Special Issue

Autoimmune Disease and Malignancy of the Thyroid Gland in the COVID-19 Pandemic Era

Message from the Guest Editor

The SARS-CoV-2 virus utilizes the ACE2 receptor in conjunction with the transmembrane protease serine 2 (TMPRSS2) as the primary molecular complex that allows it to infect host cells and enter cells. It is interesting to note that the levels of ACE2 and TMPRSS2 expression in the thyroid gland are much higher than in the lungs because of this. When dealing with COVID-19, it is essential to keep in mind that there is a possibility of the thyroid being damaged. Following on from the previous two points, COVID-19 may be responsible for inflammation of the thyroid gland by inducing aberrant immune responses and a cytokine storm. Furthermore, there is accumulating evidence that the incidence and severity of thyroid disorders induced by COVID-19, such as thyrotoxicosis, hypothyroidism, and the transformation of pre-existing benign thyroid nodules into thyroid cancer, rose dramatically throughout the course of the pandemic. In light of this, research into the mechanisms through which COVID-19 increases vulnerability to thyroid autoimmune diseases and modifies thyroid cancer outcomes should receive great importance.

Guest Editor

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Deadline for manuscript submissions

closed (31 August 2023)

Biomedicines

an Open Access Journal by MDPI

Impact Factor 3.9
CiteScore 6.8
Indexed in PubMed

mdpi.com/si/163386

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