Hyperthyroidism and COVID-19: Cross-talk with the Renin-Angiotensin System

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Abstract

In December 2019, the new acute respiratory syndrome coronavirus 2 (SARS-Cov-2) emerged in Wuhan, China, with an infection of pandemic proportions. Data from Wuhan showed that mortality from coronavirus disease 2019 (COVID-19) is strongly associated with cardiovascular diseases. Similar to SARS-Cov-1, which was responsible for the SARS epidemic from 2002 to 2004, SARS-Cov-2 also utilizes the host protein angiotensin II-converting enzyme (ACE2) as a coreceptor to gain intracellular entry. Hence, upregulation of ACE2 has been proposed as a potential factor in infectivity and a higher risk of harmful outcomes of COVID-19. In this context, data obtained from experimental models of hyperthyroidism have demonstrated increased cardiac ACE2, which can theoretically facilitate SARS-Cov-2 entry. However, there is currently no consistent scientific research on how COVID-19 specifically affects hyperthyroid patients, and more clinical and experimental evidence is urgently required to clarify this point. In this review, we highlight important known and unknown features of COVID-19 related to ACE2 and hyperthyroidism.