Case Report

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Subacute thyroiditis following COVID-19 vaccination: Case presentation

Aleksandra Z Tomic[1], Sonja S Zafirovic[2], Zoran M Gluvic[1], Vladimir S Samardzic[1], Mirjana T Macvanin[2], Maja LJ Radunovic[3], Esma R Isenovic[2]

[1]Clinic for Internal Medicine, Department of Endocrinology and Diabetes, Zemun Clinical Hospital, Faculty of Medicine, University of Belgrade, Belgrade, Serbia

[2]Department of Radiobiology and Molecular Genetics, VINČA Institute of Nuclear Sciences - National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia

[3]Faculty of Stomatology, Pancevo, University Business Academy, Novi Sad, Serbia

Abstract

Background: Subacute thyroiditis (SAT) is an organ-specific disease that various drugs, including COVID-19 vaccines, can trigger. COVID-19 infection has been associated with thyroid gland damage and disease SARS-CoV-2 direct action, euthyroid sick syndrome, and immune-mediated mechanisms are all potential mechanisms of thyroid damage. It denotes thyroid gland inflammation, most commonly of viral origin, and belongs to the transitory, self-limiting thyroid gland diseases group, causing complications in approximately 15% of patients in the form of permanent hypothyroidism. Some authors say SAT is the most common thyroid disease associated with COVID-19.

Purpose: The occurrence of SAT many weeks after administering the second COVID-19 vaccine is rare and has limited documentation in academic literature. This study aims to present the occurrence of SAT after administering the COVID-19 vaccine. We present the case of a 37-year-old man who developed SAT 23 days after receiving the second dose of Pfizer BioNTech's COVID-19 mRNA vaccine.

Research design and study sample: Due to neck pain and an elevated body temperature (up to 38.2°C), a 37-year-old male subject presented for examination 23 days after receiving the second Pfizer BioNTech mRNA vaccine against SARS-CoV-2 viral infection. The patient denied ever having an autoimmune disease or any other disease. Painful neck palpation and a firm, slightly enlarged thyroid gland with no surrounding lymphadenopathy were identified during the exam. The heart rate was 104 beats per minute. All of the remaining physical findings were normal.

Data collection and/or Analysis: Data collected during the disease are integral to the medical record.

Results: Hematology and biochemistry analyses at the initial and follow-up visits revealed minor leukocytosis, normocytic anaemia, and thrombocytosis, followed by a mild increase in lactate dehydrogenase and decreased iron levels. The patient's thyroid function and morphology had recovered entirely from post-vaccine SAT.

Conclusions: Results from this study emphasise the need for healthcare professionals to promptly report any case of SAT related to COVID-19 vaccination. Further investigation is warranted to understand the immunopathogenesis of COVID-19-associated thyroiditis and the impact of COVID-19 immunization on this condition.

Corresponding author email: sonjazafirovic@hotmail.com

Keywords
COVID-19, Pfizer BioNTech vaccine, subacute thyroiditis

Introduction

The COVID-19 pandemic began in 2020 and is still ongoing. SARS-CoV-2 variants with novel mutations are emerging, allowing the virus to avoid existing prevention and treatment measures.[1] Unfortunately, the virus's characteristics and a lack of vaccinated people have contributed to the emergence of various virus strains.[2] COVID-19 clinical manifestations range from asymptomatic infection to severe disease forms, with serious complications that significantly increase morbidity and mortality. In immunocompromised patients, systemic SARS-CoV-2 infection may result in life-threatening syndromes such as adult respiratory distress syndrome (ARDS), haemostatic abnormalities, or bacterial super-infections most typically caused by resistant bacteria present in hospital settings.[3,4]

COVID-19 vaccine development represents a potent weapon for infection control and prevention. Inadequate vaccination coverage, on the other hand, contributed to the emergence of resistant SARS-CoV-2 strains, lowering vaccination's overall efficacy, but it still protects against severe clinical forms of the disease in most cases. COVID-19 vaccines and SARS-CoV-2 infection may aggravate or trigger immune-mediated diseases.[5–7]

COVID-19 infection has been associated with thyroid gland damage and disease.[8] SARS-CoV-2 direct action, euthyroid sick syndrome, and immune-mediated mechanisms are all potential mechanisms of thyroid damage.[9] Subacute thyroiditis (SAT) is an organ-specific disease that various drugs, including COVID-19 vaccines, can trigger.[10–12] It denotes thyroid gland inflammation, most commonly of viral origin, and belongs to the transitory, self-limiting thyroid gland diseases group, causing complications in approximately 15% of patients in the form of permanent hypothyroidism.[12] According to some authors, SAT is the most common thyroid disease associated with COVID-19.[13]

We present the case of a 37-year-old man who developed SAT 23 days after receiving the second dose of Pfizer BioNTech's COVID-19 mRNA vaccine.

Case report

Due to neck pain and an elevated body temperature (up to 38.2°C), a 37-year-old male patient presented for examination 23 days after receiving the second Pfizer BioNTech mRNA vaccine against SARS-CoV-2 viral infection. The patient denied ever having an autoimmune disease or any other disease.

Painful neck palpation and a firm, slightly enlarged thyroid gland with no surrounding lymphadenopathy were identified during the exam. The heart rate was 104 beats per minute. All of the remaining physical findings were all normal. The blood pressure (BP) reading was 110/70 mm Hg. Electrocardiography (ECG) revealed sinus rhythm and an incomplete right bundle branch block with no ST or T changes. Preliminary thyroid function tests (TFTs) laboratory findings revealed a suppressed TSH, elevated free thyroxine (FT4), and negative results for the presence of various anti-thyroid antibodies (microsomal, thyroglobulin, and TSH receptor antibodies).

At the initial and follow-up visits, haematology and biochemistry analyses revealed normocytic anaemia, raised inflammatory biomarkers (minimal leukocytosis and thrombocytosis), a modest increase in lactate dehydrogenase (LDH), and a decrease in iron levels. Other haematological and biochemical results (Table 1, and Figure 1(a) and (b)) were within reference ranges. Since the outbreak began, RT PCR SARS-CoV-2 testing has been negative. A thyroid ultrasonography revealed a moderately enlarged thyroid gland with heteroechoic, inhomogeneous parenchyma with decreased vascularity and no noticeable nodular changes (Figure 2(a)–(d)). SAT was diagnosed based on clinical presentation, physical examination, laboratory results, and thyroid ultrasonography. Treatment included acetylsalicylic acid (600 mg/day), propranolol (40 mg/day), pantoprazole (40 mg/day), and vitamin D3 (4000 IU/day). During the illness, a persistent febrile state (body temperature up to 37.6°C) was seen, as well as slight neck pain and moderate flu-like symptoms. The patient had mild-to-moderate anterior neck pain and a PR of 120 beats per minute after palpation, accompanied by morning hyperglycemia (7.94 mmol/L). As a result, 3 weeks after beginning therapy, the dose of acetylsalicylic acid was increased to 900 mg/day, propranolol (100–120 mg/day), pantoprazole (80 mg/day), vitamin supplements, and metformin extended-release formulation (750 mg/day). Since laboratory evidence of COVID-19 was unavailable, an initial dose of 4000 IU/day was decreased to 1000 IU/day. The supplementation with vitamin D, initially at 4000 IU/day, then at 1000 IU/day, was following the treatment of viral SAT, COVID-19, and diffuse toxic goitre.[14,15] Clinical follow-ups revealed that the physical finding was gradually improved. Biochemistry laboratory findings gradually regressed (Table 1, Figure 1(a) and (b)), and thyroid function test restoration 3 weeks and nearly 2 months after the initial check-up. Furthermore, thyroid ultrasonography findings (Figure 3(a) and (b)) were normalized 4 and 6 months after the initial thyroid ultrasonography. Gradual improvements in physical, laboratory, and imaging findings led to treatment reduction and discontinuation.

Table 1. Initial and control (a) haematology and (b) biochemistry findings.

			DATE		
	Ref. value	Units	03.04.2021	09.04.2021	29.04.2021
(a)					
WBC	3.4–9.7	10^9/L	9.9↑	10.8 ↑	8.6
RBC	4.34–5.72	10^{12}/L	5.04	4.8	4.69
HGB	138–175	g/L	146	136↓	128↓
HCT	0.415–0.53	L/L	0.43	0.411↓	0.39↓
MCV	83–97.2	fL	85.4	85.7	83.1
MCH	27.4–33.9	pg	28.9	28.3	27.2↓
MCHC	320–360	g/L	339	330	327
PLT	158–424	10^9/L	390	482↑	560↑
(b)					
Na^+	135–148	mmol/L	141	138	139
K^+	3.5–5.1	mmol/L	4.2	5.1	4.5
Urea	2.5–7.5	mmol/L	4.2	6.3	4.8
Creatinine	59–104	µmol/L	78.25	73.2	68.76
Glucose	3.9–6.1	mmol/L	5.21	5.17	4.67
Total bilirubin	0–20.5	µmol/L	15.3	7.3	7.9
AST	0–37	U/L	10	17	28
ALT	0–41	U/L	17	32	61
LDH	50–200	U/L	250↑	296↑	345↑
CK	0–200	U/L	70	58	52
Total proteins	62–81	g/L	75.1	73.3	69.3
Albumin	34–55	g/L	40.8	37.4	37.3
Iron	11–30	µmol/L	2.4↓	—	9.5↓
Ferritin	30–400	µg/L	95.5	—	202

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CK: creatine kinase; HCT: haematocrit; HGB: haemoglobin; K: potassium; LDH: lactate dehydrogenase; MCH: mean corpuscular haemoglobin; MCHC: MHC concentration; MCV: mean corpuscular volume; Na: sodium; PLT: platelet count; RBC: red blood cell; WBC: white blood cells.

Commercial tests were used to determine haematological, biochemical, and immunological parameters on the DxH 900 'Beckman Coulter', DxC Au 700, and Au 480 'Beckman Coulter', according to the manufacturer's instructions.

Discussion

Upper respiratory tract infections, such as infections caused by Coxsackie, cytomegalovirus, Epstein-Barr, influenza, or adenovirus, are frequently followed by SAT, an inflammatory condition of the thyroid gland.[16,17] SAT can be triggered by antiviral drugs (interferon alpha),[18] cytostatic treatment, or vaccines (influenza, H1N1).[19,20] The loss of follicular epithelium mediated by viral infection-induced inflammation is a critical pathophysiological event in post-infectious SAT.[8] Drug-induced destructive thyroiditis (caused by lithium or cytostatic therapy) has a similar clinical, laboratory, and histological appearance but a different mechanism (hypersensitivity reaction, cell apoptosis, and decreased vascularization).[21,22] In light of the current COVID-19 pandemic, infection with SARS-CoV-2[23] may cause worsening of pre-existing thyroid dysfunction or the development of new thyroid diseases and conditions characterized histologically as extensive damage to thyroid follicular epithelium.[24] In patients infected with SARS-CoV-2,[25] histological thyroid examination reveals extensive apoptosis without lymphocytic infiltration, implying that destructive thyroiditis may cause thyrotoxicosis.[26] The aetiology of this chronic thyroid condition is unknown; however, it is most likely caused by genetic, immunological, and environmental factors.[24] The SARS-CoV-2 infection may cause a hyperactive immune response that leads to the release of pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor α (TNF α), resulting in a 'cytokine storm'. Increased IL-6 concentration causes thyrotoxicosis,[27] disruption of proteins involved in thyroid hormone transport, and impaired pituitary cell TSH secretion in the acute phase. As a result, increasing evidence suggests that pituitary-thyroid axis abnormalities have a role in the pathogenesis of SARS-CoV-2 infection.[26,28,29]

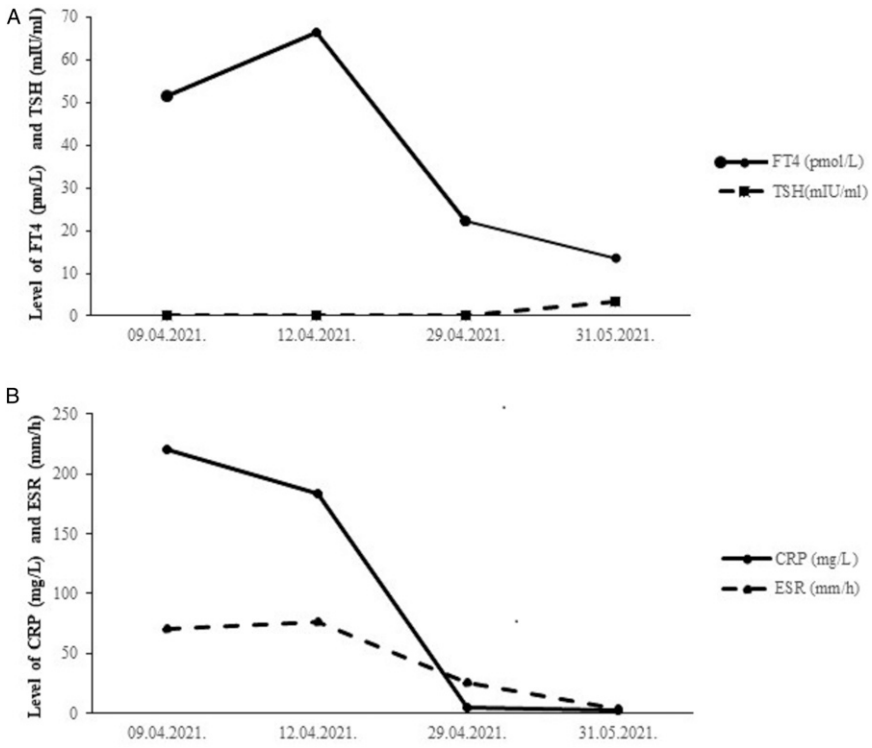

Figure 1. Initial and follow-up (a) thyroid function tests and (b) observed inflammatory parameters. CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; FT4: free thyroxine; TSH: thyroid stimulating hormone.

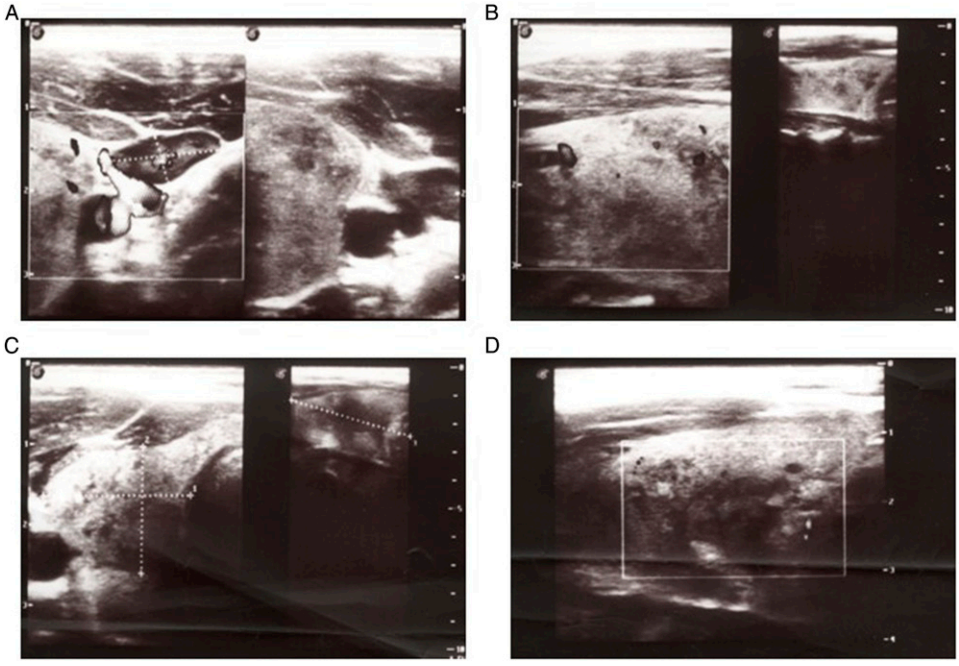

Figure 2. The thyroid gland's left lobe in (a) transverse (left side: an enlarged reactive lymph node near the thyroid's left lobe) and (b) longitudinal scans. The thyroid gland's right lobe in (c) transverse and (d) longitudinal scans. Ultrasonography of the thyroid gland was performed on the Toshiba Xario SSA-660A device by linear probe 7.5 MHz.

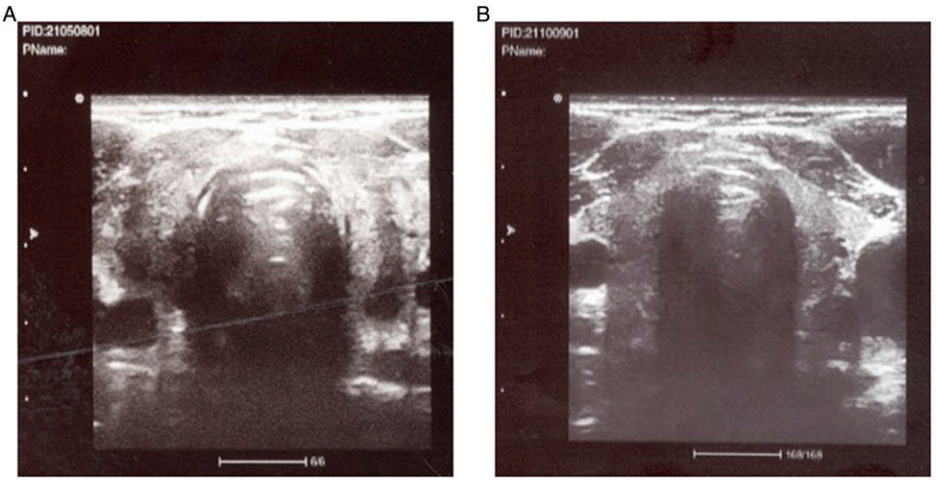

Figure 3. Thyroid gland in the transverse scan: (a) 4 months later, and (b) 6 months later. Ultrasonography of the thyroid gland was performed on the Toshiba Xario SSA-660A device by linear probe 7.5 MHz.

There have been reports of SAT after administering vaccines against various viruses,[20,30] including COVID-19 vaccines.[10] ASIA syndrome (autoimmune/inflammatory syndrome induced by adjuvants) is a post-vaccine phenomenon that occurs after receiving the inactivated SARS-CoV-2 vaccine and vaccinations against human papillomavirus, hepatitis B, and influenza.[10] Endocrine manifestations of ASIA syndrome range from type 1 diabetes, premature ovarian failure, and adrenal insufficiency to SAT.[31] Although the pathogenesis of post-vaccine reactions is unknown, adjuvants are suspected of causing autoimmune diseases or disrupting immune tolerance in genetically predisposed hosts or subjects with pre-existing manifested or non-manifest autoimmune diseases.[10,32,33] Cross-reactivity is conceivable due to molecular mimicry between SARS-CoV-2 viral spike proteins and thyroid cell antigens.[33]

Previous viral infection has been identified as a risk factor for developing SAT in patients with genetic susceptibility. Human leukocyte antigens (*HLA-B35*, *HLA-C04*, and *HLA-A11*) have been linked to SAT susceptibility and may also play a role in the pathophysiology of SAT associated with COVID-19 immunization.[34–38] It is common for post-vaccine or COVID-19-associated SAT to present clinically. Neck discomfort was the most common clinical symptom of post-COVID-19 vaccine-associated SAT, followed by tiredness, fever, myalgia, and various clinical thyrotoxicosis signs (palpitation, tachycardia, and sweating).[39] Females in their twenties and thirties are more likely to have the post-COVID-19 vaccine-associated SAT than males. The median time between immunization and the onset of SAT symptoms ranges from 1 day to 6 weeks, with the first vaccine dose being the most prevalent.[40–42]

We present the case of a young male patient who developed SAT after receiving the second dose of the Pfizer BioNTech mRNA vaccine, despite previous research indicating that this is more common in female subjects after the first dose of the vaccine.[43] Haematology and biochemistry analyses at the initial and follow-up visits revealed minor leukocytosis, normocytic anaemia, and thrombocytosis, followed by a mild increase in lactate dehydrogenase (LDH) and decreased iron levels. Rather than an immediate or delayed post-vaccination immunological response, SAT may cause these laboratory results.[10,44] We confirmed that our patient's thyroid function and morphology had recovered entirely from post-vaccine SAT.

SAT is a rare side effect of COVID-19 vaccination, but it can also happen during COVID-19 disease. SAT caused by COVID-19 may be more common than previously thought. As a result, clinical practitioners should report any COVID-19-related complications and the occurrence of COVID-19-related SAT.

The usual protocol is employed for the treatment of COVID-19 and post-vaccine SAT. Further investigation is warranted to gain a comprehensive understanding of the immunopathogenesis of COVID-19-associated thyroiditis and the impact of COVID-19 immunization on this condition.

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Ethical statement

Consent of publication

The patient has provided written informed consent to publish this case, including images.

ORCID iD

Sonja S Zafirovic https://orcid.org/0000-0002-5486-0079

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