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Subacute De Quervain thyroiditis after SARS-CoV-2 infection☆

Tiroiditis subaguda De Quervain tras infección por SARS-CoV-2

COVID-19 (COronaVIrus Disease 2019), caused by the SARS-CoV-2 virus, is currently a pandemic affecting millions of people around the world. Cases of autoimmune thyroid disease[1] and subacute thyroiditis[2–4] associated with SARS-CoV-2 infection have been reported. We report a case of subacute thyroiditis in our setting following asymptomatic SARS-CoV-2 infection.

A 46-year-old woman with no personal or family history of interest and no ongoing treatment was seen at an endocrinology clinic due to pain in the thyroid area for the previous two months. The pain, which worsened with swallowing and neck movements, started in the right thyroid area, radiated to the right ear and, a month later, spread to the left thyroid lobe and left ear. This was accompanied by a low-grade fever, malaise and insomnia. She had no signs or symptoms of thyrotoxicosis. Examination revealed a grade 1 goitre with significant pain on palpation. The patient provided the results of laboratory testing ordered by her general practitioner that showed hyperthyroidism, with thyroid-stimulating hormone (TSH) levels of 0.11 μIU/ml (0.55–4.78), free T4 levels of 2.18 ng/dl (0.89–1.76), an erythrocyte sedimentation rate (ESR) of 68 mm/h and mild positivity for thyroperoxidase antibodies (TPO Ab), as well as a negative polymerase chain reaction (PCR) test for SARS-CoV-2 infection. She had started treatment with non-steroidal anti-inflammatory drugs (NSAIDs) with partial improvement in her symptoms.

As subacute thyroiditis was suspected, oral prednisone (40 mg per day on a down-titration regimen for six weeks) and further hormone testing with autoimmunity, thyroid ultrasound and thyroid scintigraphy were ordered.

Two weeks later, the patient showed significant clinical improvement in her thyroid function (TSH levels of 0.018 μIU/ml and free T4 levels of 1.68 ng/dl), an ESR of 23 mm/h, C-reactive protein levels of 1.3 mg/dl (0–1) and negativity for TSH receptor Ab. Neck ultrasound revealed a heterogeneous enlarged thyroid gland with normal vascularisation and a heterogeneous, hypoechogenic left thyroid nodule measuring 15 mm × 30 mm, with no cervical lymphadenopathy. Thyroid scintigraphy showed overall hypouptake of the radioactive tracer, consistent with the clinical suspicion of thyroiditis. The patient presented another negative PCR test for SARS-CoV-2.

She was kept on a down-titration regimen of prednisone, and a fine needle aspiration biopsy (FNAB) of the thyroid nodule; further hormone testing; and, in light of case reports of subacute thyroiditis after COVID-19, serology for SARS-CoV-2 were ordered. At her monthly check-up, the patient showed resolution of her signs and symptoms. Laboratory testing revealed mild thyroid hypofunction (TSH 7.75; free T4 0.66), normal C-reactive protein levels and a normal ESR. The FNAB was insufficient for diagnosis. The serology for SARS-CoV-2 showed positivity for IgG, thus confirming past COVID-19. Another FNAB was ordered, but a follow-up ultrasound did not show any thyroid nodule; it only showed areas of focal hypoechogenicity, with no nodules that could be delimited. The patient's thyroid function returned to normal within three months.

Subacute granulomatous thyroiditis, or De Quervain's thyroiditis, is a thyroid inflammatory disease of probable viral origin (direct infection or a post-viral inflammatory reaction in genetically predisposed individuals). It has been most often linked to enterovirus, adenovirus, Coxsackievirus and measles virus infections as well as parathyroiditis.[5] Our patient, previously asymptomatic, developed signs and symptoms typical of subacute thyroiditis two months after the COVID-19 pandemic was declared, but did not seek care until two months after the onset of her symptoms. Additional testing and her clinical course supported the diagnosis of subacute thyroiditis, and serology confirmed past COVID-19; active infection was ruled out on two occasions in the course of her clinical follow-up. Our case offers evidence on the relationship between SARS-CoV-2 and subacute thyroiditis. According to the latest report prepared by the Red Nacional de Vigilancia Epidemiológica [Spanish National Epidemiological Surveillance Network],[6] 50.6% of patients with COVID-19 detected as of 10 May 2020 have been asymptomatic. Hence, given the non-negligible percentage of patients who do not show symptoms, we believe that patients with signs and symptoms consistent with subacute thyroiditis must be assessed for possible past COVID-19.

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Subacute thyroiditis might be a complication triggered by SARS-CoV-2

La tiroiditis subaguda podría ser una complicación causada por el SARS-CoV-2

Subacute thyroiditis (SAT), also called subacute granulomatous thyroiditis or De Quervain's thyroiditis, is a disease characterized by sudden onset of neck pain and thyrotoxicosis. Although the cause of SAT has not been well established, clusters of the disease have been reported during outbreaks of viral infection. Hence, adenovirus and some enterovirus might be responsible for a large proportion of cases.[1]

The actual coronavirus disease 2019 (COVID-19) pandemia, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has reached more than 16 million confirmed cases worldwide, being the United Kingdom, Spain and Italy the most affected countries in Europe.

We describe a clinical case of SAT following SARS-CoV-2 infection.

On April 15th a 28-year-old nurse, with no prior illness known but a family history of autoimmune disease in first degree relatives (Celiac disease and Crohn's disease), initiated diarrhea and abdominal pain, without fever or respiratory symptoms. She was working in an in-patient COVID-19 ward of a hospital in Lleida (Spain). On May 1st a naso-pharyngeal swab test for SARS-CoV-2 was positive. On May 14th diarrhea ended, while she started with fever up to 38.5 °C, neck pain irradiated to the jaw, sore throat, palpitations and severe asthenia. Thyroid function was assessed, showing hyperthyroidism (thyrotropin (TSH) < 0.001 mU/L (normal range 0.38–5.33), free thyroxine (FT4) 37.5 pmol/L (normal range 7.0–16.0)). She also had a C-reactive protein (CRP) of 176 mg/L (normal range 0.0–6.0) and a mild leukocytosis (11.2 × 10 × 9/L) with neutrophilia (77.9%). Paracetamol (1 g/8 h) was prescribed to treat fever and neck pain. The naso-pharyngeal swab test for SARS-CoV-2 turned negative on May 21st. She arrived at our service on May 25th, without symptoms relief and blood test showed hyperthyroidism had worsened (TSH < 0.001 mU/L, FT4 67.5 pmol/L). Antibodies to thyroglobulin (TgAb), peroxidase (TPOAb) and TSH-receptor were negative. CRP was 173 mg/L and erythrocyte sedimentation rate (ESR) was 116 mm (normal range 0–20). During the physical exam, she presented with a grade 2 goiter that was excruciating when palpated and sharp in character. She had no signs of Grave's ophthalmopathy or any skin lesions. She suffered a sinus tachycardia at 150 bpm without fever at that moment. We started treatment with 500 mg of aspirin and 40 mg of propranolol, both every 6 h. Symptoms improved in 24 h, with a total relief in 2 weeks. On June 2nd FT4 was 50.4 pmol/L. On June 15th FT4 was 10.5 pmol/L, CRP was 2.1 mg/L and ESR 56 mm. Thyroid scintigraphy with 5.73 mCi of ^{99m}Tc-pertechnetate was performed on May 26th, which showed absence of uptake in the gland (Fig. 1).

SAT is thought to be the result of cytolytic T-cell recognition of viral and cell antigens in appropriate complexes, but direct data of the presence of virus or their components in the thyroid gland are limited.[1] To our knowledge, no cases of SAT were reported in the SARS epidemic of 2002.[1,6] In autopsies of patients with SARS, an extensive injury of the follicular epithelial cells and the parafollicular cells was described,[7] although SARS associated coronavirus was not found in the thyroid gland of autopsy samples.[8] To date, four cases of subacute thyroiditis during or shortly after SARS-CoV2 infection have been reported.[2–5] Three of those were in Italy and one in Turkey. No remarkable data in thyroid autopsies of COVID-19 fatalities have been reported to date.[9,10]

Figure 1 ^{99m}Tc-pertechnetate scintigraphy. Absent ^{99m}Tc-pertechnetate uptake during thyroid scintigraphy performed in day + 12 after onset of thyroiditis symptoms.