

## A Case Report of COVID-19 and Subacute Thyroiditis in Fars, Iran

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### Abstract

**Background & Objective:** COVID-19 can affect thyroid gland and causes subacute thyroiditis.

**Case Presentation:** We introduced a 60-year-old woman with an initial symptom of anterior cervical pain without any other constitutional symptoms. Nasopharyngeal and oropharyngeal swabs were detected positive for COVID-19 using RT-PCR assay. According to ultrasonographic, laboratory (lowered TSH, elevated CRP and ESR), and physical findings, subacute thyroiditis was found following the SARS-CoV-2 infection.

**Conclusion:** As subacute thyroiditis associated with SARS-Cov-2 may be represented without any fever, ruling out this infection in these patients is considerable.

**Keywords:** COVID-19, Subacute thyroiditis, Iran

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### Introduction

Subacute thyroiditis is a self-limited disease that commonly occurs with the direct toxicity of viruses, such as rubella, measles, coxsackie, adenovirus, etc., or following the inflammatory response against them (1-3).

Subacute thyroiditis is recognized in three subtypes. Even though the etiology of this disease appears to be different, the clinical manifestations are the same. The signs and symptoms of the disease include local symptoms (dysphagia, pain over the thyroid gland) and constitutional symptoms (often absent) (4). Coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2, is typically distinguished by a respiratory tract infection. However, multi-organ

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disorders are recognized following the infection, including cardiovascular dysfunction, urinary tract injury, neurological involvement, gastrointestinal complication, and endocrine system disorder, such as thyroiditis (5). COVID-19 can affect the thyroid gland through different mechanisms, such as binding to angiotensin-converting enzyme 2 (ACE2) receptor on thyroid follicular cells and autoimmune effects (5). The real prevalence of thyroiditis caused by COVID-19 is greatly unknown (6). This study introduced a case of subacute thyroiditis followed by the COVID-19 infection.

### **Case Presentation**

A 60-year-old woman with the only symptom of anterior cervical pain (neck pain), which lasted for two weeks, was referred to Valiasr Hospital located in Fasa University of Medical Sciences, Iran.

She did not have any past medical history. General and systemic examinations revealed unremarkable, except mild symmetrical enlargement and tenderness in palpation of the thyroid gland. The pain was limited to the thyroid. The ultrasonographic finding showed heterogeneous and hypoechoic inflammatory regions, suggesting thyroiditis. Her vital signs were shown in normal ranges, including body temperature of 37 °C, blood pressure of 120/70 mm Hg, heart rate of 76-bpm, respiratory rate of 12 breath/min, and SPO<sub>2</sub> of 98% in the air room. Free T<sub>3</sub> and T<sub>4</sub> thyroid hormone levels were in the normal range, while thyroid stimulating hormone (TSH) level was lower than the normal limit. Moreover, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) inflammatory markers were elevated, indicating an inflammatory response. Laboratory findings are presented in Table 1. Moreover, regarding the high prevalence of Coronavirus disease in our area, sampling from

the nasopharynx and oropharynx was performed, and real-time RT-PCR was then implemented using AmpliSens® kit (RIBO-Prep, Russia) for COVID-19 detection in laboratory unit.

Nasopharyngeal and oropharyngeal swab samples were recognized as positive for COVID-19 using real-time PCR. The patient was finally treated with prednisolone (15 mg/day) drug with a taper of 5 mg every two weeks. After this period, the resolution of the symptoms was observed, and the TSH level also returned to the standard limit.

### **Discussion**

Thyroiditis is classified into four types: infectious thyroiditis, autoimmune thyroiditis, Riedel's thyroiditis, and subacute thyroiditis (3). Among them, subacute thyroiditis has shown the highest association with viral infections (2). Various cases have been reported of the potential of SARS-CoV-2 to destroy the thyroid gland during or after the disease, leading to subacute thyroiditis (7-13). This study presented a woman with subacute thyroiditis along with COVID-19. According to previous reports, most cases were female (82%), the same as our study (2). She manifested neck pain and thyroid tenderness without any virus-like illness of Corona infection. The most clinical presentations of COVID-19-associated subacute thyroiditis described hyperthyroidism, tachycardia (47%), neck pain and tenderness (82%) (6). Besides, the other most frequent complaints have been reported as fever and cough during COVID-19 infection that were presented before SAT manifestations. While our case did not show any symptoms of fever or respiratory infection, which was in compliance with Sohrabpour et al. 2020 (14). Laboratory findings of the majority of reports indicated suppressed TSH, and high free T<sub>3</sub> and T<sub>4</sub> levels (8, 9, 11, 15, 16); nevertheless, our case considerably denoted suppressed TSH, and normal free T<sub>3</sub> and T<sub>4</sub> that was similar to Maris et al. 2020 (13). Since the effects of SARS-CoV-2 are extremely

**Table 1.** Clinical laboratory results

Measure (unit)	Result (normal range)	Measure (unit)	Result (normal range)
WBC (K/uL)	10.1 (4-11)	Cr (mg/dL)	0.8 (0.4-1.2)
RBC (M/ $\mu$ L)	5.02 (2.8-5.4)	BUN (mg/dL)	16 (7-12)
Hb (g/dL)	12.8 (11-16)	Na (mEq/L)	138 (136-145)
HCT (%)	40.5 (35-47)	K (mEq/L)	4.1 (3.5-5.5)
MCV (fl)	80.7 (80-95)	Calcium (mg/dL)	9.7 (8.3-10.5)
MCH (pg)	25.5 (27-32)	Phosphorus (mg/dL)	3.3 (3.2-5.6)
MCHC (g/dL)	31.6 (32-36)	T4 (ng/dL)	11.2 (4.4- 12)
Plt (K/uL)	407 (150-500)	T3 (ng/dL)	1.51 (0.7-1.99)
Lymphocyte (%)	15.8 (20-45)	TSH (CLIA)	0.051 (0.4-4.7)
Neutrophil (%)	77.1 (40-75)	CRP	39 ( $\leq$ 6)
Monocyte (%)	5.6 (1-8)	ESR (mm/h)	122 ( $\leq$ 20)
Basophil (%)	0.3 (0-3)		
Eosinophil (%)	1.2 (0-8)		

variable on thyroid function, there are various abnormalities in thyroid hormone tests (2). In this case, the inflammatory markers such as ESR and CRP were elevated, the same as in previous reports (9, 11, 14, 15, 17, 18). The most suggested mechanisms for the effect of COVID-19 on thyroid function include direct and indirect ways. In the direct mechanism, the most reliable evidence, the virus can damage the thyroid follicular cells through ACE2 and TMPRSS2 receptors, while inflammatory-immune responses against the virus are proposed for the indirect injury (5).

We concluded that subacute thyroiditis caused by COVID-19 might be limited to the thyroid gland region without any fever. Physicians should be attentive to screen subacute thyroiditis caused by COVID-19 disease through thyroid function tests and inflammatory markers.

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### **Conflict of interest**

The Authors declare no conflict of interest.

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