

## Hashimoto encephalopathy induced by coronavirus

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### Abstract

**Background:** Multiple factors can induce thyroid disease. The mechanism is not precise; a possible mimicry effect in the thyroid tissue could be the cause. Encephalopathy could be one manifestation of this thyroid disorder, but its mechanism is not well understood. There is a lack of data showing coronavirus as a possible cause of Hashimoto encephalopathy.

**Case Report:** A 34-year-old obese Hispanic female was brought to our emergency department due to acute "Behavioral changes". Her family noted her anxiety with repetitive head movement.; the patient presented with psychotic behavior, reporting family members were killed (The same that brought her), having auditory hallucinations; the family could not precisely predict the duration of this behavior. Her history includes asthma (never intubated, not on steroids), morbid obesity, galactorrhea, and extensive HTN and DM. She denied chest pain, palpitations, nausea, vomit, fever, malaise, recent flu-like symptoms. No abnormality in the physical examination was noted except for repetitive head movement; due to her acute mental status changes and lactic acidosis, she was admitted under critical care service. During her admission, the TSH/T3/T4: 47.10/34/0.15, Antimicrosomal Ab and Coronavirus PCR were positive (Table1); CXR: No acute pathology, CT Head: Unremarkable, ultrasound neck showed Enlarged heterogeneous thyroid gland, which limits evaluation of underlying parenchyma; no measurable demonstrated solid, cystic or complex nodule (Figure 1). MRI head showed no pituitary adenoma. The patient was started on levothyroxine initially IV then transitioned to 200 mcg PO, and received ceftriaxone, dexamethasone, enoxaparin (As prophylaxis) for C19; after thyroid replacement was initiated; the patient started improving mental status drastically, she was transferred to the medical floor, later improved to baseline, and then was discharged. As an outpatient, the repeated thyroid panel showed normal values, and mental status was status as her baseline previous to her admission.

**Conclusion:** Our case report describes a case of Hashimoto encephalopathy induced by SARS-CoV2 virus. Besides reports relating this thyroiditis with viral other viral entities, there is no data about this thyroid disorder in the 2020 pandemic.

**Keywords:** Hashimoto, Thyroiditis, Encephalopathy, Coronavirus.

### Introduction

Multiple factors predispose to thyroiditis [1]. This includes genetically susceptible and environmental causing stressors, including systemic infections [2,3]. The acute inflammatory process could result in a thyroid disorder, especially viral illness, due to a possible mimicry effect in the thyroid tissue [4,5].

Despite viruses could be a cause of this disease, most individuals do not a manifest sign of the thyroid disorder, and those who do could report anterior neck tenderness, fatigue, constipation, diarrhea, or any of the classic thyroiditis associated sign or symptoms [6,7].

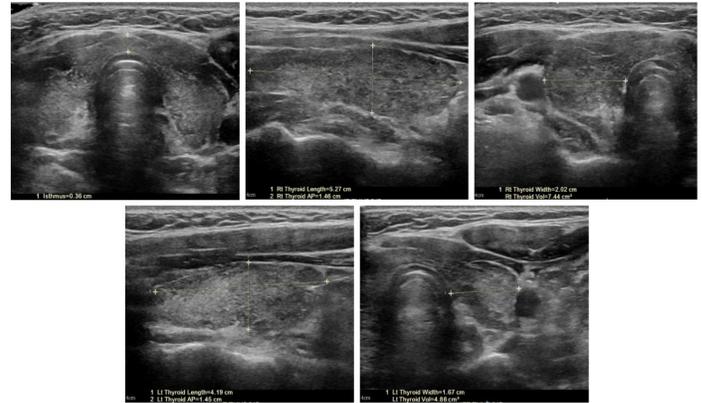
The literature has established the possibility of different viral

illnesses as a cause of thyroiditis, but there is a lack of data showing coronavirus as a possible cause of Hashimoto encephalopathy.

### Case

A 34-year-old obese Hispanic female was brought to our emergency department due to acute "Behavioral changes". Her family noted her anxiety with repetitive head movement, the patient presented with psychotic behavior, reporting family members were killed (The same that brought her), having auditory hallucinations; the family was not able to precise the duration of this behavior. Her history includes asthma (never intubated, not on steroids), morbid obesity, galactorrhea, and extensive HTN and DM. She denied chest pain, palpitations, nausea, vomit, fever, malaise, recent flu-

like symptoms. No abnormality in the physical examination was noted except for repetitive head movement, due to her acute mental status changes and lactic acidosis, she was admitted under critical care service. During her admission, the TSH/T3/T4: 47.10/34/0.15, Antimicrosomal Ab and Coronavirus PCR were positive (Table 1); CXR: No acute pathology, CT Head: Unremarkable, ultrasound neck showed Enlarged heterogeneous thyroid gland, which limits evaluation of underlying parenchyma; no measurable demonstrated solid, cystic or complex nodule (Figure 1). MRI head showed no pituitary adenoma. The patient was started on levothyroxine initially IV then transitioned to 200 mcg PO, and received ceftriaxone, dexamethasone, enoxaparin (As prophylaxis) for C19; after thyroid replacement was initiated; the patient started improving mental status drastically, she was transferred to the medical floor, later improved to baseline, and then was discharged. As an outpatient, the repeated thyroid panel showed normal values, and mental status was status as her baseline previous to her admission.



**Figure 1:** Thyroid Ultrasound.

**Table 1: Pertinent laboratory values.**

Laboratory	Value	Normal range
Thyroid Stimulating Hormone (TSH)	58.6	0.40-4.50 mIU/L
Triiodothyronine (T3)	34	60-181 ng/dL
Thyroxine (T4)	0.15	4.8-10.4 ug/dl
Microsomal Ab	296	<=35.0 IU/mL
Thyroid Peroxidase	>900	<9 IU/mL
Coronavirus RNA	Detected	Detected/Non-Detected
D-Dimer	<150	0-230 ng/mL
LDH	326	100-190 units/L
Ferritin	46	13-150 ng/mL
CPK	1154	20-200 units/L
Cerebrospinal Fluid		
Color	Colorless	
Appearance	Clear	
WBC	2	
RBC	33	
Total Cells	0	
Glucose	93	
LDH	12	
Protein	46	
VDRL	Not Detected	
HSV	Not Detected	
Lyme	Not Detected	

## Discussion

The exact mechanism of the thyroid affection in viral illness is not entirely understood. However, these agents could induce thyroid autoimmunity via multiple mechanisms, it could be affecting the self-antigens, mimicry self-molecules, inducing polyclonal T cell activation, forming immune complexes, and promoting the major histocompatibility complex molecules on thyroid epithelial cells [8].

Thyroiditis could be classified as silent or subclinical thyroiditis, transient hyperthyroidism, acute and subacute infectious thyroiditis, and chronic autoimmune thyroiditis (Graves disease and Hashimoto thyroiditis) [9]. Antithyroid antibody tests may be helpful to confirm the disease, for example, the thyroid peroxidase autoantibodies, thyroid-stimulating hormone receptor autoantibodies, and thyroglobulin autoantibodies [10].

The different manifestations, laboratory or histopathology findings observed varies between the different thyroid disorders; Hashimoto's thyroiditis is well known to be painless, the reason why it is also called silent thyroiditis or, in chronic cases, is also known as subacute lymphocytic thyroiditis [11]. Also, this thyroiditis is related to insulinitis and type-1 diabetes and as well as other inflammatory processes like colitis; as an autoimmune and inflammatory disease TLR3/4 overexpression could be present, favoring the environmental pathogens [12].

There are controversies at the time of the diagnosis for Hashimoto Encephalitis, Chong et al. concluded in a possible syndrome due to the clinical manifestations seen, but there is no evidence between the antithyroid antibody concentrations and its pathogenesis [13].

A cerebral syndrome with evidence of serum thyroid autoimmune disease is known as Hashimoto encephalopathy [14]. This entity could cause cerebral dysfunction, behavioral changes, elevated protein level in the cerebrospinal fluid, and electroencephalogram changes; all of these findings are reverted after levothyroxine is started [15], exposing clear evidence of the importance of a clinical assessment in time matter and how it can drastically change the clinical status and outcome of the patient [16].

The autoimmune markers are not necessarily specific, but if they are positive and the other possible diagnosis is being ruled out, it goes in pro of the suggested identifiable diagnosis by the test's specificity. The term "Hashimoto encephalopathy" has been an object of discussion in medicine. However, some authors have exposed the lack of proof to establish that this medical condition could be the leading cause of encephalopathy; there is literature exposing that there is not enough data to establish it, there is clear evidence of improvement after the thyroid disease is addressed.

### Conclusion

Our case report describes an exceptional case of Hashimoto encephalopathy induced by SARS-CoV2 virus. Besides reports relating this thyroiditis with viral other viral entities, there is no data about this disorder in the 2020 pandemic.

### Grant support

None

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