Journal of
Diabetes and Clinical Practices

King, J Diabetes Clin Prac 2021, 4:6

Commentary Open Access

Significant Risk Factors of Thyroid in COVID 19

Robert King*

Faculty of Medicine, Universitas Padjadjaran, Indonesia

Introduction

Autoimmune thyroid disease (AITD) is still quite frequent, and people with both an underactive and hyperactive thyroid seek advice from their doctors if they are in a high-risk category. Thyroid abnormalities may be a one-off occurrence, but they might potentially signal the onset of autoimmune polyglandular syndrome in the future. They can also occur before or after connective tissue or rheumatoid arthritis (RA). The methods through which AITD may be connected to systemic autoimmune disorders are still being researched. However, it has been hypothesised that RA patients do not fall into a high-risk category for COVID-19, and as immunological dysfunction is linked to AITD, this is likely to be the case for AITD as well [1].

Increased free thyroxine (T4) and triiodothyroxine (T3), suppression of thyrotropin (TSH), the emergence of anti-thyroglobulin antibodies, and a rise in inflammatory markers and white blood cells, as well as an altered thyroid ultrasound picture, were all found in laboratory testing. It's critical to know that thyroid function and imaging were both normal a month ago. In addition, after two weeks of commencing prednisone medication, the initial symptoms had completely disappeared, and thyroid function and inflammatory indicators had significantly improved [2,3].

To enter and infect host cells, both SARS-CoV-1 and SARS-CoV-2 employ ACE2 in combination with the transmembrane protease serine 2 (TMPRSS2) as the essential molecular complex. Interestingly, the thyroid gland has higher amounts of ACE2 and TMPRSS2 expression than the lungs. In addition, the in silico approach reveals that 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) in males and females, respectively, helping to explain the differences in immune responses and the resulting thyroid manifestations. Other cellular components and proteases are considered to have a secondary role in SARS-CoV-2 uptake by host cells. Integrins are one of the primary groups of plasma membrane structural proteins that might be involved in SARS-CoV-2 cell invasion. To influence downstream signal transduction, ACE2 interacts to integrin. T4 modulates the production of the genes that make up the monomeric protein that makes up integrins, and thyroid hormones are thought to increase integrin internalisation. As a result, thyroid hormones may have a favourable impact on integrin-mediated SARS-CoV-2 uptake.

In the case of ACE2 and TMPRSS2, olfactory receptors (ORs) have been found to be expressed peripherally, with a broad expression profile in the thyroid gland. The molecular mechanism behind the loss of smell (anosmia) in COVID19 patients is an impairment of ORs signaling/function in the nasal neuro-epithelium or the olfactory bulb. ORs are co-expressed with major mediators of SARS-CoV-2 cell entry (ACE2, TMPRSS2, cathepsin L), hence it's possible that their destruction is implicated in COVID-19 sequelae from other peripheral organs, including the thyroid. Furthermore, SARS-CoV-2 may have an indirect effect on the thyroid gland, since "hyperactivity of Th1/Th17 immunological responses" and "cytokine storm" linked with COVID-19 may cause and maintain thyroid gland inflammation [4].

Thyrotoxicosis

SAT (also known as De Quervain thyroiditis) is a self-limiting thyroid condition caused by a viral or postviral inflammatory process. Because neck discomfort is a defining feature of the clinical illness, another term for it is "painful subacute thyroiditis." The clinical course of SAT generally consists of three phases: thyrotoxicosis for the first several months, hypothyroidism for roughly three months, and finally euthyroidism. Many viruses have been linked to the onset of SAT, and proof of infection might come from epidemiological, serological (or circulating viral genome) or direct evidence data.

In three cases, the presence of viral RNA in oropharyngeal or nasopharyngeal swabs, as well as quantitative detection of serum specific IgG and IgM, showed SARS-CoV-2 infection. Covid-19 symptoms were modest in eight of nine instances, but the eldest patient with SAT developed interstitial pneumonia (i.e. 69 years old). It's worth noting that SAT happened following COVID-19 remission (i.e. clinical disappearance and negative viral detection tests) in six of nine patients (about 65 percent), with a time gap from COVID-19 spanning from 17 to 40 days. In three patients, however, SAT was present at the time of admission or during the first days of hospitalisation, associated with symptoms of SARS-CoV-2 infection. It's worth noting that two months following the COVID-19 diagnosis, the patient with SARS-CoV-2-related pneumonia had a positive control swab test. Neck discomfort (which might radiate to the jaw and/or ear) was evident in eight of nine cases (about 90%), with the exception of the oldest patient with SARS-CoV-2-related pneumonia who was also taking medicines for previous back surgery.

Furthermore, in five cases, neck discomfort was accompanied by fever (about 60 percent). The severity of biochemical thyrotoxicosis can range from mild to high; in fact, maximal serum free T4 (FT4) and free T3 (FT3) levels can be two times the typical upper limit. TSH receptor antibodies (TRAb) and thyroperoxidase (TPOAb) antibodies were all negative, while thyroglobulin antibodies (TgAb) were present in two individuals, one of whom required T4 for hypothyroidism. C-reactive protein (CRP) levels were all high, ranging from 8 to 122 mg/L in all patients. Goiter, lethargy, palpitations, inappetence, sweating, sleeplessness, anxiety, tremor, and weight loss are all possible indications and symptoms of early-onset SAT.

Hypothyroidism

Some investigations have found cases of COVID-19-related primary hypothyroidism. Specifically, 5.2 percent (15/287) of patients

***Corresponding author:** Robert king, Faculty of Medicine, Universitas Padjadjaran, Indonesia; E-mail: robertK21@mail.unpad.ac.id

Received November 08, 2021; **Accepted** November 22, 2021; **Published** November 29, 2021

Citation: King R (2021) Significant Risk Factors of Thyroid in COVID 19. J Diabetes Clin Prac 4: 141.

in the research had primary hypothyroidism, which was subclinical in 90% of instances (FT3 and FT4 in reference ranges) and overt in the remaining 10%. The study also discovered that COVID-19 patients with hypothyroidism had a greater in-hospital death rate than COVID-19 patients with euthyroidism. Hypothyroidism, like thyrotoxicosis, although to a lesser level, may have a detrimental influence on COVID-19 results.

Primary hypothyroidism related to chronic autoimmune thyroiditis (CAT) were found among COVID-19 patients admitted to high-level care facilities (HICUs). Primary hypothyroidism appears to have occurred during COVID-19 in patients and remained after discharge. A instance of overt primary hypothyroidism owing to CAT was reported seven days after modest COVID-19 remission. As a result, there's a chance primary hypothyroidism might develop during or after COVID-19.

Low FT4 with an excessively low/normal TSH is biochemically classified as central hypothyroidism. Hormonal alterations compatible with central hypothyroidism caused by SARS-CoV-2 infection at the hypothalamus or pituitary level of the HPT axis have only been documented in a few cases [5]. In one research, individuals hospitalised for non-mild COVID-19 who had low FT4 and low/normal TSH were found to have central hypothyroidism in 2–6% (one to three out of 50 patients). After recovering from COVID-19, these hormonal alterations were reversed, indicating that COVID-19 may have acute/transitory effects on the HPT axis.

SARS-CoV-2 may theoretically infect any organ during the viraemic phase, and involvement of the thyroid and HPT axis must be considered while dealing with COVID-19.

References

1. Hoffmann M, Kleine-Weber H, Schroeder S, Nadine K, Tanja H et al. (2020) SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell 181: 271-80.
2. Kerslake R, Hall M, Randeva HS, Demetrios AS, Kamaljit C et al. (2020) Coexpression of peripheral olfactory receptors with SARSCoV2 infection mediators: Potential implications beyond loss of smell as a COVID19 symptom. Int J Mol Med 46: 949–56.
3. Brancatella A, Ricci D, Cappellani D, Viola N, Latrofa F, et al. (2020) Is subacute thyroiditis an underestimated manifestation of SARS-CoV-2 infection? Insights from a case series. J Clin Endocrinol Metab 105: e3742–e3746.
4. Tee LY, Hajanto S, Rosario BH (2020) COVID-19 complicated by Hashimoto's thyroiditis. Singapore Med J 62: 265.
5. Lania A, Sandri MT, Cellini M, Mirani M, Lavezzi E, et al.(2020) Thyrotoxicosis in patients with COVID-19: The THYRCOV study. Eur J Endocrinol 183: 381–7.