CASE REPORT

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Ego-Dystonic Psychotic Symptoms as the Initial Manifestations of Hypothyroidism in an Elderly Man

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ABSTRACT Hypothyroidism is a common endocrinological disorder that results from under-functioning or absence of the thyroid gland, resulting in a deficiency of thyroid hormones. It has well-known neuropsychiatric manifestations like cognitive disturbances, depressed mood, and anxiety. Psychosis may develop due to hypothyroidism, but this is very rare in current clinical practice due to hormone replacement therapy. An 81-year-old male patient was brought to the emergency room after a suicide attempt due to psychotic symptoms without apparent physical signs and symptoms of hypothyroidism. After neuropsychiatric evaluation and laboratory tests, hypothyroidism was detected as the etiological factor. Thus, the thyroid hormone replacement therapy was given, and all the symptoms of the patient were relieved. In the light of this case report, it's emphasized that thyroid hormone levels must be screened for patients with new-onset psychotic symptoms.

Keywords: Hypothyroidism; psychosis; thyroid hormones; screening

Hypothyroidism is a clinical state, resulting because of absence or dysfunction of the thyroid gland. Its prevalence ranges from 0.5-18% depending on the study population.[1] The well-known neuropsychiatric manifestations of hypothyroidism are depressed mood, diminished concentration, cognitive disturbances, hypersomnia, fatigue, poor memory, and occasionally anxiety.[2] Psychotic symptoms usually appear months or years after physical symptoms and signs of hypothyroidism but rarely occur without frank physical symptoms.[3] Myxedema psychosis simply refers to the acute psychotic symptoms induced by hypothyroidism.[4] There are no typical features of psychotic symptoms secondary to hypothyroidism.[1] Thus, an underlying thyroid pathology can be easily over-looked especially without apparent physical signs and symptoms. This paper presents a case of an elderly man who developed psychotic symptoms without frank physical symptoms due to hypothyroidism and recovered completely after treatment.

CASE REPORT

An 81-year-old male patient with no history of previous psychiatric disorder was brought to the emergency room by his relatives. It was learned that the patient was about to jump from the third floor, just before the hospital admission. Because of suicide risk, the patient was admitted to the psychiatry service. A detailed history was taken from the patient and his relatives. There was no previous psychiatric disorder in the history of the patient. In his past medical history, he had hypertension that was under control with anti-hypertensive medication. The patient has remained hypothyroid after radioactive iodine treatment performed 5 years ago but had not used levothyroxine regularly for the last 1 year. The patient had undergone cauterization due to a gastric ulcer. Lastly, he had been using a hearing aid due to long-term hear loss that did not interfere with communication. Relatives of the patient reported that six months ago his son had a traffic accident, was found

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guilty and went to jail a month ago. They also reported that the patient had no symptoms of memory disturbance. He had been displaying paranoid symptoms for the last two months and underwent a comprehensive examination, but no physical signs or symptoms were observed. According to the mental state examination, his orientation was normal, but he had ego-dystonic persecutory delusions, auditory, visual, and olfactory hallucinations, and his judgment was impaired. Besides, the patient's mood and affect were evaluated as anxious because of paranoid thoughts. The patient reported that soldiers had been following him to arrest and he had been hearing voices like "You are a terrorist; you can't escape from us". He also said that he had been seeing soldiers everywhere, getting a smell of gunpowder and he had been afraid of being arrested. The patient's kidney function tests, liver function tests, electrolyte levels, amylase level, vitamin B_{12}, and folate level were normal. Abnormal laboratory values were as follows; hemoglobin level: 9.91 g/dL (reference: 13-17), ferritin: 14.78 ng/mL (reference: 30-400), thyroid-stimulating hormone (TSH): 73.78 µIU/mL (reference: 0.36-4), free thyroxine (fT4): 0.252 ng/dL (reference: 0.91-1.67), free triiodothyronine (fT3): 1.19 pg/mL (reference: 2.66-4.42), antithyroglobulin: 1,189 U/mL (reference: 0-115), antimicrosomal antibody: 77.93 IU/mL (reference: 0-34). Calcitonin and thyroglobulin levels were in the normal range. There were no pathological signs in electroencephalography and brain magnetic resonance imaging. Levothyroxine replacement was started for hypothyroidism. Additionally, iron replacement was started for iron deficiency. Olanzapine was started at a dose of 10 mg/day to alleviate his psychotic symptoms and related suicidal thoughts. On the 40th day of hospitalization, there was a complete resolution of psychotic symptoms. During his 40-day hospitalization, his Brief Psychiatric Rating Scale score decreased from 65 to 24. Also, the mini-mental examination score, which was 24/30 at admission, was 28/30 at discharge. On the 40th day of levothyroxine treatment, TSH level was 15.94 µIU/mL, fT4 level was 0.833 ng/dL, fT3 level was 1.92 pg/mL, and the patient was taking a single daily dose of 100 mcg levothyroxine. The patient was discharged after reducing the olanzapine dose to 5 mg/day and had no psychotic symptoms at discharge. He was examined three months after discharge. The patient was examined three months after discharge, his mental state examination was completely normal, and he had no psychotic symptoms. The functionality of the patient was very well. Meanwhile, fT3, fT4, and TSH levels of the patient were within the normal range with levothyroxine treatment. Evaluation in the sixth month of the treatment revealed no psychiatric symptoms except for mild anxiety that did not affect functionality. Olanzapine treatment was discontinued. He was re-evaluated 6 months after the antipsychotic treatment was discontinued and was completely symptom-free.

DECLARATION OF PATIENT CONSENT

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his clinical information to be reported in the journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

DISCUSSION

London Clinical Society reported that delusions and hallucinations accompanied almost half of the severe hypothyroidism cases with myxedema in 109 cases before the thyroid hormone replacement therapy was not discovered.[5] Asher who added the terminology "myxedema madness" reported that various forms of psychosis have been found to accompany 5-15% of myxedema patients.[3] A few case reports point out hypothyroidism can cause psychotic symptoms without overt physical complaints. For example, Haberfellner et al. reported that a young woman had been treated for one and a half year with antipsychotic agents until hypothyroidism was diagnosed.[6] Nazou et al. reported a case of acute psychosis as the first manifestation of hypothyroidism.[7] Shlykov et al. reported a case of a hypothyroid woman who presented with symptoms of Capgras syndrome.[8]

In the present case, the ego-dystonic nature of delusions, olfactory hallucinations, the onset age of onset of psychotic symptoms were pointing out an underlying organic disorder. In the history of the pa-

tient, radioiodine treatment-induced hypothyroidism and noncompliance with levothyroxine treatment were two crucial points to suspect hypothyroidism as the etiological factor. The patient had no symptoms of rhabdomyolysis like muscle pain or weakness, but whether there was a mild asymptomatic metabolic myopathy is unknown, because the creatine kinase level wasn't investigated. Hashimoto encephalopathy (HE) can also be questioned in the differential diagnosis. Unfortunately, the definitive diagnostical distinction between HE and myxedema psychosis is a challenge for clinicians and it remains a topic of investigation. The diagnosis of HE can be made after excluding possible etiological factors and it is still a subject of debate.[9] In the present case, the patient had severe hypothyroidism that could explain the symptoms, and the clinicians gave priority to hormone replacement and antipsychotic therapy. The symptomatic improvement in the patient was as expected. So, the diagnosis of HE seemed unlikely. If the patient had been euthyroid or mildly hypothyroid, further laboratory investigations might have been done to evaluate the patient in terms of HE.

The neuropathological mechanisms underlying hypothyroidism-related psychosis are still subject of debate. It has been shown that there was an increased cerebral dopamine level, dopamine receptor sensitivity, and tyrosine hydroxylase activity in rats with iatrogenically induced hypothyroidism.[10] Positron emission tomography studies revealed an overall decrease in cerebral blood flow in patients with hypothyroidism.[11] Although neuropsychiatric disturbances caused by hypothyroidism are often reversible, complete recovery may not be achieved in cases whose treatment is delayed due to chronic metabolic and structural changes in the brain.[12] Therefore, every patient with new-onset psychosis should be screened for thyroid functions.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Ferit Şahin;* ***Design:*** *Ferit Şahin;* ***Control/Supervision:*** *Zehra Arıkan;* ***Data Collection and/or Processing:*** *Ferit Şahin;* ***Analysis and/or Interpretation:*** *Ferit Şahin;* ***Literature Review:*** *Zehra Arıkan;* ***Writing the Article:*** *Ferit Şahin;* ***Critical Review:*** *Zehra Arıkan.*

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