

COVID-19 and Hashimoto's Disease

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Dear Editor,

Hashimoto's thyroiditis is a common autoimmune disorder of the thyroid gland. Consequently, it has been linked to infections such as hepatitis C, EBV, HTLV-1, parvovirus B19, *Yersinia enterocolitica*, and coxsackie viruses. As COVID-19 has been associated with a few spectra of autoimmune diseases, we investigated the potential role of COVID-19 infection in inducing Hashimoto's thyroiditis¹.

A 36-year-old Turkish woman who worked as a laboratory assistant with no other chronic illnesses, and unknown COVID-19 exposure presented to the emergency department with a 2-day history of fatigue, loss in taste, and abdominal discomfort. She was afebrile and had a respiratory rate of 18 breaths per minute and oxygen saturation of 95% while she was breathing ambient air, the abdominal examination was normal. An oropharyngeal swab for COVID-19 testing was positive. After ten days, she reported complete resolution of her symptoms but complained of neck pain and constipation. Before the symptoms began, she was denied any personal or family history of autoimmune or thyroid disease.

On examination, the patient was afebrile and hemodynamically stable with no bradycardia or hypothermia. Cardiac, respiratory, abdominal, and neurological examinations were unremarkable, and the patient was clinically euthyroid with no goiter. Her thyroid function test (TFT) showed an elevated thyroid-stimulating hormone (TSH) reading of 0.012 mIU/mL (0.55–4.56), free T3 (fT3) level of 11.54 pg/mL (2–4.4) and a free T4 (fT4) level of 5.26 ng/L (0.78–1.76). Her TSH receptor antibodies were 2.71 IU/L (<1.75), her Anti-TPO level was 276 IU/mL (0.35), and her Anti Tiroglobulin level was 267.6 IU/ml (0–115). There was no anemia; her electrolyte levels, vitamin D, and B12 levels were normal. Her thyroid ultrasound showed her right thyroid

lobe as 16x20x33 mm, the left thyroid lobe was 16x21x29 mm, and Isthmus thickness was 7.5 mm. Both of her thyroid sizes are diffusely increased. Micronodular appearance and septal thickening were observed in the parenchyma. A hypoechoic nodular appearance of 9x4.5 mm was observed in the parathyroid region adjacent to the lower pole of the left lobe. One week later, when the patient's thyroid hormone levels were checked, her thyroid function test (TFT) showed a thyroid-stimulating hormone (TSH) level of 0.01 mIU/mL (0.55–4.56), free T3 (fT3) level of 3.13 pg/mL (2–4.4) and a free T4 (fT4) level of 1.12 ng/L (0.78–1.76). Our patient was diagnosed with Hashimoto thyroiditis due to clinical and laboratory findings and positive thyroid autoantibodies.

The patient was started on oral levothyroxine 25 mcg once a day and was counseled about her diagnosis of Hashimoto's thyroiditis. Four weeks later, the patient reported that she felt energized and had started running regularly. Her TFT was still deranged but had improved (fT3 4 pg/mL, fT4 1.7 ng/L; TSH 4.3 mIU/mL)

As the mounting evidence suggests a role of viral infection in the emergence of Hashimoto's thyroiditis, we have shown that there may be a positive correlation that COVID-19 infection is involved in the pathogenesis of some cases of Hashimoto's thyroiditis². Ultimately, although the present study may demonstrate an association between COVID-19 and Hashimoto's thyroiditis, more detailed researches with a more specialized examination and precise consideration of COVID-19 species, and investigation of environmental factors are needed³.

AUTHORS' CONTRIBUTIONS

VCA: Supervision. **UHB:** Writing – original draft. **GS:** Data curation, Formal Analysis, Supervision.

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