The Association Between Some Endocrine Conditions and COVID-19: A Review

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Abstract

The review aimed to understand and explain the association between some major endocrine conditions and COVID-19. Since March 2019, COVID-19 has been identified as a pandemic by the World Health Organization (WHO) and has infected millions of individuals worldwide. According to the literature review, endocrine disorders include diabetes, obesity, hypertension, and thyroid. The development and progress of COVID-19 patients could be affected but not yet approved by all the studies. In diabetes mellitus, COVID-19 may affect cytokines and increase releases of IL-1, IL-6, and complications caused by diabetes. In obese patients, there was an increased risk of progressing to severe COVID-19. Because of the international spread of severe acute respiratory syndrome coronavirus, clinicians should pay special attention to obese patients who should be monitored closely with timely and aggressive care. COVID-19 pathophysiology and risk in a high incidence of hypertension has been found in patients with COVID-19 and can be studied in many kinds of studies, such as in China. However, hypertension is considered one of the most significant risk factors for COVID-19. In addition to the above data on associations between COVID-19 and endocrine disorders, data on thyroid function or thyroid disease in COVID-19 is not yet available and cannot be commonly reported.

Keywords: COVID-19; diabetes mellitus; bidirectional; obesity; pathophysiology; blood pressure; hypothyroidism.

INTRODUCTION

Coronavirus disease has affected millions of individuals worldwide (COVID-199) (Mathew et al., 2020; Rostam et al., 2020). This latest coronavirus, which also includes Middle East Respiratory Syndrome (MERS)-CoV and SARS-CoV-1, is a human β-coronavirus. These viruses are predominantly connected to respiratory illnesses, such as pneumonia, ARDS, and pulmonary oedema (Di et al., 2020). Endocrine disorders are no exception, and some endocrine organs are at risk of direct or indirect COVID-19 damage. Although there is still no proof of a greater predisposition in patients with diabetes and obesity to contract the infection, the coexistence of both conditions leads to a worse prognosis since both conditions impart an impaired immune system (Marazuela et al., 2020). Evidence is growing to indicate that patients with endocrinopathies such as diabetes mellitus (DM), hypertension (HTN), obesity, and cardiovascular disease are at greater risk of complications associated with COVID-19. In COVID-19 non-survivors and serious cases, studies from the UK and US suggested a high prevalence of DM and obesity, HTN (49.7 %), obesity (48.3 %), DM (28.3 %), and cardiovascular disease (27.8 %) are the most widely identified cardiometabolic comorbidities related to COVID-19 in the US (Shekhar et al., 2020).

Metabolic syndrome is a constellation of cardiovascular risk factors, including abdominal obesity, high blood pressure, dysglycemia, atherogenic dyslipidemia, and pro-thrombotic and pro-inflammatory states. Metabolic syndrome is clinically characterized as the occurrence of three or more of the following factors: increased waist circumference (population and country-specific cutoff), hypertriglyceridemia (>150 mg/dL or hypertriglyceridemia treatment), elevated blood pressure (systolic 130 and diastolic 85 mm Hg or hypertension treatment history), elevated blood pressure (systolic 130 and diastolic 85 mm Hg or hypertension treatment history), High-density lipoprotein cholesterol reduction (<40 mg/dL in males; <50 mg/dL in females) and dysglycemia (<100 mg/dL or hyperglycemia treatment) (Bonora et al., 2018; Bansal et al., 2020). The severity of COVID-19 can be impaired by hypertension, diabetes, and coronary heart disease. In addition, it may be associated with an angiotensin-converting enzyme 2 (ACE2) deficiency, and a glucolipid metabolic disorder (GLMD) mediated cytokine storm (Chen et al., 2020). Several research studies have targeted the epidemiological and clinical features of patients infected with COVID-19, but the risk factors for severity and mortality have not been thoroughly investigated. Identifying major risk factors and taking effective
clinical steps will greatly contribute to saving lives (Zhou et al., 2020; Zaki et al., 2020).

**COVID-19 AND DIABETES MELLITUS**

Diabetes Mellitus (DM) is chronic with catastrophic multi-systemic complications and may be associated with extreme Coronavirus Disease 2019 (COVID-19) (Huang et al., 2020) Larger studies from China gave more consistent prevalence rates. Diabetes was present in 7.4 per cent of 1099 (median age of 47 years) (Guan et al., 2020), and 8.2 per cent of 1 590 (median age 48.9 years) hospitalized individuals in two multicenter national studies. Also, of 7337 people admitted to nineteen hospitals in the province of Hubei (median age 54 years), 952 (13.0%) had type 2 diabetes, while a survey by the Chinese Center for Disease Control and Prevention (China CDC), which also included non-hospitalized people, showed a lower prevalence of diabetes (5.3%) among 44,672 reported cases of COVID-19 through February (Pugliese et al., 2020).

Notably, Guo et al. data indicate that an initial milder appearance of SARS-CoV-2 infection may mask the seriousness of Covid-19 in diabetes, with fewer patients reporting fever, chill, chest tightness, and shortness of breadth (Maddaloni et al., 2020). In addition, the fundamental and clinical science of the possible interrelationships between diabetes mellitus and COVID-19 was investigated (Drucker 2020). However, knowledge in this area is quickly emerging, with numerous publications appearing frequently. This review summarizes recent developments in diabetes mellitus and COVID-19 and emphasizes clinical guidelines for patients at risk of or affected by COVID-19 with diabetes mellitus. Unfortunately, most accessible research does not differentiate between diabetes mellitus and, due to its high prevalence, focuses mostly on T2DM (Kumar et al., 2020).

Compared to non-diabetics, diabetes in patients with COVID-19 is associated with a two-fold rise in mortality and COVID-19 severity. More research is needed on pathogenic pathways and therapeutic effects (Kumar et al., 2020). Several data sets from China, Italy, and the USA have consistently recorded that in patients with advanced age (>70 years of age) and pre-existing comorbidities, primarily diabetes mellitus (DM), hypertension, and cardiovascular disease, the clinical course of COVID-19 is more serious (Yang 2020). The DM and COVID-19 relationship are bidirectional. On the one hand, DM will increase the risk of SARS-CoV2 contraction and further complicate the clinical path of COVID-19, resulting in increased severity and mortality (Onder 2020).

Certain clinical and biological features determine high-risk phenotypes within the DM population, and such prognostic markers need to be characterized in future studies (Smail et al., 2022). In the sense of patient-tailored precision medicine, which emerges as an urgent priority in the era of COVID-19, more research is required to explore which subgroups of DM patients are anticipated to benefit most from particular antiviral, immunomodulatory and other treatment strategies (Koliaki et al., 2020). Given the high risk, people with DM should take special precautions during the COVID-19 pandemic. The norm should be strict social distancing and proper handing hygiene. Good glycemic regulation should be of utmost significance as it has been shown to improve the innate immune system. Although it would be prudent to adhere to or intensify the ongoing treatment, doctors may consider reviewing the prescription (Pal et al., 2020). Compromised innate immunity, pro-inflammatory cytokine environment, decreased ACE2 expression, and the use of antagonists of the renin-angiotensin-aldosterone system in people with diabetes mellitus lead to a weak COVID-19 prognosis. Direct β-cell injury, cytokine-induced insulin resistance, hypokalemia, and drugs used to treat COVID-19 (such as corticosteroids, lopinavir/ritonavir) can, on the other hand, lead to a worsening of glucose control in individuals with diabetes mellitus (Pal et al., 2020).

Several studies have attempted to understand the potentially increased vulnerability of diabetes patients to SARS-CoV-2 infection. However, no data have shown that these patients are at higher risk of contracting COVID-19. Via the ACE2 receptor, SARS-CoV-2 reaches the host cell. While consensus on the role of ACE2 in the crosstalk between diabetes and COVID-19 has not yet been achieved, some argue that diabetes patients have elevated ACE2 expression, thus facilitating viral entry and subsequent replication. Others show that patients with diabetes have low levels of ACE2 and that other causes, such as treatment with ACE/ARBs, hypoglycemic agents, and statins, are responsible for the observed rise in ACE2 (Azar et al., 2020). The degree to which clinical and demographic variables moderate this relationship is uncertain, although signs link higher BMI and higher HbA1c to worse outcomes in people with COVID-19 diabetes. COVID-19 also risks leading to worse diabetes outcomes due to disturbances caused by the pandemic, including stress and changes in routine treatment, diet, and physical activity, and posing immediate risks to people with diabetes (Hartmann et al., 2020).

Dysregulated post-infection immune response is characterized by delayed and reduced recruitment in lung tissue of CD4+ T cells, inflammatory monocytes, and macrophages. In addition to the reduced overall CD4+ T cell response, infected diabetic mice also showed a more prominent Th17 response with increased IL-17a levels, implying that a shift in cytokine profiles could be partially responsible for the severity of the disease (Angelidi et al., 2020) An independent risk factor for the prognosis of COVID-19 is diabetes.
Therefore, the prevention and treatment of diabetic patients, especially those needing insulin therapy, should be given greater attention (Shang et al., 2020). From 43 reports, the meta-analysis was based on 23007 patients. The pooled prevalence of diabetes in patients infected with COVID-19 was 15% (95% CI: 12%–18%), p = <0.0001. Furthermore, in COVID-19 patients with diabetes, the risk of mortality was found to be substantially higher relative to COVID-19 patients without diabetes, with a pooled risk ratio of 1.61 (95% CI: 1.16–2.25%), p = 0.005 (Hussain et al., 2020).

The pandemic of COVID-19 has questioned both institutional and diabetes self-management. Continuing social distancing and lockdowns have adversely influenced access to care and self-management (Mukona et al., 2020) SARS-CoV-2 infected T2DM patients reported decreased body mass index (BMI), lymphocytes, UA, albumin levels, and increased CRP levels. Oxidative stress response and nutritional intake may be associated with reduced BMI, UA, and albumin levels. In addition, the infection may be associated with reduced lymphocyte counts and elevated CRP levels (Liang et al., 2020). There are major concerns about worsening glycemic regulation, inaccessibility of suitable drugs, inaccessibility of health care or infection with SARS-CoV-2, and worse results during the COVID-19 pandemic. Although there are some recommendations for diabetes treatment and related complications during the COVID-19 pandemic, very few discuss the psychological problems of people with diabetes (Singhai et al., 2020).

Several specialist bodies have temporarily updated gestational diabetes mellitus (GDM) testing guidelines during the COVID-19 pandemic to minimize person-to-person contact. The current temporary Australian guidelines indicate that no glucose tolerance test (GTT) is needed if the fasting glucose is around 4.6 mmol/L (Van et al., 2020). A common medical condition in pregnancy is gestational diabetes mellitus (GDM). The Australasian Pregnancy Diabetes Society (ADIPS) suggests monitoring all women in each pregnancy, preferably using a two-hour glucose tolerance test (GTT). The diagnostic criteria derive from World Health Organization (WHO) guidelines based on the findings of the Hyperglycaemia and Pregnancy Outcomes (HAPO) report (Metzger et al., 2008). In patients with diabetes, there is evidence of an increased incidence and severity of COVID-19. The pathophysiology of diabetes could be influenced by COVID-19. Therefore, it is critical for patients infected with COVID-19 to regulate blood glucose and those without the disease. Innovations such as telemedicine effectively treat diabetes patients today (Singh et al., 2020). In patients infected with various viruses, including the 2009 pandemic influenza A (H1N1), SARS-CoV, and MERS-CoV, diabetes and uncontrolled glycaemia have been identified as important predictors of severity and deaths. Some studies have not found a strong link between diabetes and serious disease in the emerging SARS-CoV-2 pandemic. However, other studies from China and Italy have shown that elderly patients with chronic diseases, including diabetes, are at higher risk of significant COVID-19 and mortality, respectively (Hussain et al., 2020). The mortality rate was 9.9% in a meta-analysis of hospitalized patients in China with a diagnosis of COVID-19. A higher incidence of diabetes mellitus was separately associated with a worse prognosis. The independent impact of Covid-19 mortality on diabetes mellitus should be seen as hypothesis-generating and needs further research (Miller et al., 2020).

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COVID-19 AND OBESITY

According to estimates from the World Health Organization, obesity is a global epidemic, with at least 2.8 million people dying each year due to becoming overweight or obese (Hussain et al., 2020). Obesity is a condition characterized by the assistance of extra adiposity tissue that provides substantial morbidity and mortality due to its complications linked to several weights. Therefore, the diagnostic comparison should consist of an anthropometric test that indicates elevated fat mass and the extent to which the well-being of individual patients is adversely affected by excess adiposity (Smal 2019 and Smail et al., 2021). Although the effects of COVID-19 on obese patients have not yet been well established, the experience of H1N1 influenza can serve as a warning for treating obese patients, particularly patients with extreme obesity. From 2009 to 2010, adult obesity and extreme obesity rates rose between 2017 and 2018 and now stand at 42% and 9%, respectively (Hales et al., 2020).

The findings indicate that relative to the H1N1 experience, the proportion of patients with obesity, extreme obesity, and COVID-19 infections will increase, and the disease will likely have a more severe...
path in such patients. These results further highlight the need for improved attention, diagnosis and testing priority, and aggressive care for obesity patients and COVID-19 infections (Dietz et al., 2020). Although the data on the effect of SARS-CoV-2 in people with obesity are minimal, and their relationship has not yet been fully established, it has been found that people with excessive body weight may experience a more severe COVID-19 infection (Puig et al., 2020). As stated earlier, it is especially noticeable in people affected by other risk factors associated with a more serious disease course. This correlation was also noted during the 2009 influenza A pandemic, which identified obesity as an independent risk factor for complications. Obesity patients suffered more than 40 per cent longer after a high-fat diet than people without excessive body weight (Kassir 2020). Therefore, obesity during the COVID-19 outbreak seems to require more attention, especially in western countries where obesity prevalence is high (Rychter et al., 2020).

There was an increased risk of progressing to extreme COVID-19 in obese patients. Since severe acute respiratory syndrome coronavirus two will continue to spread internationally, clinicians should pay particular attention to obese patients who should be handled closely with prompt and aggressive treatment (Cai et al., 2020). For variables associated with COVID-19 risk, severity, and their potential for decreased therapeutic and prophylactic treatments among these individuals, mechanistic mechanisms for obesity are presented in detail. The substantial main changes in morbidity and mortality from COVID-19 are correlated with individuals with obesity. Several mechanisms explain this impact jointly. A major concern is that vaccinations for obesity may be less successful (Popkin et al., 2020).

In theory, early intubation benefits identified in patients with COVID-19 can indicate OSA alleviation in some patients. Similarly, concerns of contamination from using nasal PAP in some patients with obstructive sleep apnea could lead to deterioration. Mechanistic research is encouraged, given the possible connection between OSA, obesity, and COVID-19 (McSharry et al., 2020). In young patients, obesity is a significant predictor of COVID-19 severity. The key mechanism is connected to liver and kidney damage (Deng et al., 2020). Study shows that patients with overweight and obese who have COVID-19 are at higher risk for mortality and intubation than those with typical BMI. These results support the hypothesis that obesity is a risk factor for complications of COVID-19 and should be considered for COVID-19 management (Nakeshbandi et al., 2020). In addition, there was an increased risk of progressing to extreme COVID-19 in obese patients. Since severe acute respiratory syndrome coronavirus two will continue to spread internationally, clinicians should pay particular attention to obese patients who should be handled closely with prompt and aggressive treatment (Cai et al., 2020).

Obesity, especially in male and younger populations, plays a significant role in the risk of death from COVID-19. The lack of impact of racial/ethnic and socioeconomic inequalities on mortality can be explained by the capitated system with more equalized access to health care. The data highlight the leading role of extreme obesity over correlated risk factors, which offers an early intervention target (Tartof et al., 2020). The position of obesity, particularly given its high prevalence worldwide, is of great relevance. However, it is important to bear in mind that the assessment of obesity through BMI is highly arguable, particularly in older people. First, since its numerator (i.e. bodyweight) comes from the fat and fat-free mass amount, it should be noted that BMI does not strictly reflect adiposity. Second, the cut points that categorize overweight and obese are subjective and cohort-based for young and middle-aged people and insufficient for older people (Sattar et al., 2020). Third, with age, body fat accumulates with a decrease in muscle mass. Obesity is often underestimated in older people, who may have excess adiposity within the normal/overweight body size (so-called sarcopenic obesity). Finally, ethnic differences can determine significant variability in body fat distribution, especially regarding ectopic and visceral fat (Azzolino et al., 2020). This relative inefficiency reveals a reduced ventilatory reserve and a risk for respiratory failure, even in mild systemic or pulmonary conditions. Taken together, any respiratory insults will seriously compromise the already attenuated respiratory system of obese subjects, ensuring that these patients will fail to recover if they have acquired any serious diseases that can deleteriously impact respiratory function, such as COVID-19 (Albashir et al., 2020).

The results indicate an interplay and dynamic pathophysiology between COVID-19 and diabetes. For clinical practice and public health, it is a significant priority to understand how diabetes can lead to extreme COVID-19 and how COVID-19 can worsen diabetes pathophysiology or its effects. To counter this, a global registry (COVID) was introduced to promote the study of COVID-19-related diabetes manifestations, their effects, and best care (Rubino et al., 2020). A wealth of data has emerged on this subject over the short period of the COVID-19 pandemic, but important questions still need to be addressed. Here, the current literature on the relationship between COVID-19 and diabetes is reviewed, and future studies' priorities are considered (Vas et al., 2020).

The role of cytokines, rapamycin mammalian target (mTOR), and altered polarization of natural killer cells in the hazardous relation between COVID-19 and obesity. These pathways encourage and accelerate the deleterious downstream cellular effects of SARS-CoV-2. Also, it is well known that obesity is associated with
decreased lung capacity and poor response to mechanical ventilation, putting these individuals at high risk of serious COVID-19 disease and mortality. Also, obesity can lead to other complications, such as renal failure, cardiovascular disease, hypertension, and vascular injury, further speeding up the negative clinical outcomes of COVID-19. Obese individuals should be protected from possible viral exposure to SARS-CoV-2 with compulsory safety devices and social distancing considerations (Caci et al., 2020). It is stated that people with obesity or overweight are less involved. Moreover, community health centres, gyms, swimming pools, and parks have been closed by law in many countries as part of their quarantine policy during the COVID-19 pandemic. Such diet and social shifts may have led to a rise in body weight in individuals with obesity and the general population (Lim et al., 2020).

Many variables could be involved in the worsening of COVID-19 obesity. Obesity can increase the expression in the bronchus and blood of ACE2 and CD147-related genes, while the latter are SARS-CoV-2 receptors that invade (Radzikowska et al., 2020). This would make obese subjects more vulnerable to infection and could, to some degree, explain the higher positive SARS-CoV-2 test rate. Obesity and concomitant metabolic syndrome can cause possible harm to the function of the organ, making it more vulnerable to failure in the lungs, kidneys, and other organs (Yang et al., 2020). However, in some acute diseases, including acute respiratory distress syndrome, where patients with obesity may have better outcomes, some have proposed an obesity paradox; it is uncertain if this phenomenon exists in patients with COVID-19 (Goyal et al., 2020). On the one hand, emerging evidence indicates that obesity is a risk factor in adults for a more extreme and complicated course of COVID-19. On the other hand, the health emergency triggered by the epidemic diverts attention to infectious diseases from preventing and treating non-communicable chronic diseases (Dicker et al., 2020).

Most COVID-19 patients die due to COVID-19 pneumonia after requiring artificial ventilation for hypoxemic respiratory failure. Emerging COVID-19 lung post-mortem histopathology provides insights into the underlying pathophysiology. In short, there is evidence of diffuse alveolar injury, as in other types of viral pneumonia, but sometimes this is patchy (Lockhart et al., 2020). Therefore, it is important to collect anthropometric information for patients with COVID-19 because of the potentially critical role of body weight or adiposity in determining the incidence and severity of pneumonia (and probably other complications) (Stefan et al., 2020). Therefore, special medical care and effective intervention should be undertaken during hospitalization and later clinical follow-up in obesity patients with COVID-19, especially those with additional comorbidities (Kang et al., 2020).

It is unclear if this is driven by these populations' higher prevalence and risks of obesity (driving diabetes and hypertension). Regarding progression to more extreme severity requiring acute treatment or death, we do not yet have data on results for those with obesity and less severe COVID-19 disease. Such data would be necessary to fully understand the risk of mortality for those with obesity, particularly with the understanding that survival of critical care may be greater for those with moderate levels of obesity (Fine et al., 2020). Absolute and central obesity, in short, are risk factors for hospital admission to COVID-19. Even with a moderate weight gain, the high risk was evident. Impaired glucose and lipid metabolism may be involved in the mechanisms (Hamer et al., 2020).

**Figure 2.** Potential pathways that increase the risk of serious COVID-19 illness and death in individuals with obesity. There are a variety of possible mechanisms by which obesity can impact adverse COVID-19 outcomes. These include chronic inflammation, respiratory function deficiency, pulmonary perfusion, practical concerns in critical care environments when treating obese patients, immune dysregulation, obesity, metabolic and vascular complications, and relative decreases in main hormones. TNF-alpha, tumour necrosis factor-alpha; FRC, residual functional potential (Kwok et al., 2020).

**HYPERTENSION AND COVID-19**

Pathophysiology and risk in patients with COVID-19, a high incidence of hypertension have been observed, with HTN likely predisposing them to an increased risk of more serious illness. The danger may stem from several factors. First, hypertension is mainly associated with immune dysregulation, which occurs as higher levels of IL-17, the irregular activity of natural killer cells, and cytotoxic defects of T cells partially reversible by mineralocorticoid receptor antagonists (Shekhar et al., 2020). Therefore, the initially reported correlation between hypertension and hospitalization rates for COVID-19 in China is not surprising. Indeed, the
proportion of self-reported hypertension was 12.6% in a wide database of 20,982 patients with diagnosed COVID-19 infections and information on underlying diseases (Kreutz et al., 2020).

It is unknown whether or not uncontrolled blood pressure is a risk factor for COVID-19 acquisition or whether or not controlled blood pressure is less of a risk factor for hypertension patients. However, some organizations have also emphasized that blood pressure regulation remains a significant factor in minimizing the disease burden, even though it does not impact the vulnerability to SARS-CoV-2 viral infection (Schiffrin et al., 2020). Hypertension adversely impacts the health status of patients with COVID-19. However, broad prevalence studies showing the effects of comorbid diabetes and hypertension are urgently needed (Parveen et al., 2020). However, the outbreak of COVID-19 may also be a specific moment when specialists in pulmonary hypertension have to balance the costs and benefits of diagnostic work-up, including possible exposure to COVID-19 versus the initiation of targeted pulmonary arterial hypertension therapy in a select high-risk, high probability World Symposium Pulmonary Hypertension Group 1 patient with pulmonary arterial hypertension (Rayan et al., 2020). A severe and possibly fatal manifestation of COVID-19 is cardiac involvement and SARS-CoV-2-associated myocarditis. Hypertension treatment with a drug that can minimize inflammation in viral myocarditis and does not pose a theoretical risk of encouraging the proliferation of COVID-19 would appear to be a sensible strategy for improving patient outcomes (Antwi-Amoabeng et al., 2020).

Recent studies have shown that among patients with COVID-19, arterial hypertension, diabetes, cardiovascular disorders, and chronic obstructive pulmonary disease are prevalent. Research on the outcome of these patients is scarce, and there is very little evidence. Nevertheless, hypertension is one of COVID-19's most important risk factors. The relationship between hypertension and negative outcomes is still questionable (Tadic et al., 2020). Renin-angiotensin system (RAS) dysfunction has been identified in patients with coronavirus infection (COVID-19). Still, it remains unclear whether RAS inhibitors, such as angiotensin-converting enzyme inhibitors (ACEIs) and type one receptor blockers (ARBs) of angiotensin II, are associated with clinical outcomes. COVID-19 hypertensive patients have been enrolled to test the impact of RAS inhibitors (Meng et al., 2020). Protein for ACE2-expressing cells to join. ACE-2 is a part of activating the renin-angiotensin (RAS) system that plays an important role in hypertension. This connection between ACE2 and SARS-CoV-2 sparked interest in investigating the relationship between inhibitors of RAS and infection with COVID-19 (Salah et al., 2021). Hypertension was associated with increased poor composite outcomes in patients with COVID-19, including mortality, extreme COVID-19, ARDS, need for ICU treatment, and disease progression (Pranata et al., 2020).

THYROID AND COVID-19

The immune system, especially cellular immunity, is modulated by thyroid hormones. The dysregulation of the immune system in hypothyroidism might increase the risk of infection. However, the immune system recovers its normal function after proper hormone replacement therapy (Mitrou et al., 2011). The influence of pre-existing hypothyroidism on the effects of COVID-19 is unclear (van Gerwen et al., 2020). Effects of COVID-19 on the hypothalamic-pituitary-thyroid axis: It is known that systemic disorders are associated with low-T3 or non-thyroidal syndrome. Severe COVID-19 is expected to cause such a disease, especially when the infection is associated with fever and lower respiratory tract involvement. In addition, SARS-CoV-2 infection has been reported to influence the nervous system, typically affecting smell and taste with the involvement of cranial nerves (Boelaert et al., 2020).

Follicular cell death would manifest as low T3 and T4; damage to parafollicular cells would potentially result in low serum calcitonin levels. This has been suggested as a possible femoral head osteonecrosis mechanism in recovered SARS patients; calcitonin deficiency contributes to osteoclast disinhibition leading to osteonecrosis (Kaiser et al., 2020). Data on thyroid function or thyroid pathology in COVID-19 is not yet available. A consensus statement concerning problems related to thyroid dysfunction during the COVID-19 pandemic has been released by the British Thyroid Association and the Society for Endocrinology (BTA/SIE). Patients with underlying hypothyroidism or hyperthyroidism are recommended to continue their prescription drugs as normal (Pal et al., 2020). Hyperthyroidism is an endocrine condition characterized by the thyroid gland's inappropriately high thyroid hormone output. It is usually caused by thyroid gland autoimmune dysfunction and has a population prevalence of 1%-15% (Kaur et al., 2020). In addition to clear ultrasonographic evidence indicating subacute thyroiditis, the patient presented with tachycardia, anterior neck pain, and thyroid function tests showing hyperthyroidism. Corticosteroid therapy resulted in a fast clinical resolution. The case shows that virus-related subacute thyroiditis such as SARS-CoV-2 should be regarded as a complication of COVID-19 and treated as a differential diagnosis in infected patients with tachycardia without evidence of developing COVID-19 disease (Mattar et al., 2020).
CONCLUSIONS

I reached several important points from this review. First, patients with endocrine disorders such as diabetes, obesity, hypertension, and thyroid may be more likely to affect by COVID-19, but still controversial from one study to study. More data should be gathered to establish these associations.

Conflict of Interest: Author declare that there is no conflict of interest.

REFERENCES


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