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Subacute (de Quervain) Thyroiditis Evolving Into Permanent Hypothyroidism in a Patient With Kikuchi-Fujimoto Disease: A Case Report With Four-Year Follow-Up

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Abstract

Subacute (de Quervain) thyroiditis is a painful, usually self-limiting inflammation of the thyroid that progresses from thyrotoxicosis to recovery. Kikuchi-Fujimoto disease (KFD) is a rare, benign necrotizing lymphadenitis. Both conditions involve cytotoxic T-cell injury and specific human leukocyte antigen (HLA) associations, yet their coexistence is rarely reported. We describe a 43-year-old woman with biopsy-proven KFD on maintenance colchicine who was started on hydroxychloroquine in August 2022 for suspected KFD activity and later that month developed fever, anterior neck pain with marked thyroid tenderness, dysphagia, palpitations, and weight loss. Thyroid-stimulating hormone (TSH) was 0.012 µIU/mL, free thyroxine 2.76 ng/dL, thyroglobulin 721 ng/mL, and erythrocyte sedimentation rate 91 mm/h; thyroid autoantibodies were negative, and Doppler ultrasonography showed an enlarged, hypovascular gland. After treatment with prednisone and atenolol, she entered symptomatic hypothyroidism (peak TSH 10.6 µIU/mL) and began levothyroxine 25 µg/day. Withdrawal of levothyroxine was planned in 2025 but not undertaken because hypothyroid symptoms recurred, and the dose was increased to 50 µg/day. Four years after onset, she remains euthyroid on levothyroxine replacement and without serological evidence of systemic lupus erythematosus. Thyroid autoantibodies were negative at the onset of thyroiditis. This case documents an uncommon overlap of the two diseases and shows that subacute thyroiditis may be followed by persistent hypothyroidism despite glucocorticoid treatment and negative thyroid autoantibodies at onset, supporting long-term surveillance of thyroid function.

Categories: Rheumatology, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: histiocytic necrotizing lymphadenitis, hla antigens, levothyroxine, permanent hypothyroidism, subacute thyroiditis

Introduction

Subacute thyroiditis, also called de Quervain, granulomatous, or painful thyroiditis, is the most common cause of a tender thyroid gland [1]. It usually follows an upper respiratory viral infection and presents with anterior neck pain that may radiate to the jaw or ears, fever, malaise, and a firm, tender goiter. Inflammatory destruction of the follicles releases stored hormone, which explains the transient thyrotoxicosis, the high serum thyroglobulin, and the low radioiodine uptake seen at onset. Over the following weeks to months, most patients pass through euthyroid and hypothyroid phases before thyroid function recovers [1,2]. Recovery is not universal; however, in a population-based cohort, about 15% of patients remained hypothyroid in the long term [2], and persistent hypothyroidism is now recognized as less rare than previously assumed. The disease is uncommon and predominantly affects women in their fourth and fifth decades of life [2].

Its pathogenesis is thought to involve presentation of viral antigens, or host antigens altered by infection, by human leukocyte antigen (HLA) class I molecules to cytotoxic T lymphocytes, which then damage follicular cells [3]; susceptibility has been linked mainly to HLA-B35 and, more recently, to additional class I and class II alleles [4,5].

Kikuchi-Fujimoto disease (KFD), first described in Japan in 1972, is a histiocytic necrotizing lymphadenitis that mainly affects young women and typically presents with tender cervical lymphadenopathy and low-grade fever [6,7]. Fatigue, rash, arthralgia, leukopenia, and raised inflammatory markers may accompany the picture, which overlaps with tuberculosis, lymphoma, and systemic lupus erythematosus (SLE); for this reason, excisional lymph node biopsy remains the diagnostic standard [7,8]. Histologically, the necrotic foci reflect apoptosis driven by CD8-positive cytotoxic T cells and plasmacytoid dendritic cells [7]. The illness usually resolves within a few months, but it can recur, and some patients later develop SLE or other autoimmune diseases [8,9]. Class II alleles, particularly HLA-DPA1 and HLA-DPB1, have been associated

with KFD in Japanese patients [10].

Autoimmune thyroid disease, mostly Hashimoto thyroiditis, has occasionally been reported alongside KFD [11,12]. Painful subacute thyroiditis arising in a patient with established KFD, however, is rarely documented. We report such a patient, followed for four years, whose thyroiditis did not end in recovery but in persistent hypothyroidism requiring levothyroxine, despite negative thyroid autoantibodies at onset and glucocorticoid treatment in the acute phase.

Case Presentation

A 43-year-old Brazilian woman, a homemaker with no known comorbidities and an unremarkable family history, was referred in June 2021 from the infectious diseases clinic to the rheumatology clinic of our hospital. She had a long history of painful lateral cervical lymphadenopathy with recorded fevers of 37.8°C-38.0°C. The episodes had started in 1996 and alternated with periods of remission, without any identifiable trigger. Fine-needle aspiration of cervical nodes in 2002 and again in 2007 showed reactive lymphadenitis without malignant cells. An excisional biopsy performed in 2012 revealed lymph node tissue with extensive, confluent necrosis and no granulomatous reaction; the immunohistochemical profile favored histiocytic necrotizing lymphadenitis, and stains for acid-fast bacilli and fungi were negative. On this basis, a diagnosis of KFD had been established.

At the first rheumatology visit, antinuclear antibodies (ANA) and the remaining lupus serologies were negative, and she denied skin or mucosal lesions, arthralgia, serositis, thrombosis, and gastrointestinal or genitourinary complaints. Because the nodes were still tender and fevers recurred, she was treated with prednisone 20 mg/day, tapered gradually and then stopped. Colchicine 0.5 mg/day was added in January 2022 as maintenance therapy. Contrast-enhanced computed tomography of the neck in July 2021 showed no enlarged nodes, and thyroid function was normal (thyroid-stimulating hormone (TSH) 2.6 µIU/mL; free thyroxine (FT4) 0.76 ng/dL). A neck ultrasound in May 2022 showed three small nodes, the largest measuring 1.1 × 0.3 cm, and, as an incidental finding, a thyroid at the lower limit of normal size (5.9 cm^3) with finely heterogeneous echotexture; vascularity was not described. She remained clinically stable between June 2021 and July 2022.

Colchicine 0.5 mg/day had been used as maintenance therapy since January 2022. In early August 2022, after approximately two weeks of low-grade fever, fatigue, weakness, and prostration, recurrence or increased activity of KFD was suspected, and hydroxychloroquine 200 mg/day, a drug with reported benefit in KFD [13], was initiated. Later that month, the clinical picture evolved with palpitations, tachycardia, hand tremor, weight loss, and a new pattern of neck pain. Unlike her previous KFD-related symptoms, the pain was predominantly anterior rather than lateral, the thyroid area was exquisitely tender to palpation, and she reported proximal dysphagia for the first time, which prompted investigation for an alternative diagnosis in the emergency department. Complete blood count and renal function were normal, whereas C-reactive protein (CRP) was 9.74 mg/dL (reference <0.1 mg/dL). Neck ultrasonography with Doppler showed an enlarged gland (15.8 cm^3; reference 6-14 cm^3) with diffusely heterogeneous echotexture, together with scattered bilateral cervical lymph nodes measuring up to 1.5 × 0.6 cm (Figure *1*). The written report described no abnormal flow, but the stored color Doppler images showed reduced intraglandular vascularity (Figure *2*). The radiologist suggested thyroiditis.

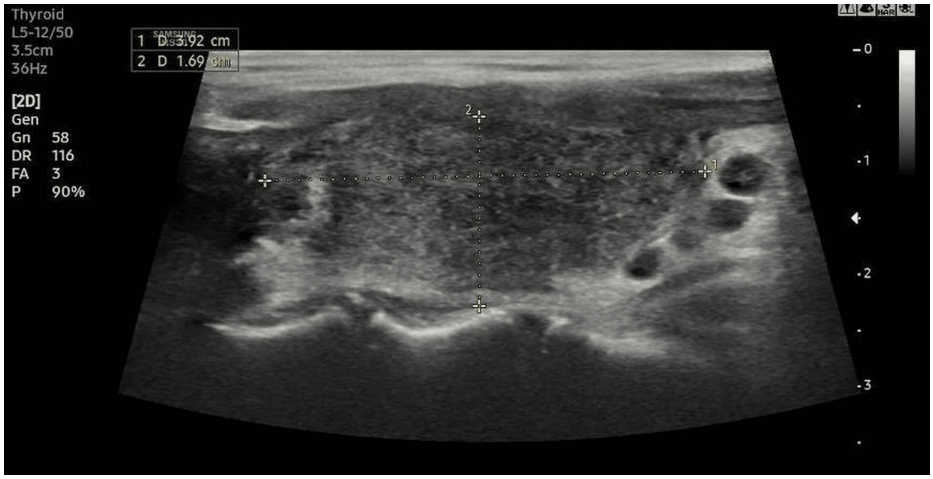


FIGURE 1: Gray-scale ultrasonography of the right thyroid lobe in August 2022 (thyrotoxic phase)

Enlarged right lobe (3.92 × 1.69 cm in this section) with diffusely heterogeneous echotexture.

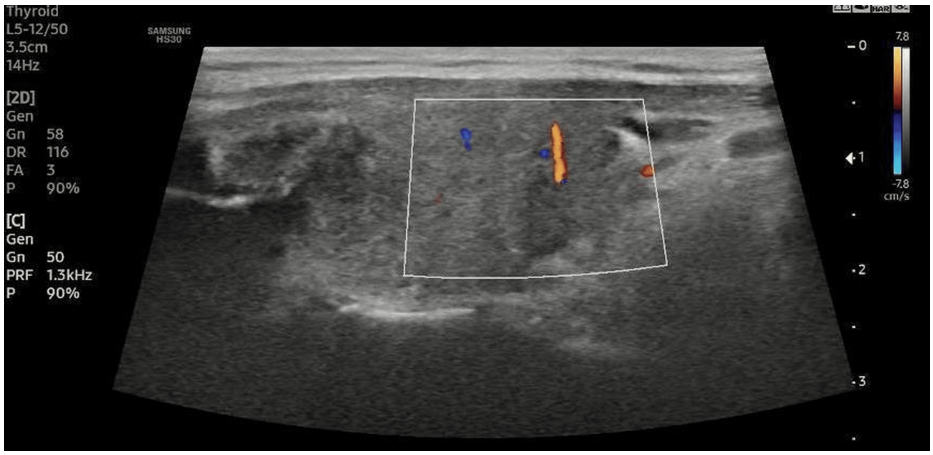


FIGURE 2: Color Doppler ultrasonography of the right thyroid lobe in August 2022

Reduced intraglandular vascularity during the thyrotoxic phase.

She denied a recent respiratory or other infection, vaccination, or other newly introduced medications apart from hydroxychloroquine, which had been started earlier that month because of suspected KFD activity. Tests obtained five days later confirmed thyrotoxicosis: TSH 0.012 µIU/mL (reference 0.35-4.94), FT4 2.76 ng/dL (reference 0.70-1.48), thyroglobulin 721 ng/mL (reference 3.5-77), erythrocyte sedimentation rate (ESR) 91 mm/h, and CRP 10.35 mg/dL. Antibodies against thyroid peroxidase, thyroglobulin, and the TSH receptor were all negative. A painful goiter, destructive-pattern thyrotoxicosis with very high inflammatory markers, negative TSH-receptor antibodies, and reduced glandular flow pointed to subacute thyroiditis rather than Graves disease [14]. She was treated with prednisone 10 mg/day for 21 days, followed by gradual tapering to 5 mg/day and then 2.5 mg/day over the subsequent months. Atenolol 25 mg twice daily was prescribed for 21 days and subsequently discontinued after improvement of the thyrotoxic symptoms. Although the clinical and biochemical findings supported subacute thyroiditis as the main explanation for the acute presentation, hydroxychloroquine was continued because concomitant KFD activity could not be completely excluded. She was referred to the endocrinology clinic.

By mid-September 2022, her symptoms had subsided. FT4 had returned to normal (0.87 ng/dL) while TSH remained suppressed (0.02 µIU/mL); CRP had fallen to 0.06 mg/dL, although ESR was still 78 mm/h. At her first endocrinology visit, about two months after onset, she was asymptomatic and was diagnosed with subacute thyroiditis in its transitional euthyroid phase. Ultrasonography in October 2022 showed a normal-sized gland (6.1 cm^3) with diffusely heterogeneous echotexture and multiple ill-defined, confluent

hypoechoic areas without nodular configuration (Figure 3), with peripheral and central vascularity of normal appearance (Figure 4). A 0.6 × 0.6 × 0.5 cm solid hyperechoic nodule with regular margins was noted in the upper third of the right lobe (Thyroid Imaging Reporting and Data System (TI-RADS) 3), and the cervical lymph nodes were no longer enlarged. The rheumatology team found no evidence of a KFD flare.

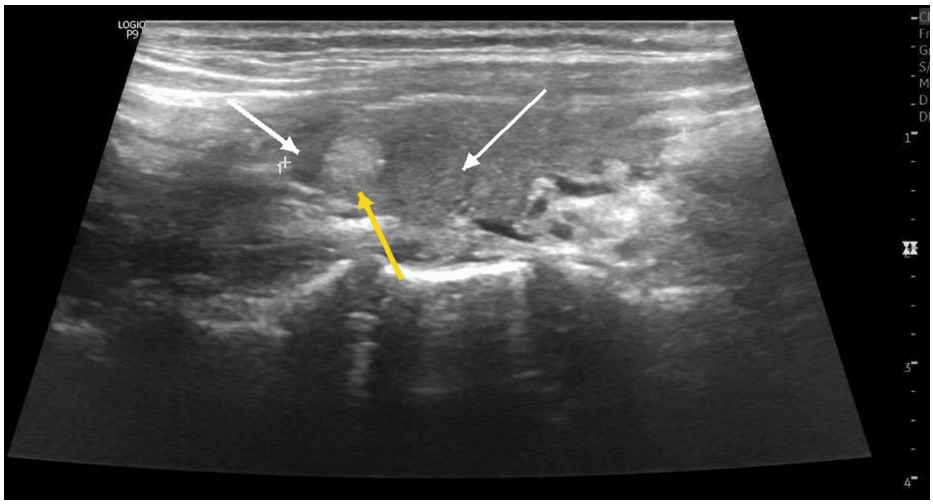


FIGURE 3: Gray-scale ultrasonography of the right thyroid lobe in October 2022

Normal-sized lobe with ill-defined, confluent hypoechoic areas. White arrows: ill-defined hypoechoic areas of thyroiditis; yellow arrow: small hyperechoic nodule (TI-RADS 3).

TI-RADS: Thyroid Imaging Reporting and Data System

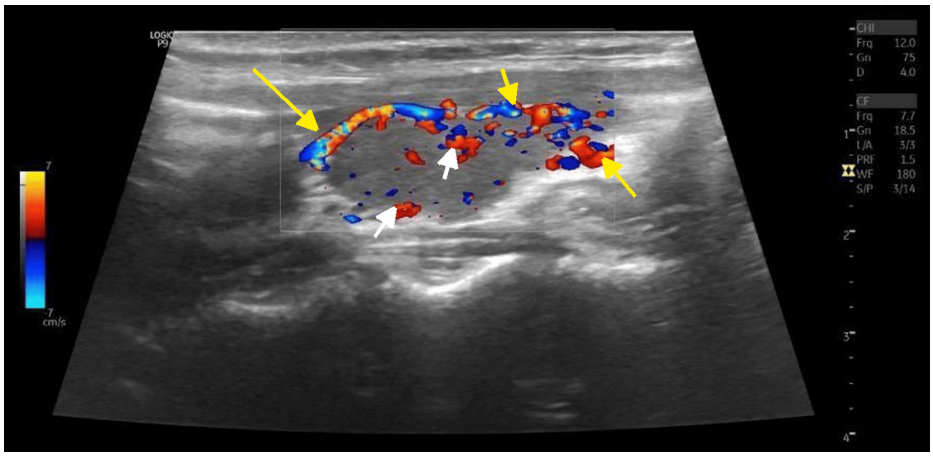


FIGURE 4: Color Doppler ultrasonography of the right thyroid lobe in October 2022

Restored peripheral and central vascularity. White arrow: thyroid parenchyma; yellow arrow: intraglandular vascular flow. Because the examinations were performed with different equipment and settings, the comparison of vascularity with Figure 2 is qualitative.

In November 2022, FT4 fell slightly below the reference range (0.68 ng/dL) and ESR normalized (16 mm/h). By December 2022, TSH had risen to 10.6 µIU/mL with FT4 of 0.70 ng/dL. She complained of intense asthenia, fatigue, weight gain, brittle nails, and hair loss that had begun about one month earlier. Levothyroxine 25 µg/day was started in January 2023. Symptoms improved, and thyroid function approached normal over the following months (TSH 5.3 µIU/mL in March 2023 and 4.1 µIU/mL in June 2023), with persistently low inflammatory markers. Ultrasonography in December 2023 showed a slightly small gland (5.0 cm^3) with persistent diffuse heterogeneity, predominantly peripheral vascularity, and no lymphadenopathy.

Throughout 2024, still on 25 µg/day, she reported residual hair loss and brittle nails, and TSH ranged from 3.26 to 5.41 µIU/mL with normal FT4 (Table *1*). In February 2025, while receiving levothyroxine 25 µg/day with satisfactory symptom control, gradual withdrawal of replacement therapy was being considered for a subsequent visit, given the possibility that the hypothyroid phase after subacute thyroiditis had been transient. In March 2025, an isolated increase in ESR to 50 mm/h was observed, with CRP of 0.4 mg/dL and normal thyroid function (TSH 2.10 µIU/mL; FT4 1.19 ng/dL). No clear clinical correlate was identified, and the ESR returned to 15 mm/h within five weeks without specific intervention. However, at follow-up in November 2025, before withdrawal had been undertaken, the patient reported recurrent fatigue, asthenia, prostration, and brittle nails. At that time, TSH (1.78 µIU/mL) and FT4 (1.11 ng/dL) remained within the reference range while she was still receiving levothyroxine 25 µg/day. Because of the recurrence of symptoms, withdrawal was not pursued, and the levothyroxine dose was increased to 50 µg/day. Levothyroxine has therefore never been discontinued, and thyroid function off replacement has not been assessed. In September 2026, four years after the onset of thyroiditis, she was asymptomatic, with TSH 1.63 µIU/mL, FT4 1.15 ng/dL, ESR 7 mm/h, and CRP 0.3 mg/dL. Repeat screening for SLE in June 2026 was again negative (non-reactive ANA, negative anti-double-stranded DNA, and normal complement levels). Her current treatment consists of levothyroxine 50 µg/day for post-thyroiditis hypothyroidism, together with colchicine 0.5 mg/day and hydroxychloroquine 200 mg/day for KFD, which remains in remission. The biochemical course is summarized in Table *1* and Figure *5*.

Date	TSH (µIU/mL)	FT4 (ng/dL)	ESR (mm/h)	CRP (mg/dL)	Clinical phase and treatment
Jun 2021	2.60	0.76	30	-	KFD follow-up; euthyroid
Aug 2022	0.012	2.76	91	10.35	Thyrotoxic phase; prednisone and atenolol
Sep 2022	0.02	0.87	78	0.06	Transitional euthyroid phase; prednisone taper
Nov 2022	-	0.68	16	-	Onset of hypothyroid phase
Dec 2022	10.6	0.70	7	0.07	Symptomatic hypothyroidism; LT4 25 µg/day from Jan 2023
Mar 2023	5.30	0.84	12	0.16	LT4 25 µg/day
Jun 2023	4.10	0.88	6	0.11	LT4 25 µg/day
Feb 2024	4.23	0.87	8	0.09	LT4 25 µg/day
Aug 2024	5.41	1.02	-	-	LT4 25 µg/day
Mar 2025	2.10	1.19	50	0.40	LT4 25 µg/day; isolated ESR rise; normal thyroid function
Nov 2025	1.78	1.11	11	0.10	LT4 25 µg/day; recurrent symptoms; dose increased to 50 µg/day
Jun 2026	4.16	1.17	12	0.20	LT4 50 µg/day; SLE serologies negative
Sep 2026	1.63	1.15	7	0.30	Asymptomatic on LT4 50 µg/day

TABLE 1: Thyroid function and inflammatory markers from baseline to the last follow-up

Reference ranges: TSH 0.35-4.94 µIU/mL; FT4 0.70-1.48 ng/dL; ESR 0-30 mm/h; CRP <0.1 mg/dL. Thyroglobulin in August 2022 was 721 ng/mL (reference 3.5-77). LT4 withdrawal, considered in February 2025, was not undertaken because hypothyroid symptoms recurred; the dose was increased to 50 µg/day in November 2025.

TSH: thyroid-stimulating hormone; FT4: free thyroxine; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; KFD: Kikuchi-Fujimoto disease; LT4: levothyroxine; SLE: systemic lupus erythematosus

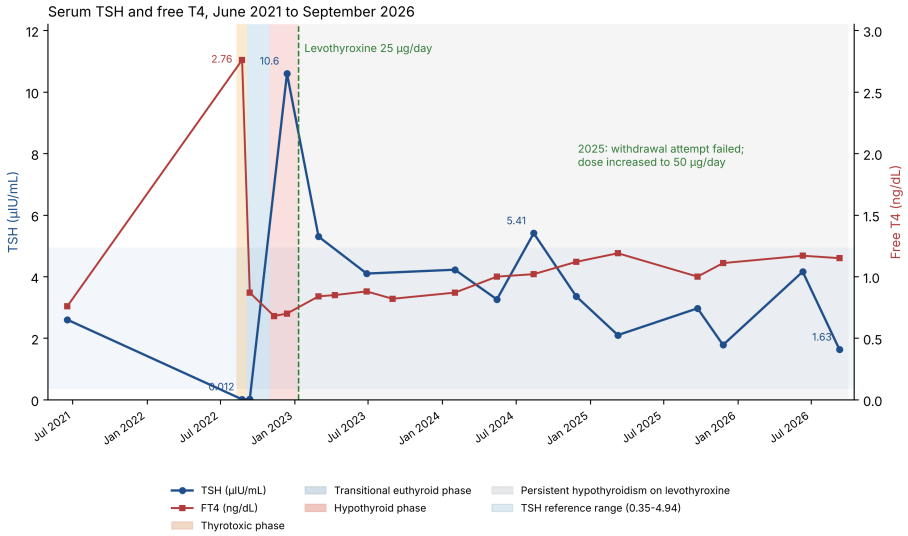


FIGURE 5: Course of serum TSH and FT4 from June 2021 to September 2026

Phases of subacute thyroiditis and levothyroxine doses over the follow-up period.

TSH: thyroid-stimulating hormone; FT4: free thyroxine

Discussion

The early course of thyroid dysfunction in this patient was classic for subacute thyroiditis; its outcome was not. A painful goiter, thyrotoxicosis with a thyroglobulin almost 10 times the upper reference limit, an ESR above 90 mm/h, absent thyroid autoantibodies, and a hypovascular gland leave little room for alternative diagnoses [1,14]. Graves disease would be expected to show increased glandular flow and positive TSH-receptor antibodies, and painless thyroiditis does not produce this degree of pain or systemic inflammation [14]. The absence of a recognizable viral prodrome does not weaken the diagnosis, because many patients cannot recall a preceding infection [3]. The prior history of KFD was the main source of diagnostic uncertainty: fever and constitutional symptoms initially suggested KFD activity and led to the introduction of hydroxychloroquine, and it was the change in the site of pain, thyroid tenderness, and new dysphagia that redirected the investigation.

The ultrasound sequence is instructive. The images from August 2022 showed reduced flow even though the written report did not mention it, and the October 2022 study showed confluent, ill-defined hypoechoic areas with returning vascularity. Because the two examinations were obtained with different equipment and settings, this comparison is qualitative. These changes follow the inflammatory activity of the disease, and their extent appears to be partly determined by the patient's HLA background [15]. Reading the Doppler images directly, rather than relying on the report alone, can help separate destructive thyrotoxicosis from Graves disease without isotopic studies [14].

Persistent hypothyroidism after subacute thyroiditis was long considered uncommon, but reported rates range from 14.3% in a Saudi cohort [16] to 31.4% in a recent Turkish series [17], depending on the definition used, the treatment given, and the duration of follow-up. Our patient has required levothyroxine for almost four years, and the recurrence of hypothyroid symptoms in November 2025 led to a dose increase rather than to the planned withdrawal. Because replacement was never discontinued and thyroid function off levothyroxine was not documented, we describe her hypothyroidism as persistent rather than definitively permanent. One feature previously associated with persistent hypothyroidism in observational studies was present: a relatively high peak TSH during the hypothyroid phase. Her TSH reached 10.6 µIU/mL, and Gokkaya and Aydin Tezcan reported that higher early maximum TSH values, above roughly 7-9 µIU/mL, were associated with subsequent permanent hypothyroidism [17]. However, this association is observational and does not establish that the elevated TSH causally determined the long-term outcome in this patient. Anti-thyroid peroxidase positivity, another recognized risk factor [18], was absent at onset, although thyroid autoantibodies were not repeated during follow-up.

The thyroid gland documented before the thyroiditis also deserves attention. In May 2022, about three months before onset and with normal thyroid function, ultrasonography showed a relatively small gland, at the lower limit of normal size (5.9 cm^3), with finely heterogeneous echotexture. These findings raise the

possibility of a pre-existing structural thyroid abnormality or reduced functional reserve. Negative thyroid autoantibodies at onset make overt autoimmune thyroiditis less likely, but antibody negativity alone does not fully exclude an underlying thyroid disorder, and no cytology was available to characterize the gland. Limited baseline reserve may therefore have contributed to the subsequent persistent hypothyroid state, which cannot be attributed exclusively to subacute thyroiditis. Interestingly, persistent hypothyroidism developed despite glucocorticoid therapy during the acute phase. Observational studies and a 2024 meta-analysis of 10 cohorts with 1,337 patients have suggested a lower risk of persistent hypothyroidism with glucocorticoids than with non-steroidal anti-inflammatory drugs (NSAIDs) [18,19]; Sencar et al., for example, observed persistent hypothyroidism in 22.8% of patients treated only with ibuprofen compared with 6.6% of those treated only with methylprednisolone [18]. Such data do not imply complete protection against long-term thyroid dysfunction, and our patient received a relatively low dose of prednisone (10 mg/day for 21 days, followed by gradual tapering). Her course illustrates that persistent hypothyroidism may still occur after glucocorticoid-treated subacute thyroiditis. The relatively high peak TSH and the possibility of reduced baseline thyroid reserve may have contributed to the long-term requirement for levothyroxine, although their individual causal roles cannot be established. Her course also shows that thyroid function should be followed beyond the expected hypothyroid phase and that any tapering of levothyroxine should be monitored closely.

The coexistence of the two diseases raises the question of a shared mechanism. Both involve antigen presentation that leads to cell death mediated by CD8-positive cytotoxic T lymphocytes [3,7]. In active KFD, high serum concentrations of interferon-gamma and interleukin-6 have been measured [20], cytokines that also take part in the inflammatory cascade of subacute thyroiditis [3]. The genetic susceptibility, however, appears to involve different loci: HLA class I alleles (B35, B*18:01, and C*04:01), together with DRB1*01, in subacute thyroiditis [4,5], and class II DPA1 and DPB1 alleles in KFD, in which no significant association with DRB1 was found [10]. To our knowledge, no shared HLA allele has been demonstrated. Thyroid involvement previously described in KFD has been predominantly autoimmune, in the form of Hashimoto thyroiditis [11,12], whereas our patient developed a destructive, antibody-negative thyroiditis. A common immunological predisposition is therefore plausible but unproven, and a chance association between two uncommon conditions remains possible.

Long-term follow-up also mattered for KFD itself, since a proportion of patients develop SLE, sometimes years after the lymphadenitis [8,9]. In our patient, repeated serological screening, most recently in June 2026, has remained negative, and the disease has stayed quiescent on colchicine and hydroxychloroquine.

This report has several limitations. Data were collected retrospectively from medical records. HLA typing, thyroid uptake studies, and thyroid cytology were not performed, and thyroid autoantibodies were not repeated during follow-up. The absence of a thyroid uptake study limits the objective exclusion of other causes of thyrotoxicosis, although the clinical presentation, inflammatory markers, negative thyroid autoantibodies, elevated thyroglobulin, and reduced intraglandular vascularity strongly supported subacute thyroiditis. In addition, hydroxychloroquine was initiated shortly before the definitive diagnosis of subacute thyroiditis because KFD activity was initially suspected, and its introduction near the onset of symptoms represents a potential confounder in interpreting the early clinical course. Hypovascularity in the acute phase was inferred from stored Doppler images rather than described in the original report, and comparison of vascularity between examinations performed with different equipment is qualitative. Because levothyroxine was never withdrawn, the permanence of hypothyroidism has not been formally demonstrated. As with any single case, causality between the two diseases cannot be established.

Conclusions

Subacute thyroiditis can develop in a patient with KFD and may initially be mistaken for a flare of the lymphadenitis; a change in the site of neck pain, thyroid tenderness, and new dysphagia should prompt evaluation of thyroid function. Although subacute thyroiditis is classically self-limiting, this case shows that it may be followed by persistent hypothyroidism despite negative thyroid autoantibodies at the onset of thyroiditis and glucocorticoid treatment in the acute phase. A high peak TSH has been associated with persistent hypothyroidism, and a small, heterogeneous gland before the episode may indicate reduced thyroid reserve, but a single case cannot establish the causal role of either feature. Thyroid function should therefore be monitored beyond the expected recovery period, and any levothyroxine withdrawal should be supervised. Whether the two diseases share an immunogenetic basis remains an open question that HLA typing in future cases could help answer.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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References

1. Pearce EN, Farwell AP, Braverman LE: Thyroiditis. N Engl J Med. 2003, 348:2646-55. 10.1056/NEJMra021194
2. Fatourechi V, Aniszewski JP, Fatourechi GZ, Atkinson EJ, Jacobsen SJ: Clinical features and outcome of subacute thyroiditis in an incidence cohort: Olmsted County, Minnesota, study. J Clin Endocrinol Metab. 2003, 88:2100-5. 10.1210/jc.2002-021799
3. Stasiak M, Lewiński A: New aspects in the pathogenesis and management of subacute thyroiditis . Rev Endocr Metab Disord. 2021, 22:1027-39. 10.1007/s11154-021-09648-y
4. Ohsako N, Tamai H, Sudo T, et al.: Clinical characteristics of subacute thyroiditis classified according to human leukocyte antigen typing. J Clin Endocrinol Metab. 1995, 80:3653-6. 10.1210/jcem.80.12.8530615
5. Stasiak M, Tymoniuk B, Michalak R, Stasiak B, Kowalski ML, Lewiński A: Subacute thyroiditis is associated with HLA-B*18:01, -DRB1*01 and -C*04:01-the significance of the new molecular background. J Clin Med. 2020, 9:534. 10.3390/jcm9020534
6. Bosch X, Guilabert A, Miquel R, Campo E: Enigmatic Kikuchi-Fujimoto disease: a comprehensive review. Am J Clin Pathol. 2004, 122:141-52. 10.1309/YF08-1L4T-KYWV-YVPQ
7. Perry AM, Choi SM: Kikuchi-Fujimoto disease: a review. Arch Pathol Lab Med. 2018, 142:1341-6. 10.5858/arpa.2018-0219-RA
8. Kucukardali Y, Solmazgul E, Kunter E, Oncul O, Yildirim S, Kaplan M: Kikuchi-Fujimoto disease: analysis of 244 cases. Clin Rheumatol. 2007, 26:50-4. 10.1007/s10067-006-0230-5
9. Dumas G, Prendki V, Haroche J, et al.: Kikuchi-Fujimoto disease: retrospective study of 91 cases and review of the literature. Medicine (Baltimore). 2014, 93:372-82. 10.1097/MD.0000000000000220
10. Tanaka T, Ohmori M, Yasunaga S, Ohshima K, Kikuchi M, Sasazuki T: DNA typing of HLA class II genes (HLA-DR, -DQ and -DP) in Japanese patients with histiocytic necrotizing lymphadenitis (Kikuchi's disease). Tissue Antigens. 1999, 54:246-53. 10.1034/j.1399-0039.1999.540305.x
11. Lee EJ, Lee HS, Park JE, Hwang JS: Association Kikuchi disease with Hashimoto thyroiditis: a case report and literature review. Ann Pediatr Endocrinol Metab. 2018, 23:99-102. 10.6065/apem.2018.23.2.99
12. Go EJ, Jung YJ, Han SB, Suh BK, Kang JH: A case of Kikuchi-Fujimoto disease with autoimmune thyroiditis . Korean J Pediatr. 2012, 55:445-8. 10.3345/kjp.2012.55.11.445
13. Rezai K, Kuchipudi S, Chundi V, Ariga R, Loew J, Sha BE: Kikuchi-Fujimoto disease: hydroxychloroquine as a treatment. Clin Infect Dis. 2004, 39:e124-6. 10.1086/426144
14. Ross DS, Burch HB, Cooper DS, et al.: 2016 American Thyroid Association guidelines for diagnosis and management of hyperthyroidism and other causes of thyrotoxicosis. Thyroid. 2016, 26:1343-421. 10.1089/thy.2016.0229
15. Stasiak M, Tymoniuk B, Adamczewski Z, Stasiak B, Lewiński A: Sonographic pattern of subacute thyroiditis is HLA-dependent. Front Endocrinol (Lausanne). 2019, 10:3. 10.3389/fendo.2019.00003
16. Alfadda AA, Sallam RM, Elawad GE, Aldhukair H, Alyahya MM: Subacute thyroiditis: clinical presentation and long term outcome. Int J Endocrinol. 2014, 2014:794943. 10.1155/2014/794943
17. Gokkaya N, Aydin Tezcan K: The effects of corticosteroid and nonsteroid anti-inflammatory therapies on permanent hypothyroidism occurring after the subacute thyroiditis. Endocr Res. 2024, 49:137-44. 10.1080/07435800.2024.2344719
18. Sencar ME, Calapkulu M, Sakiz D, et al.: An evaluation of the results of the steroid and non-steroidal anti-inflammatory drug treatments in subacute thyroiditis in relation to persistent hypothyroidism and recurrence. Sci Rep. 2019, 9:16899. 10.1038/s41598-019-53475-w
19. Yuan A, Wu J, Huang H: Comparison of treatment outcome between glucocorticoids and non-steroidal anti-inflammatory drugs in subacute thyroiditis patients-a systematic review and meta-analysis. Front Endocrinol (Lausanne). 2024, 15:1384365. 10.3389/fendo.2024.1384365
20. Kubota M, Tsukamoto R, Kurokawa K, Imai T, Furusho K: Elevated serum interferon gamma and interleukin-6 in patients with necrotizing lymphadenitis (Kikuchi's disease). Br J Haematol. 1996, 95:613-5. 10.1046/j.1365-2141.1996.d01-1964.x