Having thyroid and its risks on individuals during COVID 19.

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Introduction

Autoimmune thyroid disease (AITD) is still common, and patients with both an underactive and hyperactive thyroid should seek medical treatment if they are in the high-risk category. Thyroid abnormalities could be a one-time event, but they could also indicate the start of autoimmune polyglandular syndrome in the future. They can also happen before or after rheumatoid arthritis or connective tissue disease (RA). The mechanisms through which AITD may be linked to systemic autoimmune illnesses are still under investigation. However, it is thought that RA patients are not at high risk for COVID-19, and as immunological dysfunction is connected to AITD, this is likely to be true for AITD as well [1].

In laboratory testing, increased free thyroxine (T4) and triiodothyroxine (T3), suppression of thyrotropin (TSH), the appearance of anti-thyroglobulin antibodies, a surge in inflammatory markers and white blood cells, as well as an altered thyroid ultrasound picture, were all discovered. Thyroid function and imaging were both normal a month ago, which is crucial to remember. Furthermore, after two weeks on prednisone, the initial symptoms had vanished totally, and thyroid function and inflammatory markers had greatly improved.

Both SARS-CoV-1 and SARS-CoV-2 need ACE2 in conjunction with the transmembrane protease serine 2 (TMPRSS2) as the key molecular complex to enter and infect host cells. The thyroid gland, interestingly, has higher levels of ACE2 and TMPRSS2 expression than the lungs. Furthermore, the in silico approach reveals that in males and females, ACE2 expression levels in the thyroid are positively and negatively linked to immune signatures (CD8+ T cells, interferon response, B cells, and natural killer (NK) cells), which may help to explain differences in immune responses and the resulting thyroid manifestations [2].

Other cellular components and proteases are thought to play a supporting role in the uptake of SARS-CoV-2 by host cells. Integrins are a subset of plasma membrane structural proteins that may be implicated in SARS-CoV-2 cell invasion. ACE2 interacts with integrin to modulate downstream signal transduction. Thyroid hormones are considered to promote integrin internalisation by modulating the genes that make up the monomeric protein that makes up integrins. As a result, thyroid hormones may have a beneficial effect on SARS-CoV-2 uptake mediated by integrins.

Olfactory receptors (ORs) have been reported to be expressed peripherally in the cases of ACE2 and TMPRSS2, with a broad expression profile in the thyroid gland. An impairment of ORs signaling/function in the nasal neuro-epithelium or the olfactory bulb is the biological mechanism underlying the loss of smell (anosmia) in COVID19 patients. ORs are co-expressed with important mediators of SARS-CoV-2 cell entrance (ACE2, TMPRSS2, cathepsin L), therefore their elimination could be linked to COVID-19 complications in other organs, including the thyroid. Furthermore, SARS-CoV-2 may have an indirect effect on the thyroid gland because COVID-19-linked "hyperactivity of Th1/Th17 immunological responses" and "cytokine storm" may generate and maintain thyroid gland inflammation.

Thyrotoxicosis

A viral or postviral inflammatory process causes SAT (also known as De Quervain thyroiditis), a self-limiting thyroid ailment. "Painful subacute thyroiditis" is another moniker for it because neck discomfort is a defining aspect of the clinical condition. The clinical course of SAT usually comprises three phases: thyrotoxicosis for the first several months, hypothyroidism for three months, and then euthyroidism. Many viruses have been related to the start of SAT, and epidemiological, serological (or circulating viral genome) or direct evidence data may be used to prove infection.

SARS-CoV-2 infection was confirmed in three cases by the presence of viral RNA in oropharyngeal or nasopharyngeal swabs, as well as quantitative detection of serum specific IgG and IgM. In eight of nine cases, Covid-19 symptoms were mild, but the oldest patient with SAT developed interstitial pneumonia (i.e. 69 years old). It's worth mentioning that SAT occurred in six of nine patients (approximately 65%) after COVID-19 remission (i.e. clinical disappearance and negative viral detection tests), with a time gap between COVID-19 and SAT ranging from 17 to 40 days [3].

SAT, on the opposite hand, was evident at the time of admission or throughout the primary days of hospital care in 3 patients, and was coupled to SARS-CoV-2 infection symptoms. It's price mentioning that the patient with SARS-CoV-2-related respiratory illness had a positive management swab check 2 months once being diagnosed with COVID-19. With the exception of the oldest patient with SARS-CoV-2-

related respiratory illness World Health Organization was additionally receiving medicines for past back surgery, neck discomfort (which might radiate to the jaw and/or ear) was gift in eight of 9 cases (about 90%).

In five of the instances, the neck pain was accompanied by a fever (about 60 percent). Biochemical thyrotoxicosis can range in severity from mild to severe; in fact, peak serum free T4 (FT4) and free T3 (FT3) levels can be two times higher than the average upper limit. TSH receptor antibodies (TRAb) and thyroperoxidase (TPOAb) antibodies were all negative, however two people had thyroglobulin antibodies (TgAb), one of whom needed T4 for hypothyroidism. All of the patients' C-reactive protein (CRP) levels were high, ranging from 8 to 122 mg/L. Early-onset SAT symptoms and signs include goitre, lethargy, palpitations, inappetence, sweating, insomnia, anxiety, tremor, and weight loss [4].

Hypothyroidism

COVID-19-related primary hypothyroidism has been discovered in some studies. Primary hypothyroidism was found in 5.2 percent (15/287) of the patients in the study, which was subclinical in 90% of cases (FT3 and FT4 in reference ranges) and overt in the remaining 10%. COVID-19 patients with hypothyroidism had a higher in-hospital death rate than COVID-19 patients with euthyroidism, according to the study. Hypothyroidism, like thyrotoxicosis, can have a negative impact on COVID-19 results, albeit to a smaller extent.

COVID-19 patients admitted to high-level care institutions had primary hypothyroidism due to chronic autoimmune thyroiditis (CAT) (HICUs). Primary hypothyroidism appears to have developed in patients during COVID-19 and persisted after discharge. Seven days after modest COVID-19 remission, a case of overt primary hypothyroidism due to CAT was observed [5]. As a result, there's a potential you'll develop primary hypothyroidism during or after COVID-19.

Central hypothyroidism is defined as a low FT4 along with an extremely low/normal TSH. Only a few cases of hormonal changes compatible with central hypothyroidism caused by SARS-CoV-2 infection have been observed at the hypothalamus or pituitary level of the HPT axis. People hospitalised for non-mild COVID-19 who had low FT4 and low/normal TSH were found to have central hypothyroidism in 2–6% of cases, according to one study (one to three out of 50 patients). These hormonal changes were reversed after recovering from COVID-19, showing that COVID-19 may have acute/transitory effects on the HPT axis.

During the viraemic phase, SARS-CoV-2 might theoretically infect any organ, hence the thyroid and HPT axis must be considered while dealing with COVID-19.

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