

Subclinical Thyroiditis Related to COVID-19: A Case Report and Literature Review

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1. Abstract

COVID-19 can cause different clinical manifestation by affecting different organs in the body. Involvement of thyroid gland is very rare in COVID-19 infection than any other systems of the body. We described a 41-year-old female with subclinical thyroiditis most likely related to corona virus infection and reviewed literatures regarding cases of thyroiditis due to COVID-19 have been reported. The objective of this article is to create awareness regarding this novel entity and the association with thyroid dysfunction.

2. Introduction

The coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), which is responsible for the disease COVID-19 (coronavirus disease 2019). While SARS-CoV-2 is known to cause substantial pulmonary disease, including pneumonia and acute respiratory distress syndrome (ARDS), clinicians have observed many extrapulmonary manifestations of COVID-19. Our clinical experience and the emerging literature suggest that the hematologic, cardiovascular, renal, gastrointestinal and hepatobiliary, endocrinologic, neurologic, ophthalmologic, and dermatologic systems can all be affected [1]. We present a case of subclinical thyroiditis in COVID-19 infected patient.

3. Case Presentation

A 41-year-old female with past medical history of bipolar disorder was brought in by emergency medical service (EMS) for bizarre behavior and agitation. EMS witnessed her screaming and crawling on the floor. On examination, she was disheveled, hy-

perverbal, disorganized and irrational. She initially was admitted to the psychiatric emergency department and found her PCR test for COVID-19 was positive. For this reason, she was transferred to inpatient medicine unit. She reported experiencing dry cough, shortness of breath and generalized weakness for 2 weeks prior to the admission. She mentioned experiencing weight loss, denied neck pain, difficulty swallowing, palpitations, heat/cold intolerance, sleep disturbances, constipation, diarrhea, diaphoresis, appetite changes, fever, chills, chest pain, dizziness, nausea, vomiting, abdominal pain, suicidal/homicidal thoughts, hallucinations or delusions. She had contacted with COVID-19 positive relative. Prior to this episode her bipolar disorder was well controlled on Vrylar (cariprazine). She had no history of treatment with lithium. In addition, she had no family history of thyroid disease. Her physical examination was unremarkable, there was no thyromegaly or tenderness on thyroid gland palpation.

4. Discussion

Subacute thyroiditis is a self-limited inflammatory medical condition. It is usually caused by viral infection [2]. The natural course of this disease involves three phases- initial hyperthyroidism followed by a period of hypothyroidism and eventually return to euthyroid state. It resolves without intervention within a few weeks. However, most cases do not follow all phases of the disease. Sometimes patient may remain asymptomatic or exhibit mild symptoms. Diagnosis is based on clinical and laboratory data. Tissue diagnosis is rarely needed [4, 5]. In this particular case, the patient was asymptomatic, that's why subclinical thyroiditis

was entertained. Review of literature shows that evidence for viral infection in subacute thyroiditis was linked to many viruses such as mumps virus, coxsackievirus, adenovirus, Epstein-Barr virus, rubella and cytomegalovirus, though a specific viral cause is not always found [6]. The key mechanism of SARS-CoV-2 infection is the virus invades into the host cell through the angiotensin converting enzyme-2 (ACE2) receptor. More recent studies based on SARS-CoV-2 in 2020 have shown that ACE2 expression levels were highest on thyroid follicular cells among other organs, which gives insight of a plausible mechanism for pathophysiology of thyroiditis in COVID-19 infection [7]. Unfortunately, based on our knowledge, no studies have found yet which demonstrate the presence of corona virus in the thyroid follicular cell. We reviewed various literatures highlighting subacute thyroiditis related to COVID-19 virus. Those published case reports demonstrated typical clinical features including neck pain in the setting of subacute thyroiditis, generalized weakness and palpitations in addition to lab findings significant for low TSH, with elevated Free T4 and T3 [8, 9]. Our patient's lab results (low TSH level and normal free T4) were consistent with subclinical thyroiditis. Because the patient had no clinical symptoms of subacute thyroiditis.

Laboratory data on Admission:

| Variable | Patient's result | Reference ranges |
|--|------------------|------------------|
| Thyrotropin (TSH) μ U per milliliter | 0.07 | 0.45-5.33 |
| Triiodothyronine (T3) pg/ml | 84 | 2.20-4.10 |
| Free thyroxine (FT4) (ng/DL) | 1.7 | 0.58-1.64 |
| White-cell count (per μ l) | 6600 | 4000–11,000 |
| Ferritin (μ g/liter) | 254 | 11-300 |
| Erythrocyte sedimentation rate (mm/hr) | 38 | 0-22 |
| C-reactive protein (mg/liter) | 5.9 | <8 |
| Aspartate aminotransferase (U/liter) | 66 | 10-40 |
| Alanine aminotransferase (U/liter) | 88 | 10–50 |
| SARS-CoV-2 (PCR) | positive | negative |
| PCR for RSV, Influenza A and Influenza B | negative | negative |

Imaging study result:

Chest x-ray was significant for bilateral lung opacity.

Thyroid Function test results:

| Variable | At the time of admission | 3 month later | Reference ranges |
|----------------|--------------------------|---------------|------------------|
| TSH uL/mL | 0.07 c | 1.13 | 0.45-5.33 |
| Free T4 ng/DL | 1.7 | 1.6 | 0.58-1.64 |
| Total T3 pg/ml | 84 | 104 | 100-200 |

5. Conclusion

This case illustrates the potential for subclinical thyroiditis as a result of COVID-19 infection. Due to current COVID-19 pandemic, we suggest to rule out possibility of SARS-Cov-2 infection in patient presenting with clinical and or biochemical features of hyperthyroidism or subclinical hyperthyroidism.

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