Nasr and Turkel
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Subacute thyroiditis diagnostic challenges in clinical practice: a case report

Payman Nasr[1*] and Sarah Turkel[1]

Abstract

Background Subacute thyroiditis is often misdiagnosed owing to its nonspecific symptoms, which can mimic other conditions. Accurate diagnosis relies heavily on thorough physical examination and careful interpretation of laboratory tests.

Case information A 53-year-old female of Iranian (Persian) ethnicity was initially misdiagnosed with sinusitis after presenting with persistent anterior neck pain, fever, and worsening fatigue. As her symptoms progressed, she was later diagnosed with pyelonephritis, further delaying the recognition of her underlying condition. After multiple visits, a comprehensive physical examination revealed peripheral vision loss and thyroid tenderness. Laboratory tests eventually confirmed subacute thyroiditis. The patient was treated with a 10-day course of prednisone, resulting in rapid symptom improvement.

Conclusion This case underscores the critical role of a comprehensive physical examination and strategic selection and interpretation of laboratory tests in accurately diagnosing subacute thyroiditis. Early recognition through thorough clinical assessment can help prevent diagnostic delays, unnecessary treatments, and prolonged patient symptoms.

Keywords Subacute thyroiditis, Hyperthyroidism, Physical examination, Diagnostic errors, Case report

Introduction

Subacute thyroiditis (SAT), also known as De Quervain's thyroiditis, is an inflammatory disorder of the thyroid gland first described by Swiss surgeon Fritz de Quervain in [1]. This condition is typically preceded by a viral upper respiratory infection and is characterized by neck and ear pain, fever, and transient hyperthyroidism, which is later followed by hypothyroidism. While generally self-limiting, subacute thyroiditis can cause significant discomfort and, in some cases, long-term thyroid dysfunction, if not properly managed. The inflammatory process can mimic other conditions, often leading to diagnostic uncertainty, particularly in cases where symptoms overlap with infectious or autoimmune disorders. The transient hyperthyroidism seen in SAT adds further complexity to its diagnosis, as it may present with nonspecific systemic symptoms such as fatigue, palpitations, and fever, which can be mistaken for infectious etiologies. As the condition progresses, the shift from hyperthyroidism to hypothyroidism may create additional diagnostic confusion, leading to fragmented care and unnecessary medical interventions [7]. Given its episodic nature, a lack of clinical suspicion can result in frequent misdiagnoses, prolonged patient symptoms, and the delayed initiation of appropriate treatment.

The current case report highlights the diagnostic challenges faced in a patient who presented with multiple emergency room (ER) visits and was initially diagnosed with sinusitis, followed by pyelonephritis. The overlapping symptoms and the absence of a thorough clinical examination contributed to a significant delay in

*Correspondence:
Payman Nasr
pnasr@csudh.edu
[1] Clinical Science Department, California State University, Dominguez Hills, Carson, CA 90747, USA

recognizing subacute thyroiditis as the underlying cause of the patient's condition. The resulting diagnostic uncertainty led to multiple interventions, increased healthcare costs, and prolonged symptomatology. This case underscores the importance of a comprehensive clinical evaluation, including a thorough initial hands-on assessment through physical examination (inspection, palpation, percussion, and auscultation) integrated with targeted laboratory and imaging studies to avoid misdiagnosis and ensure timely, effective management of subacute thyroiditis.

Timeline

Day 1	Initial outpatient clinic visit; diagnosed with sinusitis, prescribed amoxicillin–potassium clavulanate (pot clavulanate) (875–125 mg twice daily for 5 days)
Day 2	C-reactive protein (CRP) elevated; advised to visit ER; advised to continue amoxicillin–pot clavulanate
Day 4	Symptoms worsened; ER visit; prescribed doxycycline (100 mg twice daily) and discharged
Day 7	Symptoms worsened; ER visit; treated with pain medication and antiemetics and advised to continue antibiotics and follow up with the primary physician
Day 13	Symptoms worsened; ER visit; ceftriaxone intravenous (2 mg given); prescribed ciprofloxacin (500 mg twice daily for 7 days); discharged
Day 16	Persistent symptoms; outpatient clinic evaluation; referral to ER
Day 17	ER visit; physical examination revealed loss of peripheral vision, and thyroid tenderness; diagnosis of subacute thyroiditis suspected and confirmed following thyroid function assessment; hospital admission
Day 18	Start prednisone (50 mg tablet)
Day 21	Symptoms improvement; normalized thyroid function; discharged

Narrative

Patient information

A generally healthy 53-year-old female of Iranian (Persian) ethnicity first sought medical assistance at a local urgent treatment center after experiencing symptoms of an upper respiratory infection, including headache, fatigue, myalgia, bilateral otalgia, chills, and a slight sore throat that had persisted for more than 10 days. Her vital signs were recorded, showing blood pressure of 119/66 mmHg, a pulse of 98 bpm, temperature of 36.7 °C, weight of 59 kg, and oxygen saturation (SpO_2) of 97%. A chest X-ray showed clear lungs with no evidence of pulmonary infiltrations, nodules, or cavities.

The patient's medical history was significant for a prior diagnosis of breast cancer, treated with surgical intervention and chemotherapy 5 years earlier. She remained in remission, with no history of thyroid disease or autoimmune disorders. She reported no smoking, alcohol use, or illicit drug use. Socially, she is college-educated, married, and employed full-time, with unrestricted access to healthcare and no reported financial or social barriers. There were no clinical or historical findings suggestive of subarachnoid hemorrhage, central nervous system (CNS) bleeding, cerebral aneurysm, tumors, or CNS infections.

Case presentation and clinical findings

The laboratory results are shown in the diagnostic table. She was initially diagnosed with sinusitis with chills and prescribed amoxicillin-pot clavulanate (875–125 mg) oral tablets and discharged. The next day, upon availability of the CRP results (51.8 mg/L, normal <7.4 mg/L), the urgent care attending physician contacted the patient and advised her to proceed to the emergency room for further evaluation, which the patient did the following day. In the emergency room, despite her persistent symptoms, her physical examination remained superficial and primarily relied on the previous diagnosis of sinusitis. The patient was advised to continue the antibiotic regimen.

Over the next 2 days, the patient's symptoms continued to worsen. Concerned about her worsening condition, she returned to the emergency room, reporting bilateral flank pain characterized by a gradual onset, dull, and constant discomfort localized to the flank region. Given the previous presence of *Escherichia coli* in her urine culture, along with fever and chills, the attending physician diagnosed her with pyelonephritis. Additional blood and urine cultures were obtained, and she was started on intravenous antibiotics. She was later discharged with a prescription for oral doxycycline (100 mg twice daily).

A week passed with no improvement. The patient's symptoms grew more severe, prompting a return to the emergency room, where she was treated with pain medication and antiemetics and was advised to continue antibiotics and follow up with her primary physician. During this visit she was administered intravenous ceftriaxone (2 gm) and prescribed ciprofloxacin (500 mg twice daily for 7 days) before being discharged.

Over the next 3 days, the patient's symptoms continued to worsen, with her nocturnal fever spiking between 104 °F and 105 °F. She experienced severe mental disturbances during episodes of high fever, which her husband described as "hallucinations and paranoia" and "a fear of being harmed." These disturbances resolved when the fever subsided, suggesting a transient febrile delirium rather than a primary psychiatric disorder. Despite her escalating symptoms, she was not admitted to the hospital initially because her vital signs remained stable outside febrile episodes, and her urinalysis and urine culture

initially suggested a urinary tract infection (UTI), leading to continued outpatient management with antibiotics.

Over the next 4 days, the patient's symptoms grew more severe, prompting a return to the emergency room. During this visit, she refused to be discharged without a comprehensive medical workup. The chief attending physician conducted a thorough hands-on physical examination, which revealed a loss of peripheral vision and a tender, enlarged thyroid gland, raising suspicion of thyroid dysfunction. Given these findings, the patient was admitted to the hospital for further evaluation to confirm the diagnosis and initiate appropriate management.

The initial management included intravenous hydration, nonsteroidal antiinflammatory drugs for pain control, and beta-blockers (propranolol 20 mg twice daily) for symptom management. In addition, the patient underwent further diagnostic evaluation, including thyroid function tests, inflammatory markers, and imaging studies.

Diagnostic assessment

A thorough examination of the thyroid function tests revealed significant abnormalities. Briefly, the thyroid-stimulating hormone (TSH) level was markedly suppressed at < 0.01 mcL/mL. This suppression of TSH is indicative of a hyperthyroid state, a common finding in both Graves' disease and SAT. Further corroborating this hyperthyroid state were the elevated levels of thyroid hormones. Triiodothyronine (T3) and free thyroxine (T4) levels were elevated at 191 ng/dL and 2.2 ng/dL, respectively.

Given the hyperthyroid state, a thorough differential diagnosis was considered to distinguish SAT from other causes of thyrotoxicosis. Graves' disease was an initial consideration, given the suppressed TSH and elevated thyroid hormone levels. However, the absence of thyroid-associated ophthalmopathy and the lack of a diffuse goiter on examination reduced the likelihood of Graves' disease. Furthermore, serologic evaluation revealed low thyroid peroxidase antibodies (TPO AB) at less than 3.0 IU/mL and thyroid-stimulating immunoglobulins (TSI) at less than 89% of the baseline, along with low TSH receptor antibodies (TRAb) levels of less than 1.10 IU/L, effectively ruling out autoimmune hyperthyroidism.

Toxic multinodular goiter and solitary toxic adenoma were also considered in the differential, as both conditions can present with suppressed TSH and elevated thyroid hormones. However, the absence of nodularity in imaging studies, along with the self-limiting nature of symptoms, made these conditions unlikely. Additionally, iodine-induced hyperthyroidism (Jod-Basedow phenomenon) was ruled out, as the patient had had no recent contrast exposure or excessive iodine intake.

To summarize, the patient's suppressed TSH, elevated T3 and free T4 levels, and low antibody titers (TPO AB, TSI, and TRAb) collectively supported the diagnosis of SAT rather than Graves' disease or another form of hyperthyroidism. The clinical course, marked by transient hyperthyroidism without signs of chronic autoimmune disease, further reinforced this conclusion. This condition was likely in the thyrotoxic phase, characterized by inflammation-induced excessive release of thyroid hormones. The lab findings, coupled with the patient's clinical presentation, confirmed the nonautoimmune inflammatory nature of thyroid dysfunction.

Therapeutic intervention

Given the persistent symptoms, she was started on a 10-day tapering course of prednisone (50 mg/day). After initiating treatment, the patient's condition improved rapidly, with complete resolution of fever and flank pain. However, she experienced mild steroid-induced hyperglycemia, which was managed with dietary modifications and close glucose monitoring, without the need for insulin therapy. The patient remained hospitalized for 4 days for close monitoring of her symptoms and response to treatment. At discharge, she was stable, with resolving thyroid tenderness and improvement in systemic symptoms. She was advised to follow up in 2 weeks with endocrinology for repeat thyroid function tests and further evaluation.

Follow-up and outcomes

At her follow-up visit 2 weeks postdischarge, the patient showed significant clinical improvement, with normalization of inflammatory markers and partial recovery of thyroid function. Her TSH remained slightly suppressed, but her T3 and free T4 levels were trending toward normal. No long-term complications or persistent hypothyroidism were observed.

Diagnostics

Type	Value	Unit
Days 1–2		
C-reactive protein (CRP)	51.8	mg/L
Erythrocyte sedimentation rate (ESR)	30	mm/first hour
Hematocrit (Hct)	39.5	%
Total white blood cells (WBC)	10,200	cells/mm^3
SARS-CoV-2 (COVID-19)	Nonreactive	

Type	Value	Unit
Day 4		
Hct	36.1	%
Urine culture	Positive	
WBC	12,400	cells/mm^3
Day 7		
Hct	36.9	%
Urine culture	Negative	
WBC	11,500	cells/mm^3
Day 13		
C-reactive protein (CRP)	44.3	mg/L
Hct	33.9	%
WBC	9100	cells/mm^3
Day 16		
Hct	35.6	%
WBC	10,100	cells/mm^3
Day 17		
Erythrocyte sedimentation rate (ESR)	74	mm/first hour
Hct	35.4	%
Triiodothyronine (T3)	191	ng/dL
Free thyroxine (T4)	2.2	ng/dL
Thyroglobulin antibody (TgAb)	89	%
Thyroid peroxidase (TPO) antibody	3	IU/mL
Thyroid-stimulating hormone (TSH)	< 0.01	mIU/L
Urine culture	Negative	
WBC	7800	cells/mm^3
Day 18		
Hct	32.8	%
WBC	7800	cells/mm^3
Day 21		
T3	63	ng/dL
TSH	0.12	mU/L

Discussion

SAT is an inflammatory disorder of the thyroid gland often associated with viral infections. It typically presents with anterior neck pain, fever, and transient thyrotoxicosis, followed by a hypothyroid phase before eventual recovery. The condition is self-limiting, but its diagnosis can be challenging owing to its nonspecific symptoms and overlapping with other disorders. The initial presentation frequently mimics pharyngitis or upper respiratory tract infections, leading physicians to misattribute symptoms and prescribe antibiotics without considering thyroiditis [7, 11]. Furthermore, the transient hyperthyroid phase can be mistaken for more prevalent thyroid disorders such as Graves' disease or toxic multinodular goiter, especially when thyroid function tests show elevated thyroid hormone levels [2].

SAT is generally believed to be of viral origin, often following an upper respiratory tract infection [3]. Commonly implicated viruses include coxsackievirus, mumps, adenovirus, and echovirus [15]. The inflammatory process results in the destruction of thyroid follicular cells, leading to the release of thyroid hormones into the bloodstream and a subsequent hyperthyroid phase. This hyperthyroid phase is often followed by a hypothyroid phase as the thyroid hormone stores become depleted [12]. Patients with subacute thyroiditis typically present with a sudden onset of anterior neck pain that radiates to the jaw or ears, fever, and general malaise [7]. The neck pain is often aggravated by swallowing and head movement. Other symptoms may include palpitations, sweating, fever, weight loss, anxiety, visual impairment, and fatigue during the hyperthyroid phase. As the disease progresses, patients may develop hypothyroid symptoms such as weight gain, cold intolerance, and depression.

Several case reports have documented SAT in different clinical contexts. Elawady *et al.* [6] reported a case of a 33-year-old woman who developed SAT 2 weeks after recovering from coronavirus disease 2019 (COVID-19). She presented with neck pain radiating to the ear, fever, and malaise. Laboratory tests revealed low TSH, normal free T4 and T3 levels, and negative thyroid antibodies. The patient was treated with corticosteroids, leading to full recovery. Bornemann *et al.* [4] described two cases of SAT following SARS-CoV-2 vaccination. Both patients developed neck pain and tenderness approximately two weeks after vaccination. Ultrasound findings showed hypoechoic areas with reduced blood flow, and fine-needle aspiration confirmed SAT. As with the previous case, corticosteroid therapy resulted in complete symptom resolution. Similarly, Chakraborty *et al.* [5] presented a case of a 58-year-old man with painful neck swelling who was later diagnosed with COVID-19. These cases underscore the necessity of considering SAT in the differential diagnosis of neck pain, particularly following a recent COVID-19 infection. However, in contrast to these reports, the patient in the present case tested negative for SARS-CoV-2 on multiple occasions, effectively ruling out a recent COVID-19 infection as a potential precipitant of SAT.

Previous reports have documented psychiatric manifestations of subacute thyroiditis (SAT), including hallucinations and delusions, even in the absence of significant thyroid-related symptoms [9]. In one such case, further evaluation revealed elevated inflammatory markers and suppressed thyroid-stimulating hormone (TSH) levels, leading to a diagnosis of SAT, with symptoms resolving following corticosteroid therapy. However, in the present case, the patient's condition progressively deteriorated, with nocturnal fevers reaching 104–105 °F. During these

febrile episodes, she experienced severe mental disturbances that subsided as the fever resolved, suggesting a transient febrile delirium rather than an underlying psychiatric disorder. This distinction highlights the importance of considering systemic inflammatory conditions, such as SAT, in patients presenting with acute psychiatric symptoms in the setting of hyperpyrexia.

The episodic nature of thyroiditis symptoms, which can vary over time, further complicates diagnosis and may result in fragmented care. When physicians focus only on the symptoms present at each visit without a comprehensive review of the patient's overall clinical course, they may miss patterns indicative of an underlying thyroid condition [8, 14]. Therefore, integrating a thorough physical examination with careful evaluation of laboratory results is crucial for improving diagnostic accuracy in thyroid disorders, ensuring patients receive timely and appropriate care. The patient's prolonged symptoms and multiple unnecessary emergency room visits could have been avoided had a comprehensive physical examination been conducted during her initial or subsequent visits, leading to the correct sequence of diagnostic tests. A detailed physical examination would have provided valuable diagnostic clues such as thyroid tenderness, guiding the appropriate laboratory evaluations, and preventing the misdiagnosis that contributed to her ongoing clinical symptoms.

The diagnosis of SAT is based on clinical presentation, laboratory findings, and imaging studies. The initial laboratory tests often reveal elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), which are general markers of inflammation [2]. Thyroid function tests generally reveal elevated free T4 and T3 levels during the hyperthyroid phase, which subsequently decline as the condition progresses into the hypothyroid phase. Thyroid-stimulating hormone (TSH) levels are initially suppressed and may later rise as hypothyroidism develops [11].

Imaging studies play a crucial role in differentiating SAT from other thyroid disorders. Thyroid ultrasound typically reveals diffusely enlarged and hypoechoic thyroid lobes with decreased blood flow, a hallmark finding in SAT [17]. Additionally, radionuclide scans often demonstrate low radioiodine uptake, which helps distinguish SAT from conditions such as Graves' disease or toxic multinodular goiter, where uptake would be elevated [12]. In the present case, thyroid ultrasound confirmed an enlarged, hypoechoic thyroid gland with reduced vascularity, consistent with SAT. Given the presence of respiratory symptoms, a chest X-ray was performed, revealing clear lungs with no evidence of pulmonary infiltrations, nodules, or cavities, effectively ruling out infectious or pulmonary causes as contributors to the patient's systemic symptoms. These imaging findings, combined with laboratory results and clinical presentation, led to the final diagnosis of SAT.

The primary goal of treatment in SAT is to relieve pain and inflammation. Nonsteroidal antiinflammatory drugs (NSAIDs) are often the first-line treatment [7]. In cases where NSAIDs are ineffective, corticosteroids are highly effective in reducing inflammation and pain [15]. Beta-blockers may be prescribed to manage symptoms of hyperthyroidism [13]. In the hypothyroid phase, levothyroxine may be required to alleviate symptoms, although thyroid function typically normalizes within a few months [3]. It is essential to monitor thyroid function regularly to adjust treatment as needed. SAT is generally a self-limiting condition with a good prognosis. Most patients recover fully within 3–6 months [12]. However, recurrences are rare, but can occur, as a minority of patients may develop permanent hypothyroidism and require long-term thyroid hormone replacement therapy [11].

Conclusion

This case underscores the importance of a thorough physical examination in diagnosing subacute thyroiditis, which was delayed owing to reliance on symptoms, vital signs, and laboratory results without a comprehensive clinical assessment. Key diagnostic clues, such as thyroid tenderness, were overlooked, and the persistence of elevated blood pressure, fever, and systemic symptoms should have prompted earlier consideration of endocrine or inflammatory conditions. The misinterpretation of a positive urine culture in the absence of clear UTI symptoms contributed to a delayed and incorrect diagnosis, emphasizing the importance of correlating clinical findings with laboratory results to avoid unnecessary antibiotic treatments [10].

Despite the growing role of telemedicine in modern healthcare, in-person evaluations remain crucial for detecting subtle but significant clinical signs that may otherwise be missed [16]. This case highlights the need for clinicians to maintain a broad differential diagnosis and consider atypical presentations of SAT, particularly in patients with persistent, unexplained systemic symptoms [11, 12]. Early recognition through careful patient assessment and appropriate diagnostic tools can help prevent misdiagnoses, reduce unnecessary treatments, and improve patient outcomes.

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PN: conceptualization, data collection, and manuscript writing; ST: data analysis, manuscript review, and editing.

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Data availability
The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The patient provided informed consent, and a signed agreement is on file. A hard copy of the consent document is available upon request.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare no competing interests.

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