Autoimmune Thyroid Disease–Induced Catatonia With Normal Thyroid Function

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Catatonia is an acute state marked by psychomotor, neurological, and behavioral changes.[1] We present a case of a patient with catatonia and worsening episodes of anxiety, insomnia, and alterations in cognition found to concurrently have autoimmune thyroid disease (AITD). Due to an unremarkable initial workup, accurate diagnosis was challenging. A robust review of the medical causes of catatonia eventually yielded a productive diagnostic workup.

Case Report

A 26-year-old white woman presented to the emergency department (ED) after 5 months of progressively worsening premenstrual and cyclic episodes of anxiety, insomnia, stereotypic movements, disorientation, memory lapses, blurry vision, and anorexia, which led to multiple prior hospitalizations. Blurry vision was intermittent and endorsed specifically during periods of anxiety and insomnia. She denied diplopia or eye pain. Family history was significant for cyst thyroidectomy and Graves disease, as well as nonspecific anxiety and depression. At symptom onset, the patient had an elevated thyroid-stimulating hormone (TSH) level of 11.9 mcIU/mL (reference range, 0.5–5.0 mcIU/mL), but repeat laboratory tests showed TSH, T4, and T3 values within normal limits. Workups were negative, including routine blood work, vitamin B_{12}, magnesium, toxicology screens, and antinuclear antibodies. Sleep studies, brain magnetic resonance imaging, computed tomography scan, electroencephalography, cerebrospinal fluid analysis, and infectious workups were also unremarkable. Her diagnosis remained uncertain, and she was repeatedly discharged home after stabilization.

Previous trials of escitalopram, mirtazapine, and trazodone did not improve her condition. Lorazepam and transcranial magnetic stimulation were also trialed and improved symptoms, but treatment was discontinued due to patient reluctance.

She was admitted to the hospital from the ED due to a sharp decline in functional status. She displayed mutism, poor eye contact, agitation, and significant paranoia with persecutory delusions and was treated with olanzapine. The physical examination was negative for ophthalmopathy. She displayed subtle signs of echopraxia, so further evaluation of catatonia was performed with the patient scoring a 19 on the Bush-Francis Catatonia Rating Scale (BFCRS).[2] Lorazepam challenge was administered with positive response. Lorazepam 0.5 mg intravenous 3 times daily was initiated, and olanzapine was discontinued. Lorazepam was subsequently titrated to response and switched to oral route. Symptoms improved substantially, confirming catatonia. Lamotrigine was given for suspicion of underlying bipolar disorder but was discontinued due to temporally associated transaminitis. Given the initial TSH abnormality, antithyroid peroxidase antibody was obtained and found to be elevated at 963 IU/mL (reference range, 0–9 IU/mL), suggesting Hashimoto thyroiditis. Due to medical stability, she was discharged home with lorazepam and endocrine follow-up.

Postdischarge, under continued lorazepam treatment, endocrinology evaluated thyroid-stimulating immunoglobulin (TSI), a subtype of TSH receptor antibody. The patient's TSI was elevated at 1.76 IU/L (reference range, ≤0.56 IU/L), which approaches 100% specificity for Graves hyperthyroidism[3] and is only simultaneously seen in 10%–15% of those with Hashimoto thyroiditis.[4] Due to normal thyroid hormone levels and lack of gross ophthalmopathy, no treatments for Graves disease were initiated, and she was scheduled for frequent follow-up and laboratory monitoring.

Discussion

This case demonstrates an example of AITD-induced catatonia when multiple previous medical workups were unrevealing. AITD creates antibodies against the thyroid gland, leading to respective antibody sequelae: hyperthyroid, neutral, or hypothyroid state.[4,5] Psychomotor manifestations such as psychosis, mania, depression, dementia, and catatonia[4] have all been described in AITD. Previous studies suggest psychiatric symptoms resolve when abnormal thyroid function is treated.[6,7] We expect the patient's episodes to subside with appropriate management.

Diagnosis of catatonia incorporates 3 or more of any 12 *DSM-5* criteria, 1 qualitative scaling by BFCRS,[2] and evaluation for underlying causality.[8,9] While psychotic and major mood disorders classically explain a sizable portion of catatonia, the *DSM-5* now acknowledges medical causes (Table 1).[1,8,10] In the absence of clear psychological diagnosis, etiology must be established in each catatonic patient for proper treatment to counteract catatonia reoccurrence[9] or exacerbation of symptoms.

Table 1.
General Medical Conditions Associated With Catatonia

	Encephalopathies	Major neurocognitive disorders (dementias)	Developmental disorders
General medical conditions	Autoimmune (especially anti-NMDAR) Autoimmune thyroid disease Chronic lymphocytic encephalitis CNS lung cancer metastases Epilepsy Infectious Metabolic Paraneoplastic syndromes	Frontotemporal dementia Lewy body dementia Neurological structural injury (vascular and traumatic)	Autism spectrum disorder 22q13.3 microdeletion syndrome

Abbreviations: CNS = central nervous system, NMDAR = *N*-methyl-D-aspartate receptor.

Our patient presented with AITD-induced catatonia in the absence of compelling workup. This case demonstrates why physicians should keep a broad medical differential in patients with acute catatonic states.

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