CLINICAL VIGNETTE

Hashimoto Thyroiditis Following COVID-19 Infection

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Introduction

Coronavirus is a cause of respiratory infections worldwide for at least the last century. It emerged as major respiratory pathogen causing severe acute respiratory syndrome (SARS) during a 2002-2003 outbreak with thousands of infections and 10% mortality. In 2012 a new novel Coronavirus strain caused significant casualties during a breakout in the Middle East, which was known as the Middle East respiratory syndrome (MERS-COV). In December 2019 a novel strain of coronavirus was found to be a cause of several clusters of pneumonia cases in Wuhan, China, which quickly spread through to Hubei Providence in China and became the COVID-19 pandemic. By February 2021 more than 100 million cases were reported with 2 million deaths. Clinical presentation of COVID 19 varies ranging from asymptomatic infections to severe pneumonia associated with multiple organs failure requiring hospitalization and ICU admission. Throughout the past year COVID-19 infections affected multiple organs, with potentially every organ system affected. We present a case of young male who developed Hashimoto thyroiditis soon after recovering from COVID-19 infection.

Case Presentation

A 30-year-old male with no significant past medical history presented to the office with myalgias, cold intolerance, hoarseness of voice, constipation and swelling of both ankles, feet, and hands for 2 weeks. The patient was diagnosed with COVID-19 less than a month prior to symptom onset. His COVID infection was mild, and he was treated at home with OTC analgesics. He denied any hyperthyroid symptoms including palpitation, heat intolerance, weight loss, or diarrhea. He also denied odynophagia, dysphagia, thyroid tenderness or goiter. He had no family history of thyroid disease or autoimmune disorders. Physical exam showed periorbital edema and hoarseness of the voice. Skin was dry and cold, neck exam showed no goiter, or masses. Chest and abdomen exam was unremarkable. There was non-pitting edema on lower extremities. Neurological exam showed delayed relaxation phase with hypoactive deep tendon reflexes. Lab evaluation included TSH elevated to 100 mcUl/ml, free T4 0.01 ng/dl, antiperoxidase antibodies; 600 IU/ml, Creatinine 1.81 mg/dl and reduced GFR to 52 mgl/dl with normal electrolytes. Given the patient's presentation and labs results he was diagnosed with Hashimoto thyroiditis. He was started on levothyroxine 100 mcg daily and his symptoms improved over the following weeks. Repeat testing after 6 weeks showed TSH: 2.6 MUI/ml, T4 3.1 ng/dl, and Creatinine 1.1, and GFR >89. The patient stopped taking levothyroxine after seeing his results without physician discussion. Two weeks after stopping levothyroxine his symptoms reoccurred and repeat labs one month off levothyroxine showed TSH 123 mUI/ml, anti-peroxidase antibody 345 IU/ml, and T4 0.02 ng/dl. He was restarted on same dose of levothyroxine and remains symptom free with normal TSH of 2.6 mUI/ml and T4 of 1.4 ng/dl.

Discussion

Hypothyroidism is defined by elevation in TSH in response to a decrease in T4 level. Overt clinical hypothyroidism is 5-8 times more common in females with the majority linked to chronic autoimmune thyroiditis. Serum anti-peroxidase antibodies are usually elevated in autoimmune thyroiditis and the level of elevation a prognosis. This condition is known as Hashimoto thyroiditis and frequently involves other family members.[1] Our patient presented atypically as a young adult male without a family history following recovery from COVID-19 infection. Over the past year others reported thyroid diseases associated with COVID -19 infection. Two types of thyroid dysfunction appear related to COVID 19. Hypothyroidism due to non-thyroid illness syndrome with decreased T3 respond to illness and subsequent decreased T4 with persistent illness.[2,3] The second condition is thyrotoxicosis due to subacute thyroiditis. A few cases of euthyroid sick syndrome, have been reported severely ill hospitalized patients frequently in the ICU. COVID-19 can also cause inflammatory storm with increased Cytokines excretion. This may explain the trigger of Hashimoto thyroiditis following COVID-19 infection. Several others report COVID 19 causing subacute thyroiditis. Typical presentation includes thyrotoxicosis with elevation in FT4 and suppression and TSH along with pain, odynophagia and goiter. It is not clear if the virus itself infects the gland or if it is a response to Cytokine release during the viral infection.[3] Also, this patient had reduced kidney function when hypothyroid with improved GFR with levothyroxine treatment. Use of serum creatinine to evaluate renal function in hypothyroidism may be inaccurate as it generally returns to baseline with normal thyroid testing. This could be due to decrease in creatinine clearance and increased creatinine release during hypothyroidism.[4] Different Biomarkers may better assess renal function in patients with severe hypothyroidism. This case presents a possible correlation between autoimmune thyroiditis and

COVID-19. Further studies are needed to validate our observation.

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