Aksaray University Journal of Medicine Sciences
Aksaray Üniversitesi Tıp Bilimleri Dergisi

asujms 2025; 5(1):33-35

Case Report

Graves' Thyrotoxicosis Case Presenting with COVID-19

COVİD-19 ile Prezente Graves Tirotoksikoz Olgusu

Uğur ERGÜN[1*]
[1*]Balıkesir Atatürk City Hospital, Internal Medicine, Balıkesir / TÜRKİYE

Abstract

Graves' disease is an autoimmune multifactorial thyroid disease caused by environmental factors in genetically susceptible patients. Viral infections are thought to play a role in the pathophysiology of this disease. Several potential mechanisms have been proposed for the pathophysiology of Graves' disease, including antigen exposure, cytokine release and inflammatory response. A review of the literature shows that subacute thyroiditis developing after viral infection has been reported as Graves' disease. It is a potential risk factor for Graves' disease and should be considered in the differential diagnosis in the etiology. For this purpose, we present a case compatible with Graves' disease that developed after COVID-19 viral infection, which will contribute to the literature.

Keywords: Graves' disease, angiotensin-converting enzyme 2, COVID-19

Öz

Graves hastalığı, genetik olarak duyarlı hastalarda çevresel faktörlerin neden olduğu otoimmün multifaktöriyel bir tiroid hastalığıdır. Viral enfeksiyonların bu hastalığın patofizyolojisinde rol oynadığı düşünülmektedir. Graves hastalığının patofizyolojisi için antijene maruz kalma, sitokin salınımı ve enflamatuar yanıt gibi çeşitli potansiyel mekanizmalar önerilmiştir. Literatür incelendiğinde viral enfeksiyon sonrası gelişen subakut tiroiditini Graves hastalığı olarak bildirilen yayınlar bulunmaktadır. COVID-19 viral enfeksiyonu Graves hastalığı açısından potansiyel bir tetikleyici faktör olduğu, etiyolojide ayırıcı tanılarda düşünülmesi gerekmekedir. Bu amaçla literatüre katkı sağlayacağı COVID-19 viral enfeksiyon sonrası gelişen Graves hastalığı ile uyumlu olguyu sunuyoruz.

Anahtar Kelimeler: Graves hastalığı, angiotensin dönüştürücü enzim-2, covid-19

Corresponding Author: Uğur ERGÜN
Balıkesir Atatürk City Hospital, Internal Medicine , Balıkesir /TÜRKİYE
E-mail: mdbalkes10@gmail.com
ORCID: 0000-0002-6111-0030

Recieved : 30.08.2024
Accepted : 13.09.2024

INTRODUCTION

It is a new coronavirus that first emerged in the Chinese city of Wuhan, creating a pandemic impact. Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), known as the seventh virus in the viral classification, is known to infect humans. It has often been associated with cross-species infections and occasional cases of worldwide spread. According to available evidence, transmission via droplet and contact routes is known to occur. It starts to be seen between 4-6 days after clinical exposure, which occurs with symptoms such as coughing and sneezing. However, studies have shown that the incubation period can last up to 14 days. The most common symptoms reported in the literature are fever, sputum, fatigue, malaise, cough, etc., which are seen in other viral infections such as seasonal flu (1). Graves' disease (GD) is an autoimmune disease characterized by diffuse goiter and hyperthyroidism. It is generally seen in female patients between the ages of 20-40. When clinical findings are examined, symptoms such as irritability, palpitations, inability to tolerate heat, sweating, etc. are observed due to excess thyroid hormone within the scope of physical and psychological symptoms. Involvements such as ophthalmopathy, dermopathy and acropathy are specific to the disease. Increased serum tetraiodothyronine and/or triiodothyronine levels, immeasurably suppressed serum Thyroid Stimulating Hormone (TSH) levels, antibodies against TSH receptors (TRAB), and increased blood flow along with a heterogeneous appearance in the parenchyma on ultrasonography are the main diagnostic criteria. Traditional options such as Anti-thyroid Drugs (ATDs), Radioiodine (RAI) or surgery are used in the treatment method. It has remained largely unchanged for many years, although many young people relapse after a course of ATDs or require lifelong thyroid hormone replacement after definitive treatment (2).

Generally, the clinical severity of SARS-CoV-2 is broad-spectrum, and as a result of studies, current evidence suggests that Coronavirus Disease 2019 (COVID-19) can affect organs and systems. Angiotensin Converting Enzyme 2 (ACE2) plays an important role in the pathogenesis of COVID-19 and plays a role in the internalization of SARS-CoV-2 into human cells. ACE2 is expressed in thyroid follicular and parafollicular cells and allows SARS-CoV-2 to enter the thyroid gland. The mechanism has not been clearly elucidated, and this has led to interest in determining whether a history of thyroid dysfunction is associated with a worse clinical course of COVID-19 or an increased risk of SARS-CoV-2 infection. When the literature is reviewed, several studies have been conducted on the subject, and it seems that neither a history of hyperthyroidism nor a history of hypothyroidism is associated with worse clinical outcomes (3).

It is obvious that viral factors in the etiology of GD play a role. In this case, the clinical findings of thyrotoxicosis that occurred after viral infection were important. We think that SARS-CoV-2 infection, which we consider as a potential viral trigger, may have this effect through the ACE2 mechanism. In this context, we present it as a contribution to the literature in order to direct studies aimed at elucidating the relationship between thyroid diseases that may arise from subacute thyroiditis and that it should be considered in differential diagnoses

CASE REPORT

A 25-year-old male patient was admitted to the emergency clinic with excessive sweating, palpitations, weight loss and diarrhea. The patient stated that his symptoms were present for the last two weeks and they were progressively worsening, and he has lost 15 kilograms of his body weight in the last month. After the initial assessment, the patient was consulted to the endocrinology department. The patient did not have a significant prior medical history other than COVID-19, which was confirmed with positive Polymerase Chain Reaction (PCR) test. His COVID-19 symptoms included myalgia, fever, and cough. He was diagnosed with mild viral pneumonia and did not require hospitalization. He had a complete resolution from COVID-19 one month before the emergency service visit. His family history included hypothyroidism in his mother. On physical examination, his body temperature was 36.8 ^{0}C, heart rate was 130 beats per minute, blood pressure was 120/80 mmHg and respiratory rate was 22 per minute. His skin was warm and sweaty. He had punctate hyperpigmented lesions on the skin. Comprehensive metabolic panel revealed that glucose 95 g/dL, sodium 137 (135-142) mmol/L, potassium 4.2 (3.5-5.5) mmol/L and creatinine 0.97 (<1.10) mg/dL levels were in the normal range and TSH, Thyroxine (T4) and Triiodothyronine (T3) levels were 0.015 (0.30-5.00) U/L, 4.78 (0.6-1.6) ng/dL and 24.78 (2.10-4.50) ng/dL respectively. These results prompted consideration for a thyroid abnormality. Thyroid ultrasound imaging revealed heterogeneous and increased vascularization of the thyroid gland parenchyma. The patient was diagnosed with GD and was prescribed propranolol 40 mg twice daily, and methimazole 40 mg once daily. Thyroid function tests one month later were repeated and TSH, T4 and T3 levels were 0.015 (0.30-5.00) U/L, (1.31 (0.6-1.6) ng/dL, 6.17 (2.10-4.50) ng/dL respectively. The patient was followed up with medical treatment.

DISCUSSION

In this study, we present a case of Graves' disease after COVID-19 infection. The patient had no history of thyroid disease and no history of drug use, and had no symptoms of thyrotoxicosis. Whether COVID-19 had contributed to the development of GD, or the occurrence of GD was coincidental, requires further

studies (4,5). The relationship between Graves' disease and other viral infectious diseases has not been clarified despite studies. Various potential mechanisms for the pathophysiology of GD have been proposed, such as antigen exposure, cytokine release, and inflammatory response. There are reports showing that thyrotoxicosis clinic usually occurs after subacute thyroiditis following viral infectious diseases (6).

The first step in COVID-19 pathophysiology is viral entry via binding of the viral trimeric spike protein to the human receptor ACE-2, which is considered the main factor in transmission. Previous studies have linked SARS-CoV-2 infection with subacute thyroiditis and other autoimmune diseases such as systemic lupus erythematosus and cold agglutinin disease (7-8). It is clear that COVID-19 infection causes multiple clinical manifestations that can affect all body systems compared to a simple viral infection (9). At the same time, the importance of stress in the development of thyrotoxicosis in GD patients is still under debate. Cross-sectional studies have shown that stressful life events are more common in the months before the development of GD. Other studies have also found that the time between possible exposure and onset of disease symptoms in cases of subacute thyroiditis associated with COVID-19 infection is shorter than in pre-pandemic cases (10).

In our case, clinical findings of thyrotoxicosis following SARS-CoV-2 infection were significant. This is a rare case report of which we suggest SARS-CoV-2 as a potential viral trigger for the development of GD.

CONCLSION

In conclusion, SARS-CoV-2 infection is a potential risk factor for GD and should be considered in the differential diagnosis in the etiology. Further studies are needed to clarify the relationship between thyroid diseases that may arise from subacute thyroiditis after viral infection such as COVID-19.

Declarations

Financial Diclosure: The authors declared that this study has received no financial support.
Patient’ Consent: Written informed consent was obtained from the patient who participated in this case.
Competing Interest: The authors declared no competing interest.

REFERENCES

1. Chams, N., Chams, S., Badran, R., et al. COVID-19: a multidisciplinary review. Frontiers in public health. 2020;8:383.
2. Lane, L. C., Wood, C. L., Cheetham, T., et al. Graves’ disease: moving forwards. Archives of disease in childhood. 2023;108(4):276-281.
3. Barajas Galindo, D. E., Ramos Bachiller, B., González Roza, L., et al. Increased incidence of Graves' disease during the SARS-CoV2 pandemic. Clinical Endocrinology. 2023;98(5):730-737.
4. Brancatella, A., Ricci, D., Viola, N., et al. Subacute thyroiditis after SARS-CoV-2 infection. The Journal of Clinical Endocrinology & Metabolism. 2020;105(7): 2367-2370.
5. Asfuroglu Kalkan, E., Ates, I. A case of subacute thyroiditis associated with Covid-19 infection. Journal of endocrinological investigation. 2020; 43:1173-4.
6. Swift, T., People, M. ACE2: Entry Receptor for SARS-CoV-2. Science. 2020;367(6485): 1444-1448.
7. Paul L. Swiecicki, Livia T. Hegerova, Morie A. Gertz. Cold agglutinin disease. Blood, The Journal of the American Society of Hematology. 2013;122 (7):1114–1121.
8. Stasiak M., Lewinski A. New aspects in the pathogenesis and management of subacute thyroiditis. Rev Endocr Metab Disord. 2021;(1):1–3.
9. Ross DS, Burch HB, Cooper DS, Greenlee MC, Laurberg P, Maia AL, et al. American Thyroid association guidelines for diagnosis and management of hyperthyroidism and other causes of thyrotoxicosis. Thyroid. 2016; 26:1343–1421.
10. Mattar SA, Koh SJ, Rama Chandran S, Cherng BP. Subacute thyroiditis associated with COVID-19. BMJ Case Rep. 2020;13(8):e237-336.