



Psychotic Depression Related to Hashimoto's Thyroiditis: A Case Report

Hashimoto Tiroiditine Bağlı Psikotik Özellikli Depresyon: Bir Olgu Sunumu

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Abstract

Hashimoto's thyroiditis can lead to depression, anxiety disorder, and sleep problems. However, this autoimmune disease has been reported to be less related to psychotic depression. The case applied to our outpatient clinic with introversion, anhedonia, and guilt thoughts. The patient was admitted to our clinic with a preliminary diagnosis of psychotic depression. TSH value was found to be high in the patient's hormone analysis. Afterward, treatment was started with the diagnosis of Hashimoto's thyroiditis according to the patient's anti-TPO value and thyroid USG results. After levothyroxine treatment, the patient's depression scores, TSH, and anti-TPO values decreased.

Keywords: Hashimoto's thyroiditis, psychotic depression, hypothyroidism

Öz

Hashimoto tiroiditi depresyona, anksiyete bozukluğuna ve uyku sorunlarına yol açabilir. Bununla birlikte, bu otoimmün hastalığın psikotik özellikli depresyonla daha az ilişkili olduğu bildirilmiştir. Olgu içe dönüklük, zevk alamama ve suçluluk düşünceleri ile polikliniğimize başvurdu. Hasta psikotik özellikli depresyon ön tanısı ile kliniğimize yatırıldı. Hastanın hormon analizinde TSH değeri yüksek bulundu. Ardından hastanın anti-TPO değeri ve tiroid USG sonuçlarına göre Hashimoto tiroiditi tanısı ile tedaviye başlandı. Levotiroksin tedavisi sonrası hastanın depresyon skorları, TSH ve anti-TPO değerleri düştü.

Anahtar Kelimeler: Hashimoto tiroiditi, psikotik özellikli depresyon, hipotiroidi

INTRODUCTION

Hashimoto's Thyroiditis (HT) is an organ-specific autoimmune disease characterized by lymphocytic infiltration in the thyroid gland. HT is the most common cause of hypothyroidism, but it may rarely be present with hyperthyroidism. Thyroid-stimulating hormone (TSH) level increases in patients with HT-induced hypothyroidism and sub-clinical hypothyroidism. Higher levels of anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin antibodies in serum are more sensitive to HT diagnosis (1).

HT can cause symptoms of many psychiatric diseases. The most common psychiatric symptoms of HT are psychomotor retardation, depressive mood, anxiety, and

sleep disturbances. Additionally, psychotic and manic/hypomanic symptoms are relatively less common in patients with HT (2).

The relationship between HT and psychiatric diseases has been mentioned in many studies. In case reports in the literature, acute psychosis, affective psychosis, and epileptic seizures have been reported together with HT encephalopathy (3-5). In a study, thyroid autoantibodies were found to be higher in bipolar disorder (6). Acute manic episodes have been described with HT (7).

While depression in HT patients with subclinical hypothyroidism is frequently encountered in the literature (8,12), information on psychotic depression is limited in HT patients with subclinical hypothyroidism.

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The authors aimed to report psychotic depression in an HT patient with subclinical hypothyroidism.

CASE REPORT

A 37-year-old female patient was referred to the psychiatric outpatient clinic with anhedonia, social isolation, crying, guilty thoughts, and sleep disturbances. She was married for ten years and had two children. She had no psychiatric disease or treatment history in the past. She was diagnosed with HT about ten years. She was irregularly using levothyroxine sodium 75 mcg per day. Her husband reported that she was depressed and anhedonic last month. At the same time, she had crying episodes and was talking to herself. According to the mental state examination of the patient, she had poor self-care and depressive affect. Her eye contact was decreased, and she was reluctant to communicate along with the examination. The patient had guilty delusion and described auditory hallucination. The psychotic symptoms were consistent with the patient's mood.

Beck Depression Inventory (BDI) and Brief Psychiatric Rating Scale (BPRS) scores of the patient were 22 and 45, respectively. According to the results of laboratory tests, the TSH level of the patient was 18.54 uIU/MI. FT3 and FT4 were normal (respectively 1.92 and 0.67). The Anti-TPO level of the patient was 769.7 IU/mL. There were no other abnormal laboratory findings, including hemogram, CRP, sedimentation, and electrolytes. Ultrasound scan of thyroid gland showed diffuse heterogeneity in the parenchyma. We did hospitalize the patient in the psychiatry inpatient unit and internal medicine consultation for the patient. After the patient was hospitalized in the psychiatry inpatient unit, venlafaxine 75 mg, levothyroxine 125 mcg, and olanzapine 10 mg were administered per day. With this treatment, the psychotic and depressive symptoms of the patient were decreased two weeks later. BDI and BPRS scores of the patient were 10 and 19 two weeks later. At the end of two weeks, TSH and anti-TPO levels of the patient were declined 0.91 uIU/MI and 609.5, respectively.

At the psychiatric examination one month later, the patient's psychotic symptoms had disappeared. The patient was able to make eye contact during the psychiatric examination and was willing to be interviewed. The patient had a decrease in guilt thoughts. There was an improvement in the patient's sleep and appetite. The patient's self-care and depressive affect also improved. Venlafaxine 75 mg and olanzapine 10 mg treatment was continued. The patient continued to receive levothyroxine 125 mcg treatment from the internal medicine outpatient clinic. During the 6-month follow-up period, the patient did not have any active psychiatric complaints. The patient's olanzapine treatment was gradually tapered and discontinued. The patient's venlafaxine treatment was tapered and stopped at the end of 1 year. The patient did not have any additional psychiatric complaints during this period.

DISCUSSION

It has been reported that disarrays in the hypothalamus-pituitary-thyroid (HPT) axis may play a role in the pathogenesis of psychiatric diseases. It is thought that in depression, the response of thyroid-releasing hormone (TRH) to TSH decreases, TRH levels increase in the cerebrospinal fluid and the blood level of antithyroid antibodies increases. Therefore, hypofunction of the thyroid gland located in HPT axis can lead to depression and cognitive disorders (9). However, the association of psychosis with thyroid dysfunction is less frequently reported (10). A scarce psychotic picture known as myxedema psychosis in the literature can be seen together with HT (11).

HT can be seen together with autoimmune diseases such as rheumatoid arthritis, vitiligo, alopecia areata, Type 1 diabetes, autoimmune liver diseases, ankylosing spondylitis, and ulcerative colitis. HT usually manifests weakness, fatigue, cold intolerance, decreased sweating, hoarseness, edema, amenorrhea, weight gain, forgetfulness, and constipation. In untreated cases, depressive complaints can be added to the table (12).

Many studies and case presentations have reported that HT can accompany depressive symptoms (13-16). Krysiak et al. reported depressive symptoms at a rate of 59% in HT patients with subclinical hypothyroidism and 37% in euthyroid HT patients in their study (17). On the other hand, psychosis is rarely seen in patients with HT due to hypothyroidism. However, there is limited information about psychotic depression that is related to HT. This case report has shown that HT can accompany psychotic depression. The case report has indicated that clinicians should be alert for different manifestations of HT.

Being female and middle-aged is considered a risk factor for the development of hypothyroidism (7). In this case, the rapid disappearance of psychotic and depressive symptoms with levothyroxine treatment and the acute onset of the psychiatric complaints suggested that HT may have caused this condition. Anti-TPO value and psychiatric symptom severity decreased aligned with levothyroxine treatment. The fact that the patient did not have a history of psychiatric illness and did not describe psychosocial stressors increased our evidence that the psychiatric complaints of our case were associated with HT. In addition, no depressive, manic, or psychotic episodes were detected in our patient's 1-year follow-up. Our case was a known HT patient, and therefore thyroid hormone replacement therapy was started early. Routine testing of thyroid hormones in patients with psychotic or affective symptoms with risk factors can improve treatment outcomes.

CONCLUSION

Although HT is a disease that is followed up in endocrinology outpatient clinics, it can present with different manifestations due to the variety of

neuropsychiatric symptoms it causes. It is clear that the application of thyroid function tests in acute psychotic symptoms, as in this case, has clinically significant results.

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Informed Consent: Informed consent was taken from the patient.

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