Short Communication

COVID-19-associated the triggering of autoimmune thyroid diseases

Keywords: corona virus disease 2019, angiotensin converting enzyme 2, trauma, sepsis, starvation, adrenal glands

Abbreviations: COVID-19, corona virus disease 2019; ACE2, angiotensin converting enzyme 2; T3, triiodothyronine; TSH, thyroid-stimulating hormone; SARS-COV-2, severe acute respiratory syndrome coronavirus

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Description

Corona Virus Disease 2019 (COVID-19), a serious acute respiratory infection, is delivered on by SARS-CoV-2. As soon as the first case was reported in Wuhan, China, there was a rapid rise in cases that quickly spread to other parts of the globe. The disease can harm the functions of various organs, and its most common clinical symptoms are close to those of other viral diseases. The Angiotensin Converting Enzyme 2 (ACE2) receptors are located in many human tissues, including the pituitary, thyroid, testis, ovary, adrenal glands, and pancreas. SARS-CoV-2 enters the respiratory system and binds to these receptors. The thyroid gland may be affected by SARS CoV-2 either directly (through viral infection of target cells) or indirectly (by aberrant immunological regulation).[1]

Whether destructive or inflammatory, thyroiditis is a frequent illness that may be accompanied by a "cytokine storm," an influx in pro-inflammatory cytokines. In 287 hospitalized COVID-19 patients who were not in the intensive care unit, a retrospective study connected high IL-6 levels with thyrotoxicosis driven on by systemic immune activation. A form of thyroiditis that is closely related to viral infections is sub-acute thyroiditis. Numerous case reports after the COVID-19 epidemic have indicated that SARS-CoV-2 is most likely to blame. In addition, recent studies suggest that COVID-19 infection may contribute to the development of Graves' disease and Hashimoto's thyroiditis. We therefore intended to gather the available data and carry out a comprehensive study to evaluate the prevalence, clinical traits, and prognosis of autoimmune thyroid disorders triggered on by COVID-19.[2]

Twenty patients with autoimmune thyroid diseases connected to COVID-19 were identified during our literature search. The vast majority of the patients were ladies in their middle years. In general, thyroid issues had a benign course and responded favorably to medical treatment. According to the cases that have been reported, COVID-19 is thought to be the cause of Graves' disease and Hashimoto's thyroiditis, either as a new onset or as a flare-up of the condition when it was in remission.[3]

An autoimmune thyroid condition known as Graves' disease is delivered on in susceptible people by a variety of environmental causes, such as viruses. Molecular mimicry is one of the suggested paths, according to the theory. The immune system's ability to tolerate TPO, TG, and TSH-R collapses during the start of the illness. The potential role of a number of infectious agents in the etiology of Graves' illness has been investigated. Foamy viruses, Parvovirus B19, Epstein-Barr virus, and hepatitis C virus are examples of well-known etiological agents. Similar to how several viruses, including parvovirus B19 and hepatitis C, are thought to have a part in the aetiology of Graves' illness, Hashimoto's thyroiditis is not fully understood. As a result, COVID-19 might contribute to the aetiology of autoimmune thyroiditis in these people. Case studies do not prove that COVID-19 causes thyroiditis, despite the fact that they suggest a connection between both of them.[4]

Alterations in thyroid function, often known as "non-thyroidal illness syndrome" (or low T3 syndrome, or thyroid sick syndrome), may occur after a severe acute or chronic illness (trauma, sepsis, starvation, hepatic disorders, major systemic illness). Patients with COVID-19 who were seriously or critically ill had significantly lower free T3, TSH, and FT3/FT4 levels than those who weren't. COVID 19 exhibited over (10.8%) and subclinical (14.6%) thyrotoxicosis, according to another study that examined thyroid function in 287 patients in a non-intensive care unit.[5] Serum IL-6 levels were found to be significantly correlated with TSH levels as a result of the systemic immunological activation induced on by SARS-Cov-2 infection. Nine patients had negative TRab, anti-TG, and anti-TPO levels, and these patients spontaneously recovered during follow-up, suggesting that destructive thyroiditis was a likely cause. In 5 of the cases that were studied, pulmonary involvement was observed as a marker of disease severity. However, some people had severe concurrent thyroid dysfunctions, and two patients passed away as a result: one had Graves' disease and thyroid storm and died from adult respiratory distress syndrome; the other had hypothyroidism and passed away from myxedema coma and rapid cardiac death.[6]

Conclusion

Meanwhile subjected to COVID-19, vulnerable people may develop an autoimmune thyroid condition. In order to prevent missing a thyroid condition and postponing therapy, especially in those with

pre-existing autoimmune thyroid disorders, it is necessary to routinely evaluate thyroid functions both during the acute phase and throughout convalescence. Future investigation may shed light on the connection between thyroid autoimmune diseases and SARS-COV-2.

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Conflicts of interest

Author declare there are no conflicts of interest

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