Should neck pain in a patient with Hashimoto's thyroiditis be underestimated? A case and review of the literature

Umut Mousa, Cuneyd Anil, Alptekin Gursoy
Department of Endocrinology and Metabolism, Başkent University Faculty of Medicine, Ankara, Turkey

ABSTRACT

Hashimoto's Thyroiditis (HT) is an autoimmune disease and the most frequent cause of hypothyroidism. Subacute thyroiditis (SAT) overlapping HT is a rare entity. A 69-year-old female patient with HT and multinodular goiter has been followed on levothyroxine replacement therapy for 7 years. She presented with neck pain radiating to the right ear persisting for 2 months. She was prescribed analgesics and antibiotics by other physicians during that period, which did not work. Her vital signs were stable with no tachycardia or fever. The right lobe of the thyroid gland was tender on palpation. Her TSH level was 3.94 mIU/ml, ESR 23 mm/h, CRP 3.2 mg/l, WBC 4900/µl at presentation. Thyroid ultrasonography revealed a hypoechoic area over the tender lobe. Power Doppler imaging revealed almost no blood flow in that area. She was started on methylprednisolone 32 mg/day. At day 10 of therapy, her symptoms had completely resolved. Ultrasonography repeated showed that the hypoechoic area had disappeared. Glucocorticoid dosage was tapered and stopped. Emergence of subacute thyroiditis in a case with preexisting Hashimoto's thyroiditis is a quite rare condition, but should be kept in mind along with a painful attack of HT in the differential diagnosis.

Key words: Acute exacerbation, de Quervain's, Hashimoto's thyroiditis, painful, subacute thyroiditis

Introduction

Hashimoto's Thyroiditis (HT) is an autoimmune disease and the most frequent cause of hypothyroidism.[1,2] Though most patients with HT have no pain, rare cases presenting with serious neck pain have been reported.[3,4] Leading pathologies that can present with neck pain include intranodular bleeding, subacute thyroiditis (SAT), and infectious pathologies of the neck and thyroid gland.[2-4] Here we present a HT case that developed SAT during her clinical follow-up.

Access this article online	
Quick Response Code:	Website: www.ijem.in
	DOI: 10.4103/2230-8210.95709

Case Report

A 69-year-old female patient with HT and multinodular goiter has been followed up in the outpatient clinic of an Endocrinology department since 2003. She was positive for antithyroid peroxidase and negative for antithyroglobulin antibodies. Her first ultrasonography revealed a heterogeneous parenchyma compatible with chronic thyroiditis and coincidental multiple isoechoic thyroid nodules in the left lobe. The largest nodule was measured as 15 × 9 mm and was located in the left lobe. A fine needle aspiration biopsy was performed from the largest nodule and was reported as being compatible with HT. She was on levothyroxine replacement therapy since the diagnosis. The patient presented to our department with a symptom of neck pain radiating to the right ear and neck persisting for 2 months. She was prescribed analgesics and antibiotics by other physicians during the 2 months period, but the pain persisted. She described some symptoms of a possible upper respiratory tract infection including fever

Corresponding Author: Dr. Umut Mousa, Department of Endocrinology and Metabolism, Baskent University Hospital, 5. Sok. No: 48, Bahcelievler Ankara, Turkey. E-mail: umutmousa@yahoo.co.uk

which began prior to the presenting symptom. Her vital signs were stable with no tachycardia or fever. The right lobe of the thyroid gland was tender on palpation. Her TSH level was 3.94 mIU/ml (0.35--4.94), ESR 23 mm/h, CRP 3.2 mg/l, WBC 4900/µl. Thyroid ultrasonography revealed that the right lobe volume was 28.5 ml, the left lobe volume was 12.0 ml. The thyroid parenchyma was heterogeneous and a hypoechoic area was noticed over the tender lobe [Figure 1]. Power Doppler imaging (PDI) revealed almost no blood flow in that area [Figure 2]. She was started on methylprednisolone 32 mg/day. At day 10 of therapy, her symptoms had completely resolved. There was no tenderness on the thyroid gland by palpation. Repeated ultrasonography showed that the hypoechoic area had disappeared and the right lobe volume had decreased to 9.5 ml [Figures 3 and 4]. Glucocorticoid dosage was tapered and stopped. No relapse has occurred ever since.

Discussion

The differential diagnosis of painful neck conditions includes a wide spectrum from benign and malign proliferative pathologies to inflammatory and infectious states of neck structures including the thyroid gland. However, the diagnosis of SAT among these is usually straightforward.[2,5,6]

Painful HT is a concept first brought into the literature by Doniach *et al*. in 1960.[7] New reports since the definition of the concept are very limited in number.[3,4,8-15] Debate seems to be continuing whether it is a different clinical entity or is simply a variant of SAT. It may be challenging to differentiate the two entities in a patient with a previous diagnosis of HT. A viral prodrome is common, though not a rule during the course of SAT.[2] Fever, neck pain,

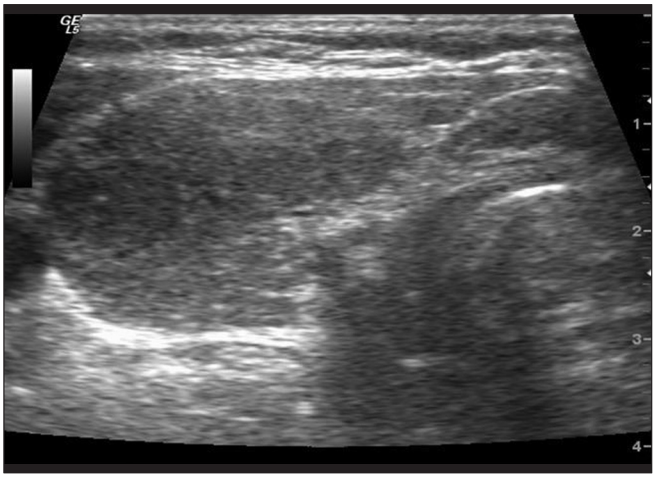

Figure 1: Gray-scale sonography of the patient's thyroid gland at presentation (right lobe, transverse view)

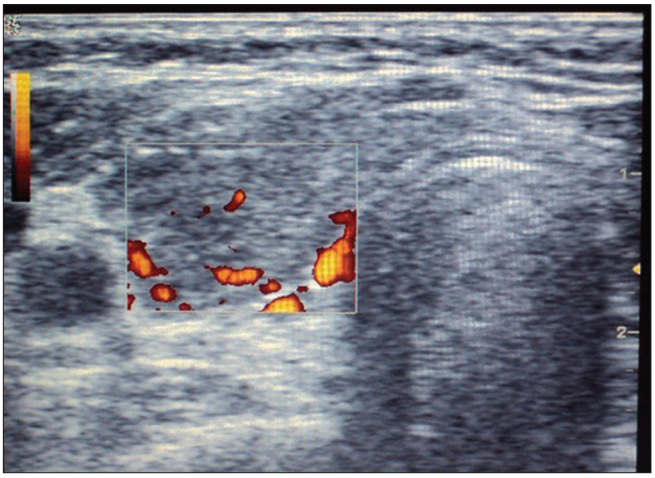

Figure 2: Doppler sonography of the patient's thyroid gland at presentation (Power Doppler mode, right lobe, transverse view)

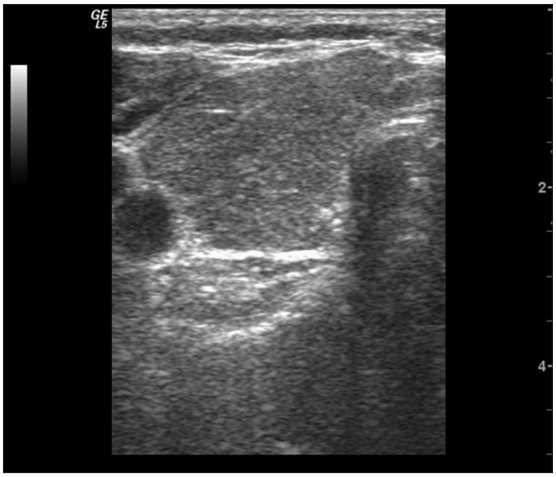

Figure 3: Gray-scale sonography of the patient's thyroid gland during follow-up (right lobe, transverse view)

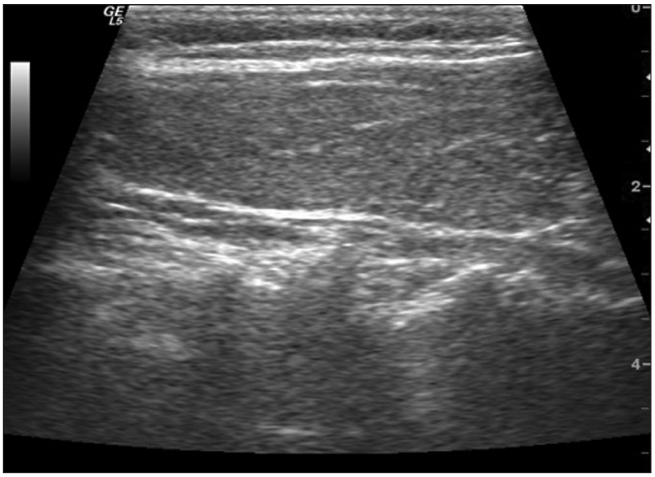

Figure 4: Gray-scale sonography of the patient's thyroid gland during follow-up (right lobe, longitudinal view)

increased ESR, and CRP can be seen in both entities which are acute inflammatory conditions.[2-4] SAT is usually characterized by thyrotoxicosis in the acute phase, but this may not be a discriminatory feature from painful HT in cases with preexisting HT such as ours.[1,2] Noritaka *et al.* reported in 2009 that ultrasonographically focal thyroiditis areas of two patients with painful HT showed increased blood flow with PDI.[15] In SAT, the blood flow is classically decreased in the hypoechoic areas.[1,15] The authors pointed out that PDI could be helpful in differentiating the two entities.[13] HT presenting as painful swelling is a rare entity and pain usually starts at presentation and not during follow-up. The cases of painful HT defined to date had generally persistant pain refractory to glucocorticoid therapy,[4] but not invariably.[16] Some patients have even undergone thyroidectomy.[12-14]

The clinical course and the laboratory findings of our case with a viral prodrome, decreased local blood flow, glucocorticoid responsiveness probably coincide with SAT. The laboratory findings were compatible with the recovery phase of SAT and the toxic phase was likely missed. We came across only one report of a preexisting HT case developing SAT.[17] More research is needed on the topic to be able to reach a consensus for a clear-cut differentiation of both entities.

The presenting clinical and laboratory findings of the patient is compatible with SAT, which is an unusual, but a much more common presentation than a painful attack of HT to be considered in differential diagnosis. It should be kept in mind that such painful attacks some of which may require intervention may develop during follow-up of HT cases.

References

1. Dayan CM, Daniels GH. Chronic autoimmune thyroiditis. N Engl J Med 1996;335:99-107.
2. Pearce EN, Farwell AP, Braverman LE. Thyroiditis. N Engl J Med 2003;348:2646-55.
3. Kasagi K. Painful Hashimoto's thyroiditis. Intern Med 2006;45:351-2.
4. Zimmerman RS, Brennan MD, McConhey WM, Goellner JR, Gharib H. Hashimoto's Thyroiditis. An uncommon cause of painful thyroid unresponsive to corticosteroid therapy. Ann Int Med 1986;104:355-7.
5. Volpé R, Johnston MW. Subacute thyroiditis: A disease commonly mistaken for pharyngitis. Can Med Assoc J. 1957;77:297-307.
6. Nishihara E, Ohye H, Amino N. Clinical characteristics of 852 patients with subacute thyroiditis before treatment. Intern Med 2008;47: 725-9.
7. Doniach D, Hudson RV, Roitt IM. Human auto-immune thyroiditis: Clinical studies. Br Med J 1960;1:365-73.
8. Suzuki H, Abe K, Fujimoto Y. Five cases with chronic thyroiditis showing acute inflammatory symptoms. Naika 1964;14:1140-6.
9. Fui SN, Jefferys DB. Subacute autoimmune thyroiditis simulating De Quervain's thyroiditis. Lancet 1979;1:622.
10. Leung AK, Hegde K. Hashimoto's thyroiditis simulating De Quervain's thyroiditis. J Adolesc Health Care 1988;9:434-5.
11. Shigemasa C, Ueta Y, Mitani Y, Taniguchi S, Urabe K, Tanaka T, *et al*. Chronic thyroiditis with painful tender thyroid enlargement and transient thyrotoxicosis. J Clin Endocrinol Metab 1990;70:385-90.
12. Kon YC, DeGroot LJ. Painful Hashimoto's thyroiditis as an indication for thyroidectomy: clinical characteristics and outcome in seven patients. J Clin Endocrinol Metab 2003;88:2667-72.
13. Ohye H, Fukata S, Kubota S, Takamura Y, Matsuzuka F, Amino N, *et al*. Successful treatment for recurrent painful Hashimoto's thyroiditis by total thyroidectomy. Thyroid 2005;15:340-5.
14. Gourgiotis L, Al-Zubaidi N, Skarulis MC, Papanicolaou DA, Libutti SK, Alexander HR Jr, *et al*. Successful outcome after surgical management in two cases of the "painful variant" of Hashimoto's thyroiditis. Endocr Pract 2002;8:259-65.
15. Onoda N, Kato Y, Seki T, Kurimoto M, Takano K, Ito Y, *et al*. Increased thyroid blood flow in the hypoechoic lesions in patients with recurrent, painful Hashimoto's thyroiditis at the time of acute exacerbation. Endocr J 2009;56:65-72.
16. Ishihara T, Mori T, Waseda N, Ikekubo K, Akamizu T, Imura H. Histological, clinical and laboratory findings of acute exacerbation of Hashimoto's thyroiditis-comparison with those of subacute granulomatous thyroiditis. Endocrinol Jpn 1987;34:831-41.
17. Takashima S, Sone S, Horii A, Honjho Y, Yoshida J. Subacute thyroiditis in Hashimoto's thyroiditis which mimicked primary thyroid lymphoma. J Clin Ultrasound 1997;25:279-81.

Cite this article as: Umut M, Cuneyd A, Alptekin G. Should neck pain in a patient with Hashimoto's thyroiditis be underestimated? A case and review of the literature. Indian J Endocr Metab 2012;16:444-6.
Source of Support: Nil, **Conflict of Interest:** None declared.