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Case Report

COVID - 19 Associated Subacute Thyroiditis in Albania

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Case Report

Subacute Thyroidis (SAT) is a self-limiting inflammatory disorder of the thyroid gland [1]. It is also thyroid disease of viral or post viral origin [2]. SAT is typically characterized by triphazic clinical course of thyrotoxicosis, hypothyroidism and return to normal thyroid function [3]. In the initial phase, many patients also present clinical and/ or biochemical manifestation. High levels of inflammatory markers such as C -reactive protein and more specifically erythrocyte sedimentation rate represent the most frequent biochemical finding at presentation [4]. Several respiratory viruses including coxackievirus, Epstein - Barr virus, cytomegalovirus, influenza virus was reported to be associated with SAT development [1]. At the present, it is unclear whether follicle damage in SAT is cause by direct viral infection or by the host immunological response to the viral infection. With the beginning of the coronavirus disease 2019 pandemic caused by severe acute respiratory syndrome coonavirus 2 a thyroid impact was considered due to the potencial of SARS -COV-2 to cause multiorgan effects [5.6]. A few cases of SAT after SARS - COV-2 infection was reported in the literature. Here I report a case of SAT which diagnosed four months after SARS-COV-2 infection [7].

A nineteen -year -old female with no history of concomitant disease. She had contact history (November 2021) with her father and her brother who had a positive nasopharingeal swab test for SARS-COV-2 RNA. She was asymptomatic and her test was negative. After three weeks she developed fatigue, muscle crumps, fever, temperature 39.5. Paracetamol and ibuprofen had a little antipyretic effect. Two days later these examinations were performed:
CRP 136 mg/L (< 5 mg/L)
Ferritine 72.8 (20 - 200 ng/ml)
Sedimentation 78 mm/h
Kidney function and liver enzymes were normal
On chest radiography was found right lobe pneumonia. The treatment included ceftriaxone, Vitamin D, multivitamin. After seven days from the bigining of symptoms, her COVID-19 IgG serology was positive 1750 (Negative < 8 RU/mL, Positive > 11 RU/mL). Our patient showed significant improvement on three days. CRP level fell to 13.8 mg/L, temperature 37.3. Symtoms started to resolve and in one-week inflammatory markers became normal. During this period our patient fell to a weight of 10 kg. Month after month tachycardia was present. These clinical findings suggested SAT. I consulted her and recommenced (February 2022) to check up the thyroid gland. One reason was because her father had history of Hashimoto Hypothyroidsm. Also, he was diagnosed with Hashimoto Hypothytoidism. (TSH 57 µU/mL and Anti-TPO 1080 IU/ml) after H1N1 infection ten years ago. Four months after the SARS-COV-2 infection, electrocardiogram demostrated sinus tachycardia.

The thyroid panel of our patient showed:
TSH 0.02 (0.25 – 5.0 µU/mL)
FT3 5.12 (4.0 – 8.3 pmol/L)
FT4 13.3 (10.6 – 19.4 pmol/L)
Anti - TPO 2.3 (< 80 IU/ml)
Anti-TG 1.8 (< 80 IU/ml)
TRAb 0.8 (<1.22 IU/L)
Other examinations were:
PCR 22 mg/L
ERS 48 mm/h
The treatment included: propranolol, prednisolone 5mg (1 - 1-1 tb for 2 weeks, 1 - 0 - 1 tb for 1 week, 1 - 0 - 0 tb for 1 week) for two months.
One month later TSH go to 0.15. Our patient referred that has gained weight (3 kg).
After she finished the treatment with prednisolone, the thyroid panel resulted:
TSH 0.2 µU/mL
TRAb 0.16 IU/L
She was advised to receive follow-up with thyroid function test every four weeks for a minimum of four months.

Discussion

SAT is thyroiditis, which usually occurs due to a viral infection in genetically predisposed patients [8]. The etiopathogenesis of SAT is not known but usually developed after a viral upper respiratory tract infection [7]. Different studies have found its associations with echovirus, mumps, influenza, cytomegalovirus, rubella, Epstein Barr, enterovirus, adenovirus, and other viruses [6,9,10]. SAT' clinical symptoms emerged approximately 21±11 days after COVID-19 infection [11]. In our case the patient was 19-year-old female and SAT clinical symptoms emerged three weeks after COVID-19 infection. Our patient had fatigue, muscle crumps, painful, tender thyroid gland, aritmia. It was the father' history that our patient to be suspected for thyroid diseases to much later. Laoratory findings were increased CRP and ESR, low TSH and focal hypoechoic areas of

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thyroid gland and the aritmia were the main reasons for SAT to be considered the final diagnosis after SARS -COV-2 infection. In the review evaluating SAT cases, 72.7% of patients had mild COVID-19 symptoms, 13.64% had an asymptomatic disease, while 13.64% developed pneumonia [11]. However, our case supports the hypothesis that SAT may occur after a mild COVID-19 episode (right lobe pneumonia). The pathogenesis and etiology of SAT remain unclear. But the most common thought is that this disease is due to a viral etiology or post-viral inflammatory reaction which is diagnosed in individuals with a genetic predisposition [6.12].

In conclusion, many different extra-pulmonary involvements may occur following COVID-19 disease. Is fact that SAT may develop as one of the little-known and rare extra-pulmonary involvements of COVID-19. Any symptoms associated with thyroid gland should be considered. We have to draw attention because after COVID-19 infection may develop SAT, whose diagnosis can be missed.

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