Clinical Medicine and Integrative Therapies

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Case Report

Post-Viral Subacute Thyroiditis Presenting With Severe Neck Pain and Transient Thyrotoxicosis: An Expanded Case Report and Literature Review

Burcin Meryem Atak Tel[1], Ramiz Tel[2], Tuba Taslamacioglu Duman[1], Gulali Aktas[1,*]

[1]Department of Internal Medicine, Abant Izzet Baysal University Hospital, Bolu, Turkey

[2]Department of Emergency Medicine, Izzet Baysal State Hospital, Bolu, Turkey

[*]Corresponding author: Gulali Aktas, draliaktas@yahoo.com

Abstract

Background: Subacute thyroiditis is an inflammatory disorder of the thyroid gland that typically follows viral infections and is characterized by anterior neck pain, elevated inflammatory markers, and transient thyrotoxicosis.

Case Presentation: In this report, we describe a 42-year-old man who presented with progressive anterior neck discomfort, fever, tachycardia, and newly uncontrolled hypertension occurring three weeks after a febrile upper respiratory tract infection. Laboratory evaluation revealed significantly elevated inflammatory markers, suppressed thyroid-stimulating hormone levels, and increased free thyroid hormones. Ultrasonography demonstrated hypoechoic, poorly vascularized areas consistent with subacute thyroiditis. The patient was treated with non-steroidal anti-inflammatory drugs and beta-blockers, resulting in complete symptomatic and biochemical recovery by the tenth day of follow-up.

Conclusion: This case aligns with established descriptions of post-viral subacute thyroiditis and adds to the expanding literature that includes cases associated not only with classical respiratory viruses but also with SARS-CoV-2 infection and vaccination. The report emphasizes the importance of timely recognition and appropriate management of this self-limited but clinically striking condition.

Keywords

Subacute hyroiditis, Ultrasonography, Treatment

Article History

Received: 12 December 2025 Revised: 09 January 2026

Accepted: 02 February 2026 Available Online: 03 February 2026

1. Introduction

Subacute thyroiditis (SAT), also known as de Quervain thyroiditis, is a benign, self-limited inflammatory thyroid disorder typically triggered by viral infections. Classically, SAT manifests several weeks after a symptomatic or asymptomatic upper respiratory tract infection, reflecting a post-viral inflammatory response [1]. Numerous viruses have been implicated, including influenza, adenovirus, coxsackievirus, measles, and Epstein–Barr virus. The condition is characterized clinically by anterior neck pain, fatigue, fever, and transient thyrotoxicosis resulting from the destruction of thyroid follicles and release of preformed thyroid hormones into the circulation [2]. Laboratory findings typically include elevated erythrocyte sedimentation rate (ESR), elevated C-reactive protein (CRP), and low radioiodine uptake. Ultrasonography generally reveals poorly defined hypoechoic areas with diminished vascularity.

The global COVID-19 pandemic has renewed interest in SAT as several cases have been reported following SARS-CoV-2 infection as well as various COVID-19 vaccines, as demonstrated in recent publications by Wijenayake et al. and Bornemann et al. [3,4]. Although these cases highlight novel immunologic triggers, classical post-viral SAT continues to occur worldwide irrespective of SARS-CoV-2 exposure.

In this context, we present an expanded case report of SAT following a recent febrile upper respiratory tract infection in a previously healthy man with no known thyroid disease which resolved quickly. The case is discussed in detail and contextualized within contemporary SAT literature.

2. Case Presentation

A 42-year-old man with a history of hypertension presented to the internal medicine outpatient clinic with a three-day history of progressively worsening anterior neck pain. He described the pain as a deep, pressure-like sensation that intensified with swallowing, speaking, or palpation of the anterior neck region. Alongside neck pain, he reported episodes of low-grade fever, increased sweating, headaches, and recently elevated blood pressure measurements. He denied tremors, unintended weight loss, heat intolerance, or other classic symptoms of hyperthyroidism at initial presentation.

His medical history included hypertension managed with perindopril 10 mg and indapamide 2.5 mg daily. He did not smoke, consumed no alcohol, and reported no history of thyroid disease. Importantly, he had experienced a febrile upper respiratory tract infection approximately three weeks prior to symptom onset. He had not received any recent vaccinations and reported no recent travel, trauma, or exposure to toxins.

On physical examination, his temperature was 37.9°C, pulse rate 106 beats per minute, blood pressure 140/90 mmHg, respiratory rate 16 breaths per minute, and oxygen saturation 96 percent on room air. The thyroid gland was notably tender on palpation without visible swelling, erythema, warmth, or palpable cervical lymphadenopathy. Cardiovascular, respiratory, and abdominal examinations were normal. No clinical features suggested Graves disease or bacterial thyroid infection.

Laboratory investigations revealed leukocytosis of 12 $\times 10^3/\mu L$, an erythrocyte sedimentation rate of 98 mm/h, and a C-reactive protein concentration of 107 mg/L, all consistent with significant systemic inflammation. Liver enzymes were mildly elevated, including aspartate aminotransferase 97 U/L, alanine aminotransferase 167 U/L, gamma-glutamyl transferase 391 U/L, and alkaline phosphatase 203 U/L. Thyroid function tests demonstrated suppressed thyroid-stimulating hormone at 0.01 mIU/L, free thyroxine at 3.45 ng/dL, and free triiodothyronine at 6.74 ng/L, indicating marked thyrotoxicosis.

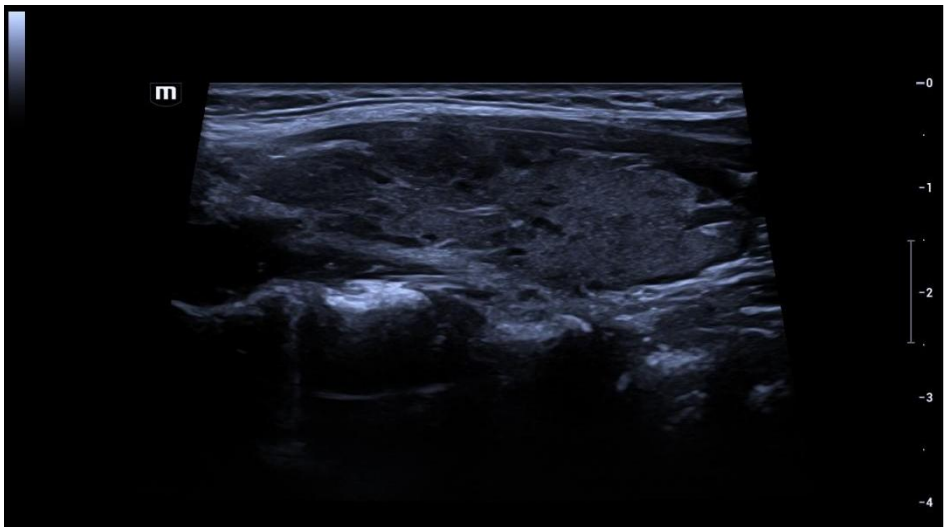

Figure 1. B mode sonography of the thyroid gland.

Hypoechoic area significant for SAT in the middle anterior part of the left thyroid lobe.

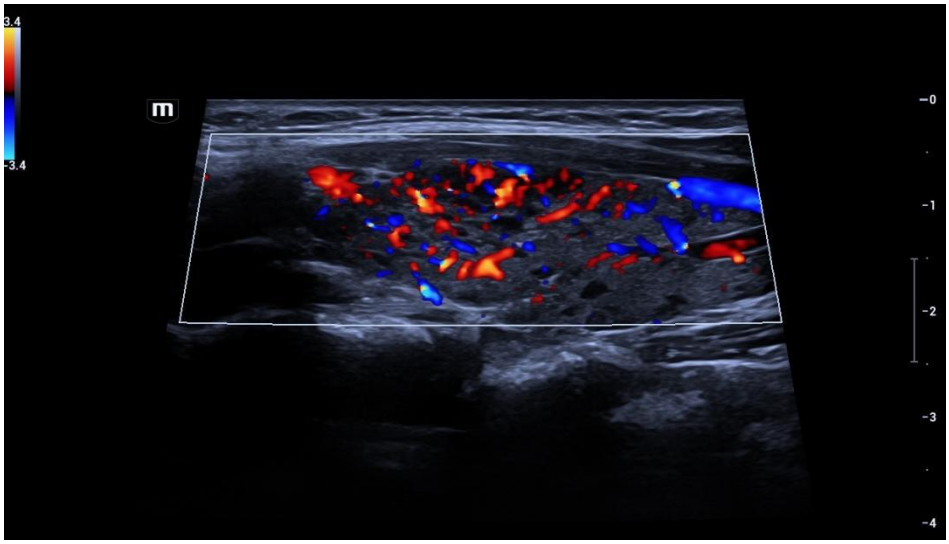

Figure 2. Doppler sonography of the thyroid gland.

Doppler ultrasound shows increased vascularity in the anterior middle section of the left thyroid lobe.

Thyroid ultrasonography revealed diffusely hypoechoic, heterogenous areas with increased vascularity, findings typical of SAT. No nodules or suspicious masses were detected. Figure 1 and 2 shows sonography findings of the patient. Abdominal ultrasonography was unremarkable.

Given the combination of clinical symptoms, elevated inflammatory markers, suppressed thyroid stimulating hormone (TSH), elevated thyroid hormones, and characteristic ultrasonographic findings, the patient was diagnosed with SAT. He was started on ibuprofen retard 800 mg twice daily to control inflammation and propranolol 20 mg twice daily to manage tachycardia and adrenergic symptoms. He was advised to rest, maintain hydration, and monitor his blood pressure regularly. Corticosteroids were reserved as a second-line option should symptoms fail to improve with (Non-steroidal anti-inflammatory drugs) NSAIDs.

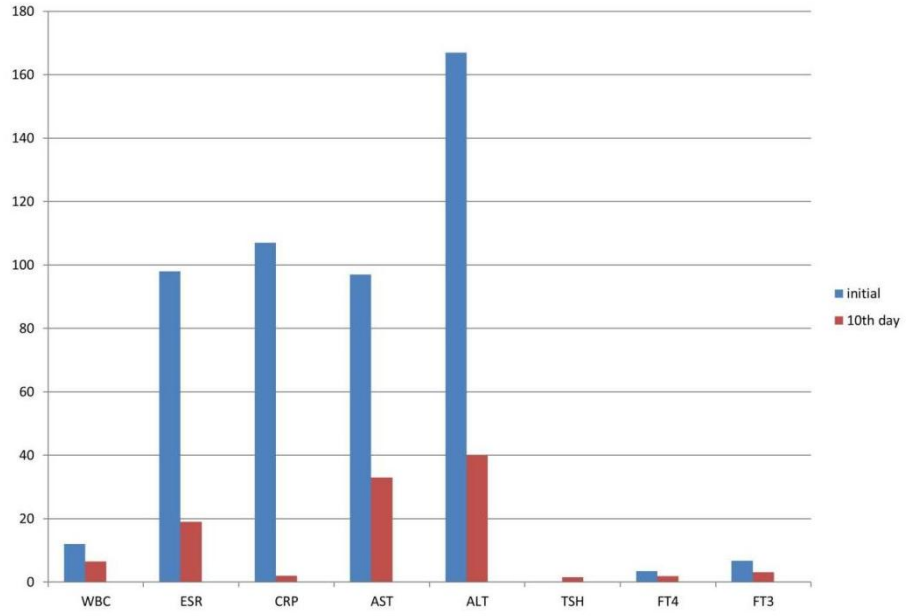

Figure 3. Changes in laboratory values from initial presentation and 10th day.

WBC: leukocyte count (k/mm^3) ESR: erythrocyte sedimentation rate (mm/h) CRP: C-reactive protein (mg/L) AST: aspartate transaminase (U/L) ALT: alanine transaminase (U/L) TSH: thyroid stimulating hormone FT4: Free thyroxine FT3: Free Tri-iodothyronine

At a follow-up appointment ten days later, the patient reported complete resolution of neck pain, headaches, sweating, and palpitations. His blood pressure had returned to its previous well-controlled range. Laboratory tests showed normalization of inflammatory markers and near-complete resolution of thyroid hormone abnormalities. No signs of hypothyroidism emerged during follow-up. He was instructed to return in six weeks for repeat thyroid testing to

monitor for a potential hypothyroid phase. Figure 3 shows the changes in laboratory values from initial presentation and 10th day. He missed his 1st month control after discharge but at third month control, he was still asymptomatic and euthyroid.

3. Discussion

This report describes a classical presentation of post-viral SAT occurring after a febrile upper respiratory tract infection. The timing of symptom onset, approximately three weeks after the initial illness, aligns with the typical latency period described in SAT, during which viral-triggered immune processes lead to thyroid follicular damage. The patient's clinical features, including anterior neck pain exacerbated by swallowing and palpation, low-grade fever, and systemic malaise, closely resemble the presentation reported in multiple published case series.

In recent literature, SAT has been increasingly reported in temporal association with SARS-CoV-2 infection, illustrating a clinical pattern similar to classic post-viral SAT. In a systematic review of 21 COVID-19–related SAT cases, mean onset of thyroid symptoms occurred approximately 25 days after the initial infection, with neck pain and fever being the most frequent presentations; all patients exhibited low TSH and elevated T3/T4, and inflammatory markers such as ESR and CRP were consistently elevated. Ultrasound typically revealed hypoechoic regions consistent with SAT, and most patients improved with anti-inflammatory treatment, though a minority developed transient hypothyroidism on follow-up [5]. Individual case reports also describe similar findings, including diffuse hypoechoic ultrasound features and rapid resolution of symptoms with steroid therapy [6]. Published data on SAT following SARS-CoV-2 vaccination show cases with comparable clinical and laboratory profiles that resolved with NSAIDs or steroids [7]. Compared with these SARS-CoV-2–related cases, our 42-year-old patient similarly developed significant anterior neck pain, elevated ESR/CRP, suppressed TSH, and elevated free T3/T4, with typical ultrasound findings of diffuse hypoechoic, heterogeneous thyroid tissue. His recovery with NSAID therapy and without progression to hypothyroidism aligns with the favorable clinical course reported in many COVID-19–associated SAT cases, suggesting that the underlying inflammatory mechanisms in viral-triggered SAT, including SARS-CoV-2–related disease, may be broadly comparable.

SAT continues to be associated with various viral pathogens [8-10]. Although recent literature has emphasized SARS-CoV-2–related SAT, the present case demonstrates that classical post-viral SAT remains prevalent independent of the COVID-19 pandemic. Studies such as those by Wijenayake and colleagues and Bornemann and colleagues have proposed immunological mechanisms including molecular mimicry and adjuvant-induced immune activation as triggers for SAT following vaccination. In contrast, this patient's illness appears to reflect traditional post-infectious thyroid inflammation likely mediated by cytolytic T-cell responses directed against thyroid follicular cells, a mechanism previously characterized in granulomatous thyroiditis [11].

The laboratory findings in this case were typical. In SAT, destructive release of preformed hormones causes elevated serum free T4 and T3 with suppressed TSH, but without the elevated radioiodine uptake seen in disorders of hormone overproduction such as Graves disease. Elevated inflammatory markers, particularly ESR CRP, frequently aid in differentiating SAT from other causes of thyrotoxicosis. Mild liver enzyme elevations, as seen in this patient, have also been reported in association with significant systemic inflammation in SAT.

Ultrasonography is an essential diagnostic tool, and the findings of hypoechoic, poorly vascularized areas strongly supported the diagnosis [12]. The lack of increased blood flow helped to differentiate SAT from hypervascular conditions such as Graves disease, and the absence of localized fluid collection reduced the likelihood of suppurative thyroiditis.

Therapeutically, non-steroidal anti-inflammatory drugs remain the recommended first-line treatment for pain and inflammation in SAT. Beta-blockers provide effective symptomatic relief of thyrotoxic features such as palpitations and tremors. Corticosteroids are reserved for patients with severe or refractory pain, inadequate response to NSAIDs, or contraindications to NSAID therapy [12]. In this case, NSAIDs alone provided rapid relief, consistent with the proportion of patients who experience full resolution without the need for steroids.

Although many cases of SAT follow a triphasic pattern with initial thyrotoxicosis, a subsequent transient hypothyroid phase, and eventual recovery to euthyroidism, the patient described here demonstrated rapid normalization of thyroid function without progression to hypothyroidism. This course is consistent with milder forms of SAT, although the patient will continue to be monitored given that 5 to 15 percent of individuals may ultimately develop permanent hypothyroidism. The disease can mimic other conditions such as tuberculosis [13]. But, present case was not mimicking other diseases.

Hashimoto's thyroiditis is the most common autoimmune form and a leading cause of hypothyroidism in iodine-sufficient populations. It arises from a complex immune dysregulation where both humoral (antibody-mediated) and cellular immunity attack thyroid tissue. Genetic predisposition and environmental triggers combine to break immune tolerance to thyroid antigens such as thyroid peroxidase (TPO) and thyroglobulin [14]. Etiologic components include genetic susceptibility, autoantibodies, cellular immunity, and environmental/non-genetic triggers. Multiple immune-related genes contribute, though inheritance is non-Mendelian and involves many small-effect variations [15,16]. Anti-

TPO and anti-thyroglobulin antibodies are frequently present; TPO antibodies are key markers and may partly mediate cytotoxicity [15]. T cell–mediated cytotoxicity against thyroid follicular cells drives ongoing damage and fibrosis [14]. Factors such as age, female sex hormones, pregnancy, infection, radiation, and certain drugs may modulate risk and severity [15,16]. None of these features were present in the present case.

SAT is not primarily autoimmune. Rather, it is thought to be triggered by a viral or post-viral inflammatory process. This leads to a pronounced inflammatory reaction with granuloma formation and acute pain [17,18]. Viral triggers have a role in etiology of the disease. Historically associated with upper respiratory viruses (e.g., mumps, influenza, adenovirus, Coxsackie), and recently described as occurring after SARS-CoV-2 infection [19,20]. The thyroid becomes an innocent bystander where immune responses to the viral pathogen induce local inflammation rather than classical autoimmune targeting of specific thyroid antigens [17]. Present case was not presented with such a course.

Another form of thyroiditis is silent (painless) thyroiditis. Silent and postpartum thyroiditis are closely related autoimmune conditions. They tend to follow the same destructive pathophysiology as Hashimoto's but with a more transient course [20]. Lymphocytic infiltration of the thyroid gland, antibody association and immune rebound mechanism suggest its autoimmune background. Histology shows lymphocytic infiltration similar to chronic autoimmune thyroid disease [20]. Anti-TPO antibodies are common, and higher pre-pregnancy levels predict postpartum thyroiditis and this condition may result from an immune rebound after pregnancy-associated immunosuppression resolves [21]. Present patient was male so silent thyroiditis was not the case.

Acute bacterial thyroiditis is rare and results from direct infection of the thyroid gland, often via hematogenous spread or anatomic defects such as pyriform sinus fistula. Common pathogens include Staphylococcus aureus, Streptococcus species and other bacteria, especially in immunocompromised hosts [22]. There was no sign of supurative thyroiditis in our case.

Table 1. Differentials and characteristics of various etiology of thyroiditis.

Feature	**SAT**	**Silent/Painless Thyroiditis**	**Postpartum Thyroiditis**	**Hashimoto's Thyroiditis**	**Acute Suppurative Thyroiditis**	**Drug-Induced Thyroiditis**
Etiology	Post-viral inflammatory	Autoimmune, self-limited	Autoimmune, postpartum immune rebound	Autoimmune (anti-TPO, anti-TG)	Bacterial infection (Staph, Strep)	Amiodarone, interferon-α, IL-2, TKIs
Pain	Severe, painful, tender thyroid	Painless	Painless	Usually painless, firm goiter	Very painful, erythema	Painless or mild pain
Onset	Subacute	Subacute	Within 1 year postpartum	Chronic	Acute, hours–days	Variable
Systemic Symptoms	Fever, malaise	Minimal	Minimal	Hypothyroid symptoms later	Fever, leukocytosis	Depends on drug
Thyroid Function Pattern	Hyper to hypo then recovery	Hyper to hypo then recovery	Hyper to hypo then recovery	Often euthyroid sometimes permanent hypothyroid	Usually euthyroid or mild hypo	Hyper or hypo depending on drug
T3/T4 Levels	High T4/T3 (release)	High T4/T3	High T4/T3	Normal/low	Usually normal	Variable
TSH	Suppressed → high later	Suppressed → high later	Suppressed → high later	Often high (hypo)	Normal or high	Variable
Radioactive Iodine Uptake (RAIU)	Low	Low	Low	Normal or low	Normal	Low (in destructive type from amiodarone)
ESR/CRP	Very high ESR/CRP	Normal or mildly ↑	Normal or mildly ↑	Normal or mildly ↑	Markedly ↑, leukocytosis	Normal
Autoantibodies	Negative	Often + anti-TPO	Often + anti-TPO	Strongly positive anti-TPO/anti-TG	Negative	Sometimes positive (interferon-induced)
Ultrasound	Hypoechoic, reduced vascularity	Hypoechoic	Hypoechoic	Diffuse heterogenous, ↑ vascularity	Focal abscess	Reduced vascularity (destructive)
Treatment	NSAIDs; steroids if severe; β-blockers	β-blockers; often self-limited	β-blockers; levothyroxine if hypo	Lifelong levothyroxine	IV antibiotics; drainage	Manage drug; β-blockers ± steroids
Outcome	Usually complete recovery	Recovery in months	Recovery in months	Permanent hypothyroidism common	Resolves with treatment	Recovery if drug stopped

Certain medications may cause destructive thyroiditis or immune activation leading to thyroid inflammation. Those include amiodarone, interferon-α, interleukin-2, lithium and immune checkpoint inhibitors. These drugs may induce thyroiditis via direct cytotoxicity, immune modulation, or both [23]. The patient reported in this case was not using any of those drugs.

There are also infrequent types of thyroiditis. For example, radiation-induced thyroiditis may result injury from therapeutic or diagnostic radiation causing thyroid inflammation [24]. Riedel's (Fibrous) Thyroiditis is another rare form of thyroiditis and associated with idiopathic fibrosing inflammation in the gland. Its etiology is unclear and may have overlap with fibrosing systemic disorders [25]. Present case didn't fit none of those two etiology. Table 1 shows various causes of thyroiditis and their characteristic features.

4. Conclusion

This extended case report presents a typical example of post-viral SAT associated with classic clinical, laboratory, and ultrasonographic features. The patient experienced rapid symptomatic and biochemical recovery following treatment with NSAIDs and beta-blockers. The case underscores the importance of considering SAT in patients presenting with anterior neck pain and thyroid dysfunction following recent viral illness. Recognition of SAT prevents unnecessary interventions, facilitates appropriate management, and contributes to the broader understanding of the variable clinical presentations of this self-limited condition.

Conflict of Interest

There is none.

Informed Consent

Informed consent was obtained from the patient.

Availability of Data and Materials

Anonymized data is available upon reasonable request.

Generative AI Statement

The authors declare that no Gen AI was used in the creation of this manuscript.

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