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Short Communication

Inducement of Autoimmune Thyroid Illness due to COVID-19

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DESCRIPTION

SARS-CoV-2 causes Corona Virus Disease 2019 (COVID-19), a severe acute respiratory illness. Following the initial case reported in Wuhan, China, the number of cases swiftly escalated and spread around the world. The disease's most prevalent clinical signs are comparable to those of other viral infections, and it can damage the activities of multiple organs. SARS-CoV-2 penetrates the respiratory system and binds to Angiotensin Converting Enzyme 2 (ACE2) receptors found in numerous human tissues, including the pancreas, thyroid, testis, ovary, adrenal glands, and pituitary. SARS CoV-2 can impact the thyroid gland either directly (through viral infection of target cells) or indirectly (through the abnormal immune regulation) [1].

Thyroiditis, whether destructive or inflammatory, is a common condition that may be linked with an increase of pro-inflammatory cytokines, known as a "cytokine storm". A retrospective research found a link between thyrotoxicosis caused by systemic immune activation and elevated IL-6 levels in 287 non-ICU COVID-19 patients hospitalized. Sub-acute thyroiditis is a kind of thyroiditis that is closely linked to viral infections. Since the COVID-19 epidemic, many case reports have shown that SARS-CoV-2 is a probable cause. Furthermore, new research suggests that Graves' disease and Hashimoto's thyroiditis may develop as a result of COVID-19 infection. As a result, we planned to compile existing literature and conduct a systematic review to analyses the prevalence, clinical characteristics, and prognosis of autoimmune thyroid diseases caused by COVID-19 [2].

During our literature search, we discovered 20 patients with COVID-19-related autoimmune thyroid disorders. The great majority of the patients were middle-aged females. Thyroid problems in general had a benign course and responded well to medical therapy. In the documented cases, COVID-19 is believed to be a causal factor for Graves' disease and Hashimoto's thyroiditis, either as a fresh beginning or as a flare-up of the illness in remission [3].

Graves' disease is an autoimmune thyroid illness that is induced in susceptible individuals by various environmental factors such as viruses. One of the proposed pathways is supposed to be molecular mimicry. The illness begins with the collapse of immunological tolerance to TSH-R, TPO, and TG. Several infectious agents have been studied for their potential significance in Graves' disease a etiology. Well-known etiological agents include foamy viruses, Parvovirus B19, Epstein-Barr virus, and hepatitis C virus. Similarly, many viruses, such as hepatitis C and parvovirus B19, are suspected to have a role in an etiology of Hashimoto's thyroiditis; however this is not thoroughly understood as it is in Graves' disease. Thus, COVID-19 may have a role in the etiology of autoimmune thyroiditis in these individuals. However, while case studies highlight the possibility of a link between COVID-19 and thyroiditis, they are not confirmation of a causative relationship [4].

During a severe acute or chronic sickness, altered thyroid functions described as "non-thyroidal illness syndrome" (or low T3 syndrome, or thyroid sick syndrome) might be seen (trauma, sepsis, malnutrition, hepatic diseases, major systemic illness). Free T3, TSH, and FT3/FT4 levels were considerably lower in COVID-19 patients who were severely or critically sick than in those who were not. Another research that looked at thyroid function in 287 patients in a non-intensive care unit found that COVID 19 had over (10.8%) and subclinical (14.6%) thyrotoxicosis [5]. Because of systemic immunological activation caused by SARS-Cov-2 infection, serum IL-6 levels were shown to be substantially associated with TSH levels. Negative TRab, anti-TG, and anti-TPO levels in nine patients and spontaneous recovery during follow-up suggest that a plausible cause was destructive thyroiditis in these individuals. Pulmonary involvement was noted in 5 of the examined cases as a sign of disease severity. However, concomitant thyroid dysfunctions were severe in some individuals, and two patients died: one with Graves' disease and thyroid storm owing to adult respiratory distress syndrome, and another with hypothyroidism due to myxedema coma and sudden cardiac death [6].

CONCLUSION

In susceptible individuals, COVID-19 may induce autoimmune thyroid illness. It is appropriate to frequently monitor thyroid functions both during the acute phase and throughout

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convalescence in order to avoid missing a thyroid condition and delaying therapy, particularly in individuals with pre-existing autoimmune thyroid illnesses. Prospective research in the future may provide light on the link between SARS-COV-2 and thyroid autoimmunity.

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