A Difficult Decision—Hashimoto’s Thyroiditis or Subacute Thyroiditis?
Zor Bir Karar - Hashimoto Tiroiditi mi Yoksa Subakut Tiroiditi mi?

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Abstract
Subacute thyroiditis generally presents with pain in the thyroid region, fever, thyrotoxicosis and elevated markers of acute inflammation. On the other hand, Hashimoto's thyroiditis is most frequently diagnosed in the hypothyroid phase with positive antithyroid autoantibodies. Here, we describe the case of a patient who presented with clinical features of subacute thyroiditis but laboratory and pathological features compatible with Hashimoto's thyroiditis. The patient was finally diagnosed with painful Hashimoto's thyroiditis. Türk Jem 2011; 15: 125-7

Key words: Hashimoto's thyroiditis, painful thyroiditis, thyroiditis

Özet

Anahtar kelimeler: Hashimoto tiroiditi, ağrılı tiroidit, tiroidit

Introduction
The typical presentation of patients with Hashimoto’s thyroiditis (HT) includes goiter and/or hypothyroidism (1). Additionally, as HT lies in the spectrum of thyroid diseases with autoimmune origin, thyroid autoantibodies, such as anti-thyroglobulin (anti-Tg) and anti-thyroidperoxidase (anti-TPO) are found to be elevated. Pain and tenderness in the thyroid region are rare, and when present should suggest subacute thyroiditis (SAT) in the first place (2). Besides these, fever, elevated serum markers of acute inflammation, and decreased radioactive iodine uptake on thyroid scan support the diagnosis of SAT. Here, we report a female patient who presented with features of both HT and SAT but was finally diagnosed with painful HT.

Case Report
A 53-year-old woman was seen in the otolaryngology outpatient clinic for fever, anterior neck pain radiating to her chin and ears, and difficulty in swallowing. She had these complaints for one week. The history of the patient revealed that, she was a non-smoker, had no known chronic disease and was not on any drugs. Her temperature varied between 37 °C and 38 °C. On physical examination, her thyroid gland was very firm and tender and diffusely enlarged, suggesting grade 1 goiter. On laboratory examination, complete blood count revealed a mild normochromic-normocytic anemia (Hb: 10.5 g/dl, Hct: 33.1%) with normal platelet and leukocyte counts. Liver, kidney and thyroid function tests were normal. Regarding markers of acute inflammation, her C reactive protein (CRP) level was high [34.1 (range: 0-10) mg/l] (Table 1). Thyroid autoantibodies were positive [anti-Tg: >3000 (normal: <115) IU/ml; anti-TPO: 66.18 (normal: <34) IU/ml] and plasma thyroglobulin level was within normal range. Thyroid ultrasonography revealed a heterogenous parenchyma with ill-defined hypoechoic regions. Parenchymal fine needle aspiration biopsy showed dense lymphocytic infiltration; accordingly, the diagnosis of HT was established. The
A patient was referred to endocrinology outpatient clinic for further investigation. On her repeat history and physical examination, there was nothing suggestive of an infectious focus. Her high thyroid autoantibody levels and biopsy results were compatible with HT, however, tenderness of thyroid on palpation, fever and high CRP levels suggested SAT. Since the patient was euthyroid and thyroglobulin levels were within normal limits, she was given just anti-inflammatory drug treatment and was called for a control visit twenty days later. On the control visit, she reported that her fever had continued for six more days and than subsided. Although much less compared to her initial presentation, she still had some difficulty in swallowing and, on physical examination there was still mild tenderness on thyroid palpation. On repeat laboratory testing, blood leukocyte count was normal, erythrocyte sedimentation rate (ESR) and CRP were high [61 (normal: <20] mm/hour and 58.1 (normal: 0-10) mg/l, respectively]. On thyroid function tests, she was found to be thyrotoxic [free T3 (fT3): 4.61 (2-4.4) pg/ml, free T4 (fT4): 2.58 (0.93-1.7) ng/dl, TSH: 0.01 (0.27-4.2) μIU/ml]. There was no uptake on Tc99m thyroid scan. The patient did not accept corticosteroid therapy and was again given nonsteroidal anti-inflammatory drug treatment with an arrangement for a control visit one month later. On the control visit, her TSH level was 4.84 μIU/ml and after another twenty days, she developed overt hypothyroidism with relief of local symptoms in the thyroidal region (fT3: 1.55 pg/ml, fT4: 0.39 ng/dl, TSH: 51.14 μIU/ml) (Table 1). L-thyroxine replacement therapy was started and she is under follow-up with a daily L-thyroxine dose of 50 μg/day.

### Discussion

Here, we described a female patient with fever, painful and tender goiter, elevated markers of acute inflammation, elevated anti-Tg and anti-TPO antibodies, transient thyrotoxicosis followed by hypothyroidism, nil uptake on thyroid scan during the thyrotoxic phase, and a thyroid fine needle aspiration biopsy showing lymphocytic infiltrate, who was diagnosed with painful HT. There are a few case series in the literature about painful HT which include patients presenting with some of the clinical and laboratory features of SAT, similar to our patient (1-5). Pain and tenderness of the thyroid are most commonly caused by SAT but less frequently caused by haemorrhage into a thyroid cyst, primary or metastatic thyroid malignancy, neck abscess, Riedel’s thyroiditis, radiation thyroiditis and HT (1-4,6). In the differential diagnosis of SAT and painful HT, some clinical and laboratory findings may be helpful. History of a preceding upper respiratory tract infection is frequent in SAT but infrequent in painful HT (2,5). In terms of thyroid autoantibody profile, high anti-Tg concentrations are noted in 80% to 90% (7), and high anti-TPO antibodies in nearly all (>90%) patients with HT (8), while thyroid autoantibodies are either negative or positive at low titers, in SAT (2,4,5). In the thyrotoxic period, patients with SAT nearly always have reduced radioiodine uptake, however, patients with painful HT may have variable uptake on thyroid scan (1,2,4,5,9).
Fine needle aspiration biopsy also may be quite useful in the differential diagnosis. Lymphocytic infiltration, differing degrees of fibrosis and follicular cell destruction are typical of HT, while granulomatous changes and giant multinucleated cells are diagnostic for SAT (5). Moreover, patients with SAT may respond dramatically to steroids, whereas those with painful HT may have only temporary or no benefit at all (1). Thus, some patients with painful HT may need thyroidectomy due to unremitting pain (1). The reason of pain in this small subset of HT is currently not known and Zimmerman and colleagues have noted no significant difference in terms of pathology between painful and non-painful HT (3). Finally, the diagnosis also may be revealed in the follow-up period. While risk of developing permanent hypothyroidism is high in HT, the rate is around 5% in SAT (10).

Conclusions

As a conclusion, overlapping clinical and laboratory features may occur between painful HT and SAT. Hence, for precise understanding and classification of thyroiditis, further clarification of the pathophysiology of these diseases is needed.