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Perspective

COVID-19 Influencing Thyroid Dysfunction

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Recently, a first case-report of thyroid dysfunction following SARS-CoV-2 (COVID-19) infection, namely subacute thyroiditis was reported [1]. This female patient had a painful, enlarged thyroid gland [2]. The exact mechanisms that SARS-CoV-2 (COVID-19) causes thyroid dysfunction are not known. Nevertheless, mechanisms that are demonstrated through SARS virus are potential central mechanism [3-5], direct viral replication [6-9], interaction with thyroid-ACE2 receptor [10-12], and inflammatory response, apoptosis, and local damage [13-16]. SARS-CoV-2 (COVID-19) and thyroid dysfunction impact each other by: 1) Graves's ophthalmopathy with actively undergoing immunosuppressive therapy are likely to increase risk of severe coronavirus infection development [17]; 2) Patients with poorly controlled thyroid dysfunction, particularly those with thyrotoxicosis, may be at risk of thyroid storm [18]; and 3) Systemic disease, including COVID-19 are related to low-T3 syndrome or non-thyroidal illness [19]. As pregnant women with hyper- or hypothyroidism are at increased risk of development of more severe COVID-19 disease, they are particularly suggested to social distancing adherence [20]. In the first trimester of pregnancy, the preferred treatment is the lowest possible dose of propylthiouracil (PTU) [21].

In conclusion, the following endocrine service is suggested in the COVID-19 crisis: satellite blood-testing services, remote monitoring services, face-to-face appointments, and telephone and video consultation.

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