Tidsskriftet Tidsskrift for Den norske legeforening

Subacute thyroiditis after COVID-19

SHORT CASE REPORT

EIRIK TJØNNFJORD

E-mail: eirik.tjonnfjord@so-hf.no
Thrombosis Clinic
Østfold Hospital Kalnes
and
Department of Haematology
Oslo University Hospital
and
Institute of Clinical Medicine
University of Oslo
Eirik Tjønnfjord, acting senior consultant in haematology.
The author has completed the ICMJE form and declares no conflicts of interest.

RAGNAR BEKKHUS MOE

Department of Endocrinology
Østfold Hospital Kalnes
Ragnar Bekkhus Moe, senior consultant.
The author has completed the ICMJE form and declares no conflicts of interest.

WALEED GHANIMA

Østfold Hospital Kalnes
and
Department of Haematology
Oslo University Hospital
and
Institute of Clinical Medicine
University of Oslo
Waleed Ghanima, PhD, principal investigator and professor.
The author has completed the ICMJE form and declares no conflicts of interest.

SAAD ABALLI

Department of Infectious Diseases
Østfold Hospital Kalnes
Saad Aballi, senior consultant with overall responsibility for the management of COVID-19 patients.
The author has completed the ICMJE form and declares no conflicts of interest.

BACKGROUND

Since the beginning of the pandemic we have learned much about acute organ complications due to COVID-19, but we are still only beginning to understand the post-infection complications.

CASE PRESENTATION

A man in his forties was diagnosed with subacute thyroiditis after a mild COVID-19 infection. This is an important differential diagnosis to consider if after a period of improvement, an infected patient develops fever, pain around the region of the thyroid (throat/neck) and/or symptoms of hyperthyroidism.

INTERPRETATION

Subacute thyroiditis is thought to be initiated by a viral infection or postviral inflammatory process, often in patients with a history of an upper respiratory infection typically two to eight weeks prior to the onset of thyroiditis. The condition is believed to be triggered by an antigen created by the virus. Subacute thyroiditis must be on the list of possible differential diagnoses in patients with COVID-19 whose condition deteriorates after a period of improvement.

We now have a good understanding of the acute complications of COVID-19, but we know less about possible long-term sequelae. The aim of this case report is to raise awareness of subacute thyroiditis as a potential complication of COVID-19.

A previously healthy man in his forties who used no regular medications was diagnosed with SARS-CoV-2 infection after a positive polymerase chain reaction (PCR) test on a nasopharyngeal swab. The infection caused mild symptoms, with a few days of fever and cough, but required no specific treatment. The man then had three weeks of good health, before gradually developing reduced general condition with fever (38.3 °C), dysphagia, headache, a mild dry cough, dyspnoea and general myalgia. Paracetamol and ibuprofen had only a brief antipyretic effect. In the out-of-hours primary care centre, he was found to have an elevated C-reactive protein (CRP) level of

88 mg/L (reference range <5), but tested negative for COVID-19. He was admitted to hospital for assessment just under six weeks after first testing positive for SARS-CoV-2.

Upon arrival at hospital, the patient was breathing normally, but was subfebrile with reduced general condition. He had a regular heart rate of 95 beats/min, blood pressure 120/80 mmHg, temperature 37.7 °C, respiratory rate 18/min and oxygen saturation of 95 %. Diffuse tenderness to palpation was noted on both sides of the throat, but there was no enlargement of the thyroid. Clinical examination was otherwise unremarkable, including a normal pharynx and no tenderness over the temples. Blood tests showed a sedimentation rate of 92 mm/hr (<10), CRP 86 (<6) mg/L, and leukocytes 11.9 × 10^9/L (3.5–11), but otherwise normal electrolytes, kidney function, liver enzymes, D-dimer, ferritin and procalcitonin.

During his first 72 hours of observation on the ward, he developed recurrent fever with a highest measured temperature of 38.9 °C. He also complained of a sore throat and myalgia. His heart rate was persistently slightly elevated (90–100/min), but his respiratory rate was normal and his peripheral oxygen saturation (SpO_2) was 95–98 % without supplemental oxygen.

All microbiological tests were negative, including another PCR test for COVID-19, a respiratory pathogen panel, and blood cultures. Serological testing suggested previous Epstein-Barr and cytomegalovirus infections, but results were negative for Hepatitis B and C. Chest X-ray and CT sinuses were also normal.

On the patient's second day in hospital, we began to suspect subacute thyroiditis and therefore ordered a metabolic panel. This showed thyroid-stimulating hormone (TSH) 0.01 mIU/L (0.35–3.6), FT4 (tetraiodothyronine) 27.8 pmol/L (9–19), FT3 (triiodothyronine) 7.5 pmol/L (2.6–5.7), thyroperoxidase (P-TPO) <3 (<6) and TSH receptor antibodies (TRAb) <0.9 (<1.8). The biochemical findings were thus consistent with mild hyperthyroidism, but not autoimmune thyroiditis as TPO and TRAb were both negative. An ultrasound of the neck showed slight diffuse enlargement of the thyroid gland with discrete surrounding oedema and heterogeneous parenchyma, consistent with thyroiditis. There was no evidence to suggest a thyroid abscess.

While the ultrasound findings were consistent with thyroiditis, areas of mild hyperaemia were also described. In cases of subacute thyroiditis, vascularity in the gland is typically low to normal. On the basis of the hyperaemia, plus the persistent symptoms of fever, myalgia and sore throat as well as the elevated CRP and sedimentation rate, other diagnoses such as temporal arteritis and polymyalgia rheumatica were also considered. However, the fact that the patient was under 50 years of age made these diagnoses less likely, and treatment was therefore initiated for subacute thyroiditis (1). Non-steroidal anti-inflammatory drugs had had little to no effect when used for several weeks prior to admission, and it was therefore decided to begin peroral prednisolone at a moderate dose of 20 mg × 1 (2). A response to glucocorticoid therapy is typically expected after one to three days. Our patient showed significant

improvement on day two, when his CRP level fell from 92 to 35 mg/L. A detailed plan was outlined in the discharge summary for tapering prednisolone over the course of a month.

At a follow-up appointment two months later, the patient was symptom-free with normal thyroid function and negative results in tests for infectious diseases. He was not taking any medications and his general health was good. He was advised to receive further follow-up by his general practitioner, with thyroid function tests every four weeks for a minimum of four months. An additional thyroid ultrasound was considered unnecessary.

Discussion

The aim of this case report is to raise awareness of subacute thyroiditis as a possible complication that can arise during or after COVID-19 infection. While it was not possible to establish a definite causal relationship between COVID-19 and subacute thyroiditis in this patient, we consider such a relationship to be likely.

Subacute thyroiditis is thought to be caused by viral infections or to occur as part of a post-viral inflammatory process. Affected individuals have often had symptoms of upper respiratory tract infection two to eight weeks previously. Clusters of cases have been reported in association with coxsackievirus, adenovirus and other viral infections (3, 4). The condition is not thought to be linked to autoimmune inflammation, but a strong association with HLA-B35 has been found in several ethnic groups (3, 4). The condition is thought to occur when a viral infection leads to the production of an antigen, either of viral origin or as a result of tissue damage caused by the viral infection, which then binds to HLA-B35 on macrophages (5, 6). The antigen-HLA-B35 complex activates cytotoxic T lymphocytes that attack thyroid follicular cells because they have structural similarity to the antigen (7). The inflammatory process in the thyroid gland causes the release of stored thyroid hormone, leading to hyperthyroidism.

The course of subacute thyroiditis is typically initial hyperthyroidism, followed by brief euthyroidism and then finally hypothyroidism before recovery and the restoration of normal metabolism (2). Each phase usually lasts two to eight weeks, but there is extensive variation. The hyperthyroid phase lasts until reserves of thyroid hormone have been depleted. As the inflammation in the thyroid gland subsides, follicular cells will gradually recover and begin to produce thyroxine again. Thyroid hormone replacement therapy may be required prior to full recovery of the follicular cells. On recovery, thyroid function is anticipated to return to normal, and any hormone replacement therapy can then be tapered and discontinued.

To the best of our knowledge, this is the first case report of subacute thyroiditis following COVID-19 infection in Norway; however, several cases have been described internationally (8–10). Subacute thyroiditis was considered highly

likely as a diagnosis owing to the typical medical history and symptoms, and the results of blood tests and the supplementary thyroid ultrasound. Medium-dose glucocorticoid therapy also proved highly effective, as would be expected.

Previous reports have indicated that subacute thyroiditis can occur either during or – more commonly – after COVID-19 infection (8, 10). Studies have shown that the thyroid gland expresses angiotensin-converting enzyme 2 receptors (11), which are essential for enabling SARS-CoV-2 to infect human cells (9). We were unable to find any detailed descriptions in the literature of how SARS-CoV-2 infection leads to T-cell-mediated thyroid gland damage, but it is reasonable to assume that the pathophysiology is similar to that seen in other viral infections.

We are likely to see more cases of subacute thyroiditis in individuals who have, or have had, COVID-19. It is therefore important to consider this complication when a person who has previously been diagnosed with COVID-19 develops signs and symptoms of hyperthyroidism as well as fever and/or neck/throat pain. Subacute thyroiditis may have non-specific symptoms, which can increase the risk of underdiagnosis. We recommend liberal use of thyroid function tests in patients with new symptoms following initial improvement after COVID-19.

The patient has consented to the publication of this article.

The article has been peer-reviewed.

LITERATURE

1. Ross DS, Burch HB, Cooper DS et al. 2016 American Thyroid Association Guidelines for diagnosis and management of hypertyroidism and other causes of thyrotoxicosis. Thyroid 2016; 26: 1343–421. [PubMed][CrossRef]

2. Benbassat CA, Olchovsky D, Tsvetov G et al. Subacute thyroiditis: clinical characteristics and treatment outcome in fifty-six consecutive patients diagnosed between 1999 and 2005. J Endocrinol Invest 2007; 30: 631–5. [PubMed][CrossRef]

3. de Bruin TW, Riekhoff FP, de Boer JJ. An outbreak of thyrotoxicosis due to atypical subacute thyroiditis. J Clin Endocrinol Metab 1990; 70: 396–402. [PubMed][CrossRef]

4. Martino E, Buratti L, Bartalena L et al. High prevalence of subacute thyroiditis during summer season in Italy. J Endocrinol Invest 1987; 10: 321–3. [PubMed][CrossRef]

5. Desailloud R, Hober D. Viruses and thyroiditis: an update. Virol J 2009; 6: 5. [PubMed][CrossRef]

6. Nyulassy S, Hnilica P, Buc M et al. Subacute (de Quervain's) thyroiditis: association with HLA-Bw35 antigen and abnormalities of the complement system, immunoglobulins and other serum proteins. J Clin Endocrinol Metab 1977; 45: 270–4. [PubMed][CrossRef]

7. Kojima M, Nakamura S, Oyama T et al. Cellular composition of subacute thyroiditis. an immunohistochemical study of six cases. Pathol Res Pract 2002; 198: 833–7. [PubMed][CrossRef]

8. Chong WH, Shkolnik B, Saha B et al. Subacute thyroiditis in the setting of coronavirus disease 2019. Am J Med Sci 2021; 361: 400–2. [PubMed] [CrossRef]

9. Muller I, Cannavaro D, Dazzi D et al. SARS-CoV-2-related atypical thyroiditis. Lancet Diabetes Endocrinol 2020; 8: 739–41. [PubMed] [CrossRef]

10. Mattar SAM, Koh SJQ, Rama Chandran S et al. Subacute thyroiditis associated with COVID-19. BMJ Case Rep 2020; 13: e237336. [PubMed] [CrossRef]

11. Rotondi M, Coperchini F, Ricci G et al. Detection of SARS-COV-2 receptor ACE-2 mRNA in thyroid cells: a clue for COVID-19-related subacute thyroiditis. J Endocrinol Invest 2021; 44: 1085–90. [PubMed][CrossRef]

Publisert: 28 June 2021. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.21.0169
Received 1.3.2021, first revision submitted 13.3.2021, accepted 30.5.2021.
Published under open access CC BY-ND. Downloaded from tidsskriftet.no 24 June 2026.