

## Fatal Case of Possible Thyroid Crisis Induced by SARS-CoV-2 Infection: A Case Report

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### Abstract

Thyroid crisis is an emergency due to impaired thyroid function caused by various conditions, particularly infections such as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that result in the dysfunction of various vital organs. We report a case of a 31-year-old Indonesian female with a 2-year history of hyperthyroidism with elevated thyroid-stimulating hormone (TSH) receptor antibodies (TRAb) who developed thyroid crisis possibly in association with SARS-CoV-2 pneumonia, sepsis, and disseminated intravascular coagulation (DIC). Prior to admission, she was treated for her hyperthyroidism with propylthiouracil and had been in stable remission for a year. She was admitted to the Emergency Room with complaints of watery stools, icteric sclerae, jaundice, coughing, and shortness of breath. The physical examination showed a World Health Organization (WHO) performance score of 4, delirium, blood pressure within normal limits, tachycardia, tachypnea, axillary temperature of 36.7°C, icteric sclerae, jaundice, and exophthalmos. There was a 3 cm palpable nodule on the right side of the neck. Auscultation of the lungs revealed bilateral pulmonary rales. Abdominal examination noted a palpable liver and enlarged spleen. Laboratory tests showed thrombocytopenia, electrolyte imbalance, hypoalbuminemia and elevated transaminases. The thyroid function tests showed a suppressed TSH level with an elevated free thyroxine (FT4) level. The SARS-CoV-2 polymerase chain reaction (PCR) swab test was positive. Initial patient management was with supportive therapy that included favipiravir and anti-hyperthyroidism medication; however, despite these interventions, her condition continued to deteriorate and she died after a few hours. This case demonstrates no difference in therapy between patients with thyroid crises and COVID-19 or other infections. Proper and timely treatment is important for reducing mortality rates.

**Key words:** COVID-19, SARS CoV-2, thyrotoxic crisis, thyroid storm, thyrotoxicosis

### INTRODUCTION

SARS-CoV-2 infection causes coronavirus disease 2019 (COVID-19) and pulmonary and systemic inflammation, leading to multi-organ dysfunction.<sup>1</sup> Mortality increases due to respiratory failure and other complications such as cardiovascular failure and DIC, and also due to the presence of comorbidities such as autoimmune diseases that alter thyroid function (e.g., Graves' disease [GD]) and COVID-19-associated thyroid disease, particularly with delayed diagnosis and management.<sup>2</sup> Thyroid crises can be triggered by events such as surgery, infection, inadequate antithyroid drug (ATD) therapy, thyroid surgery trauma, or uncontrolled diabetes mellitus (DM), with upper respiratory tract infection (e.g., SARS-CoV-2 infection) being the second-most common cause.<sup>3-6</sup> COVID-19 affects organs and organ systems, including the endocrine system, where the pituitary-thyroid axis is the direct or indirect target of SARS-CoV-2, which can cause

central hypothyroidism. Thyroid dysfunction can be found in SARS-CoV-2 infected patients where COVID-19 causes thyroid hormone imbalance in proportion to the degree of infection.<sup>1,7</sup>

### CASE

In July 2021, a 31-year-old Indonesian female with complaints of diarrhea of more than three times a day without mucus or blood was admitted to the emergency room. She reported fever and scleral icterus for the last three days. She had a history of hyperthyroidism since 2019 with elevated TRAb of 4.02 IU/L (reference value ≤1.75 IU/L), currently in stable remission after one year of propylthiouracil. There was no history of surgery. She reported occasional nonproductive cough and shortness of breath but denied chest pain. She had no history of COVID-19 vaccination or contact with COVID-19 patients. Physical examination showed a WHO performance score

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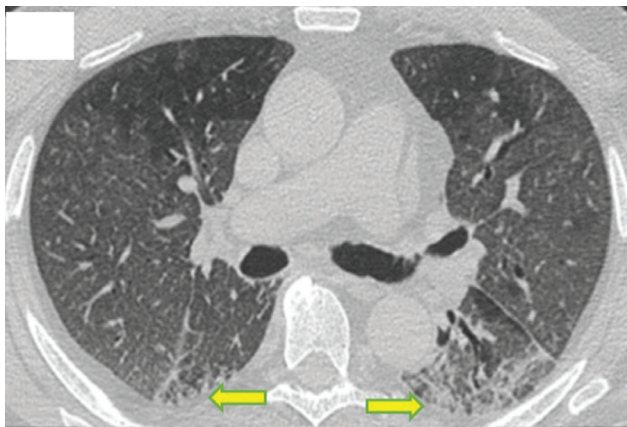
of 4, Glasgow coma score (GCS) of 11 (E3M5V3), blood pressure of 120/70 mmHg, heart rate of 132 beats/minute with a weak pulse, respiratory rate of 26 beats/minute, and axillary temperature of 36.7°C with delirium, icteric sclera, jaundice, and exophthalmos. Neck examination revealed a palpable right-sided nodule measuring 3 cm in diameter, fixed, mobile on swallowing, and nontender. There was no cervical lymphadenopathy. Lung auscultation revealed bronchovesicular breath sounds and bilateral pulmonary rales but no wheezing. Abdominal examination showed palpable liver and spleen enlargement. Examination found both extremities within normal limits.

The initial complete blood count and the basic metabolic panel are shown in Table 1. Thrombocyte, sodium, potassium, and albumin values were decreased. Transaminases were significantly elevated. TSH was 0.1 mIU/L (0.25–0.5 mIU/L), and FT4 was 7.26 ng/dL (0.93–1.71 ng/dL). The patient tested positive for the SARS-CoV-2 antigen, and six

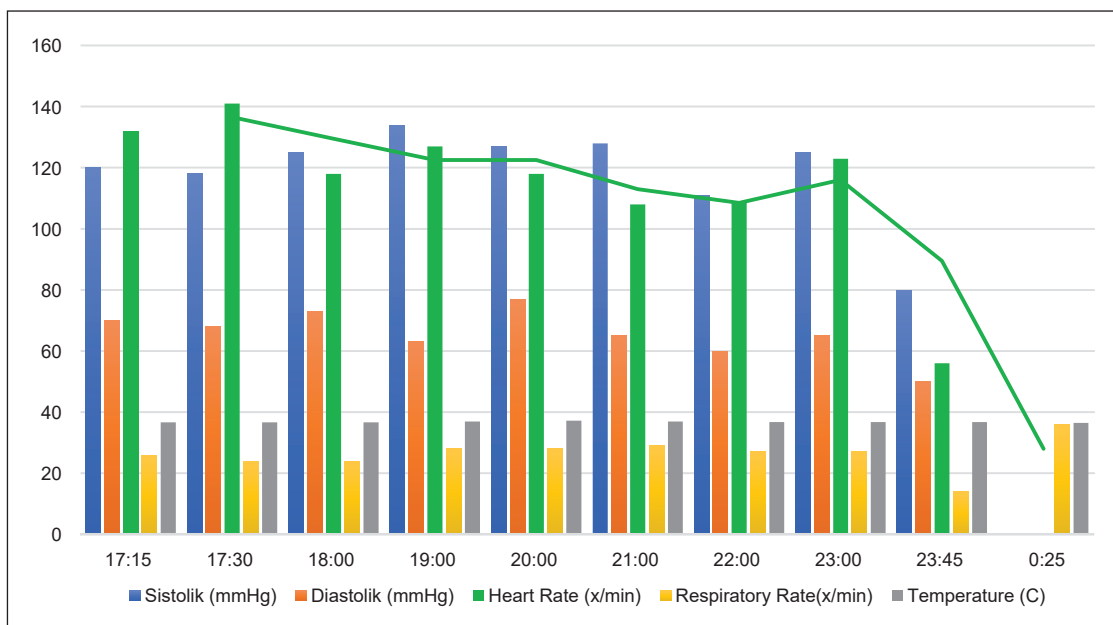
hours later, the SARS-CoV-2 PCR swab result was positive with a threshold cycle ( $C_T$ ) of 18.23 (reference  $C_T >38$ ). Electrocardiogram showed sinus tachycardia at 125 beats/minute. Chest X-ray (CXR) reported a thyroid nodule on the right cervical area and bilateral pneumonia. A multi-slice computed tomography (MSCT) scan of the chest showed ground glass opacity on the posterior dorsal lung (Figure 1).

The patient had a Burch-Wartofsky Point Scale (BWPS) score of 75 (Table 2) which fulfills the criteria of thyroid storm. By the Japan Thyroid Association (JTA) thyroid storm criteria (Table 3), patient has definite thyroid storm (TS1). Admitting impression was sepsis-induced thyroid crisis due to COVID-19 infection and DIC. Initial patient management included monitoring vital signs (Figure 2), electrolyte correction, oxygen at 10 L/min via non-rebreathing mask, fluid resuscitation with acetate Ringer’s solution at 30 cc/kg of body weight every 3 h, 40 mg of omeprazole administered intravenously every 12 h, 40 mg of propranolol administered nasogastrically every 6 h, 8 mg of dexamethasone administered intravenously every 24 h, 5 Lugol drops administered nasogastrically every 6 h, 60 mg of methimazole administered nasogastrically every 24 h, 2 mg of diazepam administered intravenously every 24 h, and the planned subcutaneous administration of 0.4 cc of enoxaparin every 24 h. Pulmonology service prescribed favipiravir.

Six hours after admission, the patient complained of worsening shortness of breath and became unconscious with a GCS of 9 (E2M5V2) and average blood pressure of 125/65 mmHg, heart rate of 123 beats/minute, temperature of 37°C, respiratory rate of 26 times/minute, 96% oxygen saturation, and ineffective breathing pattern. She was ideally for transfer to the Intensive Care Unit (ICU); however, the



**Figure 1.** An axial non-contrast MSCT of the thorax showing minimal ground-glass opacities on the posterodorsal lung (arrow).



**Figure 2.** Vital signs of patient throughout admission.

hospital did not have an ICU for patients with COVID-19, and its laboratory could not perform diagnostic PCR tests for SARS-CoV-2 infection, delaying patient management. The patient died a few hours after the start of treatment.

**Table 1.** Clinical laboratory results

Measure	Reference Interval	Result
Leukocytes (per $\mu$ L)	4,000–9,000	7,600
Absolute neutrophil count (%)	25.0–78.0	70.6
Absolute lymphocyte count (%)	17.0–57.0	16.3
Absolute monocyte count (%)	0.0–10.0	6.8
Absolute eosinophil count	0.0–10.0	3.3
Absolute basophil count	0.0–2.0	3.0
Erythrocytes (per $\mu$ L)	3,760,000–5,700,000	3,700,000
Platelet count (per $\mu$ L)	150,000–350,000	60,000
Hemoglobin (g/dL)	12.0–18.0	11.9
Hematocrit (%)	33.5–52.0	35.8
Sodium (mmol/L)	136–145	132.2
Potassium (mmol/L)	3.5–5.1	3.1
Chloride (mmol/L)	94–110	101
Urea (mg/dL)	6–4	15
Creatinine (mg/dL)	0.5–1.3	0.9
Albumin (g/dL)	3.7–5.3	3.1
Random blood glucose (mg/dL)	70–140	110
Alanine aminotransferase (U/L)	<41	200
Aspartate aminotransferase (U/L)	<37	392

**Table 2.** The patient’s Burch and Wartofsky Point Scale (BWPS)

Criteria	Points
<b>Thermoregulatory dysfunction</b>	
Temperature ( $^{\circ}$ F)	
99–99.9	5
100–100.9	10
101–101.9	15
102–102.9	20
103–103.9	25
>104	30
<b>Central nervous system effects</b>	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, or extreme lethargy)	20
Severe (seizure or coma)	30
<b>Gastrointestinal-hepatic dysfunction</b>	
Absent	0
Moderate (diarrhea, nausea/vomiting, or abdominal pain)	10
Severe (unexplained jaundice)	20
<b>Cardiovascular dysfunction</b>	
Tachycardia (beats/min)	
90–109	5
110–119	10
120–129	15
130–139	20
>140	25
Atrial fibrillation	10
<b>Congestive heart failure</b>	
Absent	0
Mild (pedal edema)	5
Moderate (bibasilar rales)	10
Severe (pulmonary edema)	15
<b>Precipitating history</b>	
Positive	0
Negative	10
<b>Total Score</b>	<b>75</b>
	<b>(Thyroid Storm)</b>
Criteria: $\geq$ 45, thyroid storm; 25–44, impending storm; <25, storm unlikely.	

**DISCUSSION**

In this case report, the patient was diagnosed with thyroid storm consistent with the BWPS for thyrotoxicosis and the 2016 JTA and Japanese Endocrine Society criteria for thyroid storm.<sup>4</sup> Thyroid crisis is a life-threatening condition often triggered by acute conditions or mental stress, developing into thyrotoxicosis and manifesting as multi-organ failure (MOF),<sup>8,9</sup> with a reported 10.7% mortality rate in Japan.<sup>9</sup> The thyroid crisis was triggered in this patient by SARS-CoV-2 infection in July 2021, at the peak of the second wave of COVID-19 infections in Indonesia.

Chen et al., studied 50 patients with confirmed COVID-19 infections, finding that 56% had abnormal TSH values, with low TSH associated with poor prognosis.<sup>10</sup> In Indonesia in July 2021, 88,659 of 3,287,727 patients with confirmed COVID-19 died.<sup>11</sup> The THYRCOV study by Lania et al., reported thyroid function changes in response to COVID-19 infection. Of their 287 patients, 31 had thyrotoxicosis (20.2%) and 15 had hypothyroidism (5.2%). Moreover, they found a significant relationship between increased interleukin 6 levels and decreased TSH, causing thyrotoxicosis, more severe systemic inflammation, and lower free-triiodothyronine (FT3) levels.<sup>12</sup>

The pathological mechanism of changes in thyroid hormones in patients with SARS-CoV-2 infection occurs via direct viral mechanisms and angiotensin-converting enzyme 2 (ACE2) in the pituitary gland, which indirectly impacts systemic effects by activating proinflammatory

**Table 3.** The Japan Thyroid Association thyroid storm (TS) diagnostic criteria<sup>4</sup>

<b>Prerequisite for diagnosis is presence of thyrotoxicosis with elevated levels of free triiodothyronine (FT3) or free thyroxine (FT4)</b>	
<b>Symptoms</b>	
1. Central nervous system (CNS) manifestations: Restlessness, delirium, mental aberration/psychosis, somnolence/lethargy, coma ( $\geq$ 1 on the Japan Coma Scale or $\leq$ 14 on the Glasgow Coma Scale)	
2. Fever: $\geq$ 38 $^{\circ}$ C	
3. Tachycardia: $\geq$ 130 beats/minute or heart rate $\geq$ 130 in atrial fibrillation	
4. Congestive heart failure (CHF): Pulmonary edema, moist rales over more than half the lung field, cardiogenic shock, or Class IV by the New York Heart Association or $\geq$ Class III in the Killip classification	
5. Gastrointestinal (GI)/hepatic manifestations: nausea, vomiting, diarrhea, or a total bilirubin level $\geq$ 3.0 mg/dL	
<b>Thyroid Storm Diagnosis Grade adapted from Akamizu, et al.<sup>9</sup></b>	
TS1 (First combination): Thyrotoxicosis and at least one CNS manifestation and fever, tachycardia, CHF, or GI/hepatic manifestation	
TS1 (Alternate combination): Thyrotoxicosis and at least three combinations of fever, tachycardia, CHF, or GI/hepatic manifestation	
TS2 (First combination): Thyrotoxicosis and a combination of two of the following: fever, tachycardia, CHF, or GI/hepatic manifestation	
TS2 (Alternate combination): Patients who met the diagnosis of TS1, except that serum FT3 or FT4 levels are unavailable	
<b>Exclusion and provisions</b>	
Cases are excluded if other underlying diseases clearly cause any of the following symptoms: fever (e.g., pneumonia and malignant hyperthermia), impaired consciousness (e.g., psychiatric disorders and cerebrovascular disease), CHF (e.g., acute myocardial infarction), and liver disorders (e.g., viral hepatitis and acute liver failure). Therefore, when it is difficult to determine whether the symptom is caused by TS or is simply a manifestation of an underlying disease, it should be regarded as being due to a TS caused by these precipitating factors. Clinical judgment in this matter is required.	
TS1, "Definite" TS; TS2, "Suspected" TS.	

cytokines causing hyperinflammation and human leukocyte antigens in the autoimmunity response.<sup>2,10</sup> Hyperinflammatory syndrome is caused by SARS-CoV-2 infection, which increases proinflammatory cytokines that can cause patients to enter into a septic condition.

COVID-19 patients have hepatocellular damage caused by SARS-CoV-2 infection via ACE2 receptor expression. ACE2 expression in cholangiocyte cells causes cholestasis and cytokine storm conditions, increasing liver damage and hepatobiliary complications.<sup>13</sup> While acute liver failure in thyroid storm is associated with increased FT3 due to mitochondrial apoptosis in hepatocytes, the pathologic mechanism for thyrotoxicosis is secondary ischemic hepatitis due to peripheral vasodilation and excessive thyroid hormone release due to acute heart failure.<sup>14,15</sup> In this patient, diarrhea and jaundice were manifestations of a thyroid crisis.

Thyroid crisis triggered by SARS-CoV-2 infection causes coagulopathy due to increased pulmonary platelet consumption, intravascular coagulation, and microangiopathic thrombosis. Coagulopathy is caused by systemic inflammation and SARS-CoV-2-specific mechanisms that cause endothelial dysfunction.<sup>7</sup> When clinicians are aware of coagulation disorders in patients with SARS-CoV-2 infection, they should perform D-dimer, prothrombin time, and fibrinogen tests to assess their DIC scores.<sup>16-18</sup> DIC events can be triggered by thrombocytopenia in patients with thyroid crisis and SARS-CoV-2 infection and, in turn, increase the risk of venous thromboembolism (e.g., pulmonary embolism and cerebral venous thrombosis) and stroke due to arterial thrombosis and embolism that causes atrial fibrillation.<sup>15</sup> Low molecular weight heparin (LWMH) can be given as thromboprophylaxis, particularly in severe and critically ill patients. It has anticoagulant properties but can also limit viral entry into cells by interacting with the SARS-CoV-2 spike protein, decreasing heparinase activity, preventing plasma leakage, and neutralizing cytokines via other biological activities.<sup>7</sup> It is preferable to look for contraindications and measure bleeding and venous thromboembolism risk using the IMPROVE and PADUA scores when providing anticoagulants as thromboprophylaxis.<sup>19,20</sup> In this patient, the IMPROVE score was 2.5 (<7 indicates a low bleeding risk), and the PADUA score was 0 (<4 indicates a low venous thromboembolism risk). Therefore, we planned to administer 0.4cc/subcutaneous enoxaparin.

The Brazilian Society of Endocrinology and Metabolism and the THYRCOV study reported that thyroid dysfunction management during the COVID-19 pandemic was the same in patients with thyroid storm and co-infection with SARS-CoV-2 or other pathogens. They found that 16% of patients with SARS-CoV-2 infection treated for thyrotoxicosis experienced a two-fold greater thromboembolic event, an increased incidence of atrial fibrillation with suppressed TSH, and a higher mortality rate.<sup>12</sup>

The treatment of hyperthyroidism caused by GD in the current phase of the COVID-19 pandemic can be divided into two scenarios:<sup>21</sup> (1) treatment of patients with a prior diagnosis of GD and on regular treatment with anti-thyroid drugs (ATDs); (2) treatment of patients with recently diagnosed GD who have not yet started therapy.

Our patient belongs to the first scenario. At the current stage of the COVID-19 pandemic, when face-to-face consultations can be difficult, it is especially important not to interrupt ATD treatment since any relapse would require an urgent medical appointment and increase the risk of complications (e.g., thyroid storm), which can be triggered by infections such as COVID-19. In general, ATD treatment is maintained for 12–24 months, after which the medication can be suspended. Alternatively, prolonged use of low-dose ATDs may be considered since it is safe and may increase the chance of Graves' Disease remission. During the COVID-19 pandemic, telemedicine may be an alternative to manage patients with hyperthyroidism, and those with thyroid storm should be treated in infection centers with ICUs.

In the second scenario (patients with a recent GD diagnosis), ATDs should be the first therapeutic option due to possible current restrictions on nuclear medicine or surgical treatment. Surgical treatment should be performed in the rare circumstance of a patient not responding satisfactorily to ATDs, developing severe side effects to ATDs, or being unable to undergo radioiodine therapy.<sup>21</sup>

In our patient, we provided supportive therapy by providing oxygen, installing a heart monitor, nasogastric tube, and urinary catheter, and giving crystalloid fluid up to 28 drops/minute. We planned to transfer the patient to the ICU when she was started to develop hemodynamic instability, had DIC and Multiorgan failure (MOF) and an APACHE score >9 (based on 2016 JTA criteria) but the hospital where she was admitted in had no ICU for patients with COVID-19.

Our case report highlights the importance of educating patients with thyroid disorders during the COVID-19 pandemic. Patients with GD should not discontinue their therapy because doing so can increase the risk of infection, including SARS-CoV-2 infection. Hospitals should also provide infection center rooms for patients suspected of being infected with COVID-19 without waiting for PCR test results.<sup>22</sup>

## CONCLUSION

We have reported a fatal case of possible thyroid crisis induced by SARS-CoV-2 infection, which directly or indirectly destroys the thyroid follicles, triggering a thyroid crisis. Prompt and appropriate management and treatment can reduce the incidence of multi-organ failure and mortality due to thyroid crisis induced by SARS-CoV-2 infection. There is no difference in treating thyroid crises before and during a COVID-19 pandemic. Maintaining a



euthyroid state is very important to prevent the relapse of thyroid disease.

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#### Ethical Consideration

Patient consent was obtained from the relative before submission of the manuscript.

#### Statement of Authorship

The authors certified fulfilment of ICMJE authorship criteria.

#### Author Contribution Statement

FH conceived the idea, validated research outputs, conducted investigation, provided study materials, curated the data, wrote the original draft preparation, reviewed and edited the manuscript, prepared data presentation, coordinated research activity planning, acquired financial support.

AMA conceived the idea, validated research outputs, conducted investigation, provided study materials, wrote the original draft preparation, reviewed and edited the manuscript, supervised the research activity planning.

HI conceived the idea, validated research outputs, conducted investigation, provided study materials, curated the data, wrote the original draft preparation, reviewed and edited the manuscript.

#### Author Disclosure

The authors declared no conflict of interest.

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