, may occur after severe acute respiratory syndrome coronavirus 2 infection even in children and should be considered in the differential diagnosis of prolonged febrile or inflammatory syndromes.
- Hashitoxicosis represents a transient destructive phase of autoimmune thyroiditis and can mimic subacute thyroiditis or other causes of thyrotoxicosis; ultrasound vascularity and antibody profile help differentiate them.
- Systemic inflammation with hepatic cytolysis or cytopenias may accompany autoimmune thyroid involvement, reflecting multi-organ immune activation rather than isolated endocrine disease.
- Thyroid function testing (thyroid-stimulating hormone, free thyroxine, antibodies) should be included in the evaluation of patients, children or adults, with unexplained post-viral symptoms or abnormal inflammatory profiles.

INTRODUCTION

Thyroid dysfunction in the paediatric population often presents with vague symptoms such as fatigue, fever, or mild asthenia, which are commonly attributed to infectious or inflammatory diseases. Viral infections have long been recognized as potential triggers of thyroid inflammation, acting through direct cytopathic effects or immune-mediated mechanisms. Classic viral associations include mumps, Coxsackie, adenovirus, Epstein–Barr virus, and more recently, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)[1].

SARS-CoV-2 can infect thyroid follicular cells through ACE2 and TMPRSS2 receptors, leading to local cytolysis and antigen exposure. The resulting cytokine storm, characterized by elevated interleukin-6, tumour necrosis factor-α, and interferon-γ, may amplify autoimmune cascades and disrupt self-tolerance. These mechanisms contribute to the increasing incidence of post-coronavirus disease (COVID) autoimmune thyroiditis and Graves' disease in both adults and children[2–4].

Hashitoxicosis, a transient hyperthyroid phase occurring in early Hashimoto's thyroiditis, results from destructive hormone release from inflamed thyroid tissue[5]. During and after viral infections, this phase can be misinterpreted as subacute thyroiditis, emphasizing the need for careful integration of clinical, biochemical, and imaging findings.

Growing evidence suggests that coronavirus disease 2019 (COVID-19) and other viral illnesses can induce thyroid dysfunction even in previously healthy individuals. Hence, thyroid assessment should be included in the differential diagnosis of patients presenting with unexplained fever, hepatic cytolysis, or systemic inflammation following viral infections[4–6].

CASE DESCRIPTION

We report the case of a previously healthy 13-year-old boy who presented with fever and asthenia five days prior to admission to the Clinical Hospital for Pneumology and Infectious Diseases in Brașov. Initial laboratory tests revealed leukopenia (2260/µl), neutropenia (1210/µl), thrombocytopenia (106000/µl), hepatic cytolysis (ALT 202 U/l, AST 235 U/l), and elevated C-reactive protein (40 mg/l). Acute viral hepatitis (A, B, C, E), Epstein-Barr virus, and cytomegalovirus infections were ruled out by negative IgM serology. Other respiratory viral pathogens, including influenza virus, respiratory syncytial virus, metapneumovirus, adenovirus, and parainfluenza virus, were excluded by reverse transcription-polymerase chain reaction (RT-PCR) testing of nasal secretions. Rhinovirus was the only pathogen identified by RT-PCR. Bacteriological cultures from blood, urine, and pharyngeal secretions were negative. The chest X-ray was normal, and abdominal ultrasound revealed splenomegaly without other abnormalities.

Leptospirosis and Q fever were also excluded by negative IgM and IgG serologies for *Leptospira spp.* and *Coxiella burnetii*. Despite this, the patient continued to have persistent fever. Thyroid function tests revealed a markedly elevated thyroid-stimulating hormone (TSH) level (11.9 µIU/ml normal value: 0.59-6.93) with a low-normal free thyroxine (FT4) concentration (12.3 pg/ml; normal value: 9.95-15.9), consistent with primary hypothyroidism. Thyroid autoimmunity was confirmed by significantly elevated anti-thyroid peroxidase antibodies (>1000 IU/ml; normal value: 0-10) and anti-thyroglobulin antibodies (2108 IU/ml; normal value: 0-95). Anti-SARS-CoV-2 neutralizing antibodies were significantly elevated, (742 BAU/ml; normal value:<33), and the patient had a documented history of COVID-19 infection without subsequent vaccination. Thyroid ultrasound demonstrated inhomogeneous echotexture with a pseudonodular pattern, supporting the diagnosis of autoimmune thyroiditis *(Fig. 1)*. These findings raise the possibility of post-COVID-19 autoimmune thyroiditis as part of a delayed immune response.

Treatment with intravenous corticosteroids led to fever resolution and normalization of blood counts, while hepatoprotective therapy improved liver function tests. After 10 days, follow-up testing showed a rapid decrease in TSH to 4.64 µIU/ml, accompanied by a sharp increase in FT4 to 17.7 pg/ml and a newly detectable free triiodothyronine (FT3) level of 4.48 pg/ml (normal value 2.51-4.92). These changes indicated a transition to a transient hyperthyroid state consistent with hashitoxicosis, caused by the destructive release of preformed thyroid hormones from inflamed thyroid tissue, a diagnosis confirmed by endocrinology



Figure 1. Thyroid ultrasonography: transverse thyroid ultrasound images showing a diffusely heterogeneous glandular architecture, with multiple hypoechoic areas and disrupted parenchymal echotexture, characteristic of chronic autoimmune (Hashimoto's) thyroiditis. No well-defined nodules or cystic formations are visible.

consultation. At 1 month, TSH continued to decrease to 3.42 µIU/ml, while both FT4 and FT3 showed a downward trend (13.8 and 4.67 pg/ml, respectively), suggesting remission of the thyrotoxic phase and gradual return to euthyroidism. Autoantibody levels remained elevated, although anti-Tg titres began to decline (1504 IU/ml), possibly indicating reduced autoimmune activity. At the 2-month follow-up, TSH rose again to 7.76 µIU/ml despite FT4 (14.6 pg/ml) and FT3 (3.64 pg/ml) remaining within normal limits. This increase in TSH suggested relapse or progression toward subclinical hypothyroidism. Anti- thyroid peroxidase (TPO) levels remained persistently elevated (>1000 IU/ml), whereas anti-thyroglobulin (Tg) antibody titres, after an initial decline to 1379 IU/ml, showed a subsequent increase to 2315 IU/ml. Ongoing monitoring by the endocrinology service is warranted. The evolution of thyroid function, reflected by changes in TSH, FT4, and FT3 levels over time, is illustrated in *Figure 2*.

DISCUSSION

SARS-CoV-2 interacts with thyroid follicular cells via ACE2 and TMPRSS2 receptors, enabling direct viral injury. The associated cytokine surge promotes antigen exposure and autoreactive T-cell activation, precipitating autoimmune thyroiditis[2,7]. Molecular mimicry between viral and thyroid proteins further drives antibody cross-reactivity[4]. In this patient, elevated anti-TPO and anti-Tg antibodies alongside anti-SARS-CoV-2 antibodies support a post-viral autoimmune mechanism. The concurrent rhinovirus infection may have acted as a "second hit," amplifying immune activation and accelerating thyroid injury, a concept aligned with the two-step model of viral autoimmunity[2,4,7].

The absence of neck pain, the presence of high antibody titres, and the ultrasound pattern (diffuse inhomogeneous echotexture with pseudonodular areas and increased vascularity) ruled out subacute thyroiditis. Subacute (de Quervain's) thyroiditis typically presents with pain, hypoechoic poorly defined areas, and reduced vascularity, while autoimmune thyroiditis (Hashimoto's) is painless with increased blood flow. The dynamic biochemical pattern, initial hypothyroidism followed by transient thyrotoxicosis, reflects destructive release of preformed hormones (hashitoxicosis) rather than Graves' disease, as thyroid receptor antibodies were negative and thyrotoxicosis resolved spontaneously[5].

Hepatic cytolysis and bicytopenia likely reflected systemic immune activation rather than specific hepatic or hematologic disease. Corticosteroid therapy normalized

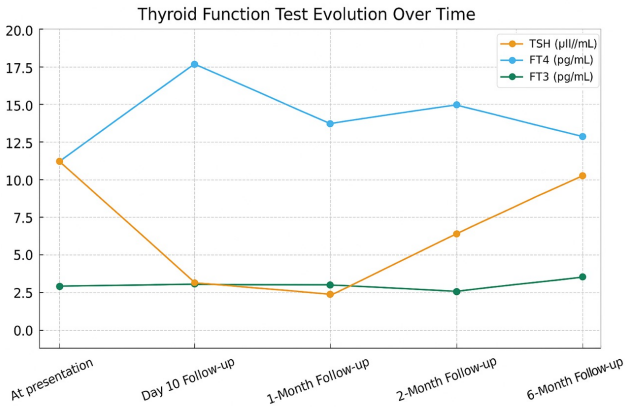


Figure 2. Dynamic evolution of thyroid function parameters during follow-up: serum thyroid-stimulating hormone (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) levels at presentation and during follow-up at 10 days, 1 month, 2 months, and 6 months. The initial elevation of TSH with low-normal FT4 is consistent with primary hypothyroidism, followed by a transient increase in FT4 and FT3 reflecting the hashitoxicosis phase, and subsequent normalization of thyroid hormones with a mild TSH rebound, indicating progression toward subclinical hypothyroidism. Reference ranges: TSH = 0.59–6.93 µIU/ml; FT4 = 9.95–15.9 pg/ml; FT3 = 2.51–4.92 pg/ml.

both liver and hematologic parameters, supporting an inflammatory mechanism. Similar multi-organ inflammatory patterns have been reported in paediatric post-COVID cases with transient thyroid dysfunction[4,6].

The triphasic evolution, from hypothyroidism to hashitoxicosis and subsequent subclinical hypothyroidism, mirrors autoimmune thyroid dynamics described in post-viral settings[5]. Persistent autoantibody elevation despite biochemical recovery suggests ongoing immune dysregulation with potential progression to permanent hypothyroidism[6]. Monitoring thyroid function every 4–8 weeks is recommended to identify transitions between functional phases and guide timely levothyroxine therapy if required.

A case of post-COVID autoimmune thyroiditis similar to ours was described by Knack et al., in an adult who developed Hashimoto's thyroiditis after SARS-CoV-2 infection and presented with overt hypothyroidism and elevated thyroid antibodies[8]. Compared with that report, our case highlights two key distinctions: the paediatric onset, rarely documented in the literature, and the presence of a transient hashitoxicosis phase, contrasting with the persistent hypothyroidism reported in adults. These differences suggest that post-viral thyroid autoimmunity may follow a more dynamic and potentially reversible course in children, whereas adults more frequently evolve toward stable hypothyroidism. This comparison broadens the phenotypic spectrum of COVID-related autoimmune thyroiditis.

Although paediatric, this case illustrates mechanisms relevant across age groups. Post-viral autoimmune thyroiditis, destructive thyroiditis within autoimmune disease, and multi-organ inflammatory manifestations are phenomena observed in both children and adults[9–11]. Clinicians should maintain a low threshold for thyroid testing in any patient—paediatric or adult—presenting with fever, cytopenias, or hepatic cytolysis after viral infections, particularly post-COVID. Early diagnosis prevents unnecessary antimicrobial therapy and ensures appropriate endocrine follow-up.

CONCLUSION

This case reinforces growing evidence that SARS-CoV-2 can trigger autoimmune thyroiditis through prolonged immune activation, potentially intensified by concurrent viral exposure. Recognition of the biphasic thyroid trajectory, from hypothyroidism to transient thyrotoxicosis and back, is key to avoiding misdiagnosis and overtreatment. Routine thyroid assessment and longitudinal monitoring are essential for timely detection and management of post-viral thyroid dysfunctions in both paediatric and adult patients.

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