LETTER TO THE EDITOR

SUBACUTE THYROIDITIS AFTER COVID-19 INFECTION

Bhagwan Das[1], Tehseen Fatima[2]
[1]Cancer Foundation Hospital, Karachi-Pakistan
[2] Hussain Lakhani Hospital, Iqra University, Karachi-Pakistan

Dear Editor,

COVID-19 infection caused by SARS-CoV2 was initially thought to be causing pneumonia and other pulmonary complications only, proved wrong as during this pandemic the world is witnessing its other extrapulmonary manifestations including endocrine and thyroid dysfunctions.[1]

Subacute thyroiditis (SAT) is a self-limiting inflammation of the thyroid gland secondary to viral infection of the upper respiratory tract.[2] Clinically it is characterised by fever, neck pain, and deranged thyroid profile. Though SAT historically reported as of post-viral origin, is a quite uncommon extrapulmonary manifestation of COVID-19.[3,4]

Recently, we have seen a 33-year-old male patient presented with a history of fever, neck pain, dysphagia, and palpitations for few days. On history taking, he said he was diagnosed with COVID-19 infection based on his symptoms and positive COVID PCR, in the preceding week and was managed conservatively at home. On examination, he had a fever of 100 °F with a pulse of 114 b/min and mild tenderness on the neck examination. On laboratory investigations, his COVID IgG antibody was positive, ESR of 80, his thyroid function tests showed suppressed TSH, < 0.001 uIU/ml (normal limit 0.4–4.2) and high FT4 level, 3.84 ng/dl (normal limit 0.89–1.76). His TC^{99}-thyroid scan was ordered, which showed negligible uptake in both thyroid lobes, suggestive of inflammation of the thyroid gland. Based on history, examination, lab investigations, and thyroid scan report, he was diagnosed with SAT and was started on flurbiprofen and propranolol. His symptoms did not improve and then was started tapering doses of prednisolone. After few days of starting steroids, his symptoms resolved and his medicines were tapered, and repeat FT4 after 3 weeks was 1.64 ng/dl and at six weeks it was 1.22 ng/dl with a repeat TSH of 3.39 uIU/ml.

The exact mechanism behind SAT and COVID-19 infection has not been duly studied yet. Different hypotheses have been postulated behind this manifestation, such as viral entry into the thyroid gland thorough Angiotensin-converting enzyme 2 receptors expression leading to direct viral replication in thyroid follicles, inflammatory infiltration, local apoptosis, and cellular damage.[4,5]

Our intent in sending this communication is to create awareness amongst clinicians regarding this additional extrapulmonary manifestation during this pandemic of COVID-19 infection. As early recognition and timely management with anti-inflammatory and steroids leads to the successful resolution of signs and symptoms and thus avoiding unnecessary costly workup.

REFERENCES

1. Lundholm MD, Poku C, Emanuele N, Emanuele MA, Lopez N. SARS-CoV-2 (COVID-19) and the Endocrine System. J Endocr Soc. 2020;4(11).
2. Desailloud R, Hober D. Viruses and thyroiditis: an update. Virol J. 2009;6:5.
3. Brancatella A, Ricci D, Cappellani D, Viola N, Sgrò D, Santini F, *et al*. Is Subacute Thyroiditis an Underestimated Manifestation of SARS-CoV-2 Infection? Insights From a Case Series. J Clin Endocrinol Metab. 2020;105(10):e3742-e6.
4. Khatri A, Charlap E, Kim A. Subacute Thyroiditis from COVID-19 Infection: A Case Report and Review of Literature. Eur Thyroid J. 2020.
5. Li MY, Li L, Zhang Y, Wang XS. Expression of the SARS-CoV-2 cell receptor gene ACE2 in a wide variety of human tissues. Infect Dis Poverty. 2020;9(1):45.

Citation: Das B, Fatima T. Subacute Thyroiditis after COVID-19 infection. J Ayub Med Coll Abbottabad 2021;33(4):701.

Address for Correspondence:
Bhagwan Das, Cancer Foundation Hospital, Karachi-Pakistan
Email: dr_bhagwandas@yahoo.com