

COVID-19 complicated by Hashimoto's thyroiditisSingapore Med J 2021; 62(5): 265 <https://doi.org/10.11622/smedj.2020106>

Dear Sir,

While COVID-19 typically manifests with respiratory symptoms, mounting evidence suggests that it may precipitate a hyperinflammatory state that makes patients susceptible to autoimmune complications. Some known autoimmune complications of COVID-19 include antiphospholipid syndrome,⁽¹⁾ autoimmune thrombocytopenia,⁽²⁾ autoimmune haemolytic anaemia⁽³⁾ and Guillain-Barré syndrome.⁽⁴⁾ We herein describe a patient with COVID-19 who developed Hashimoto's thyroiditis.

A 45-year-old Chinese man who lived in a dormitory with a COVID-19 outbreak presented with non-productive cough and rhinorrhoea for one day. On the second day of his symptoms, he was diagnosed with COVID-19 through a SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) reverse transcriptase-polymerase chain reaction test from a nasopharyngeal swab and admitted to a COVID-19 isolation ward. Seven days after the onset of a mild COVID-19 upper respiratory tract infection, he reported complete resolution of his respiratory symptoms but complained of acute-onset severe generalised fatigue and muscle weakness. He had no neck pain, constipation or weight gain. Before the symptoms began, he had been in good health and working full-time, and had not been taking any supplements or chronic medications. He had never smoked and was teetotal. He also denied any personal or family history of autoimmune or thyroid disease.

On examination, the patient was afebrile and haemodynamically stable with no bradycardia or hypothermia. Cardiac, respiratory, abdominal and neurological examinations were unremarkable. The patient was clinically euthyroid with no goitre. Investigations were performed to determine the cause of his acute lethargy. His thyroid function test (TFT) showed an elevated thyroid-stimulating hormone (TSH) reading of 6.49 μ IU/mL and a low free thyroxine (fT4) level of 9.19 pmol/L, which is typical of primary hypothyroidism. His thyroid peroxidase antibody levels exceeded the upper limit of detection ($> 2,000$ IU/mL), which was indicative of Hashimoto's thyroiditis. There was no anaemia, and electrolyte levels were normal. Inflammatory markers and creatinine kinase levels were not elevated, and the chest radiograph showed no consolidation or effusion. The patient was started on oral levothyroxine 25 mcg once a day and was counselled about his diagnosis of Hashimoto's thyroiditis. Five weeks later, the patient reported that he felt energised and had started running regularly. His TFT was still deranged but had improved (fT4 10.91 pmol/L, TSH 6.59 μ IU/mL).

To the best of our knowledge, this is the first case of a patient who developed Hashimoto's thyroiditis after a COVID-19 infection. The time interval from the onset of his first respiratory symptoms to the inception of Hashimoto's thyroiditis was similar to that of reported cases of other autoimmune complications⁽¹⁻⁴⁾ and corresponds to the time frame of the cytokine storm.⁽⁵⁾ It indicates that the hyperinflammatory state triggered by COVID-19 may predispose patients to develop autoimmune complications. This case reminds us to be vigilant of autoimmune diseases as possible complications of COVID-19, even in patients with mild COVID-19 infections.

Yours sincerely,

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