Psychiatric Manifestations in a Patient with Both Huntington's Disease and Hyperthyroidism: A Case Report

Case Report

We report the case of a 57-year-old woman patient with the underlying of Huntington's disease (HD), which was diagnosed at her age of 50 years based on the findings of motor symptoms and positive genetic test. She had received tetrabenazine 25 mg per day regularly after the diagnosis. Her premorbid personality was extroverted, and had good job performance. A few months before her diagnosis of HD in 2012, she had a first depressive episode without obvious stress. She showed persistent low mood, social withdrawal, insomnia, and loss of energy. She had mild impaired work performance and memory, requiring help in daily functions. Her depressive mood was partially improved after 3–4 years without complete remission. In 2016, her mood was lifted – displaying grandiose thoughts, a decreased need for sleep, talkativeness, distractibility, and having goal-directed behaviors. She also had decreasing body weight. Moreover, she started to show psychotic symptoms such as grandiose and persecutory delusions at the end of 2017. In October 2018, she had above manic and psychotic symptoms, along with emotional lability, visual hallucinations, and reckless behaviors. She even presented herself with violence and self-harm behaviors because of her delusions. She did not visit any psychiatric clinic before admission.

After the admission on December 24, 2018, the patient had tachycardia and hypertension. She had symptoms of disorientation, paranoid attitude, and interrupted sleep. She did not have any focal abnormality or paroxysmal discharge in the electroencephalographic examination. Although we prescribed olanzapine 5 mg on the first day of admission for her psychotic symptoms, the dose was gradually increased to 20 mg per night after four days with little improvement. Then she received valproic acid 200 mg twice daily for her expansive mood, talkativeness, and agitated behavior on the fourth day of admission. Laboratory data showed normal results except hyperthyroidism (thyroid-stimulating hormone [TSH] < 0.003 uIU/mL, free T4 > 6.6 ng/dL) on December 27, and Graves' disease was diagnosed after testing for TSH-antibody (70.37%). Therefore, we added methimazole 10 mg twice daily and propranolol 10 mg three times daily to the treatment regimen after consultation with an internist the next day. Her thyroid echogram revealed the finding of left nodular goiter. But fine-needle aspiration was not done due to her poor cooperation for the procedure. Her tachycardia and hypertension were improved. Due to her emotional lability, grandiosity, and restlessness decreased mildly, she received valproic acid to 200 mg three times daily in the second week of admission. In the beginning of the third week, her manic symptoms were improved.

The patient still displayed fluctuating consciousness and exacerbating agitation at night. She had persisting persecutory delusions and visual hallucinations. Delirium was considered due to the above symptoms. We then cross-titrated her medication from olanzapine to risperidone and slowly increased to 4 mg per night during the fourth week of her hospitalization. Then, her paranoid attitude and psychotic symptoms were decreased. She became less agitated and less interrupted in sleep after her improved delusions and visual hallucinations. In addition to her residual psychotic symptoms and intermittent restlessness, cognitive dysfunction such as disorientation, and poor memory and attention, had stayed unchanged until discharge. Her last follow-up on thyroid profile on January 26, 2019 were much improved (TSH = 0.05 uIU/mL, free T4 = 0.474 ng/dL). She was followed up in both psychiatric and endocrine clinics after discharge on February 3, 2019.

Comment

In a more detailed history taking, the patient's first manifestation of psychiatric symptoms was depression before her diagnosis of HD. The lifetime prevalence of depression in HD is 20%–56% [1]. The appearance of depression occurs before the onset of motor symptoms and peaks during the early stage. This clinical finding was compatible with the onset of depressive symptoms such as low mood and social withdrawal, as seen in our patient. Her depressive mood persisted for 3–4 years without remission. Mood disorder due to a medical condition, HD, was suspected. She had declined executive function and memory around the same time when her depression occurred, but her dementic condition did not improve when her low mood was improved in years. Characterized by impaired emotion recognition, processing speed, visuospatial, and executive function, cognitive deficit in HD patients can be seen many years before the onset of motor symptom onset [2].

Manic symptoms have a lifetime prevalence of 5%–10% in HD [3]. Major depression or mania can be the first symptom, suggestive of HD [3]. Our patient's manic episode is less likely to be symptoms from HD or primary bipolar disorder due to the time of onset and non-episodic presentation. Her manic symptoms were persistent and worsened over time in two years, possibly due to progressing hyperthyroidism and the untreated manic condition. Overactivity of the adrenergic system may explain the similarity between the clinical presentations of hyperthyroidism and mania or anxiety [4]. After the treatment with antithyroid drugs and β-blockers, her elevated free T4 and TSH levels were improved with a relatively stable heart rate

and blood pressure. Her manic symptoms such as emotional lability, grandiose delusions, and restlessness were also decreased. But we could not fully contribute her improvement of manic symptoms to the use of methimazole because she also received both olanzapine and valproic acid during the same time. Treatments of hyperthyroidism usually improve both mental and physical symptoms [4].

Our patient started developing paranoid delusions and visual hallucinations after her manic presentation for one year. Although it may present, psychosis is one of the least prevalent psychiatric manifestations of HD [1]. Psychotic symptoms in HD patients are usually treated with antipsychotic drugs, and most practitioners have a preference for the second-generation (atypical) antipsychotic drugs because of the lower risk potential of extrapyramidal side effects [3], which was why we chose olanzapine as the first-line antipsychotic for her psychosis. But the reversed sleep circadian, rambling speech, visual hallucination and agitated behavior occurred more frequently at night. This clinical finding was the suspicion from delirium caused by thyrotoxicosis because she did not have any abnormal physical symptoms or laboratory data. Delirium is common in HD, therefore, a sudden change in behavior or decline in cognitive abilities needs an evaluation for delirium [3]. The activities of neurotransmitters, such as serotonin and dopamine in the limbic system, are affected by the excessive production of thyroid hormone in thyrotoxicosis [5]. After we discovered her possible cause of delirium and kept giving her methimazole to correct her thyroid function, we then used risperidone as a higher-potency antipsychotic agent to treat her delirium. The patient showed less fluctuating consciousness and psychotic symptoms.

Untreated hyperthyroidism may influence the cerebral metabolism in the regions of the brain, responsible for memory and executive functions [6], which may have occurred in our patient because of gradually elevating thyroid levels over the years. In addition, as the HD progresses to the end stage, a need for all-day care exists due to further cognitive decline [2]. The residual disorientation, inattentiveness, residual psychotic symptoms, and confabulation in our patient are possibly due to brain's vulnerability to degeneration from HD and cognitive impairment from possible neurological manifestations from hyperthyroidism.

Patients with HD may experience many neuropsychiatric symptoms or disorders during the course of their illness. Based on the findings in this case report, we suggest that thyroid disease should be considered in all kinds of psychiatric manifestations during differential diagnosis. Our patient's complex history, as we have reviewed here, has taught us to be aware of her underlying diagnosis and any physical disease when we confronted a patient with mood and behavioral changes. (The institutional review board at Chung Shan Medical University Hospital granted an IRB-exempt status for the publication of this case report (IRB protocol number = CSMUH No. CS2-20127 and date of approval = October 13, 2020) without any stipulation of obtaining informed consent from the patient.)

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Conflicts of Interest

There are no conflicts of interest.

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