A Case of Autoimmune Thyroiditis Presenting as Apraxia

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ABSTRACT

Introduction: Autoimmune thyroiditis, commonly referred to as Hashimoto's thyroiditis, is the leading cause of hypothyroidism in iodine-sufficient regions. Neurologic manifestations are uncommon.

Case Description: A 20-year-old female with no significant medical history presented with aphasia and episodes of nonsensical speech. Initial workup was unremarkable, and she was discharged. She subsequently returned with recurrent neurologic symptoms, including apraxia and headache. Further evaluation revealed an elevated thyroid-stimulating hormone (TSH) level and markedly elevated antithyroid peroxidase (anti-TPO) antibodies. A diagnosis of autoimmune thyroiditis was made, and treatment with levothyroxine led to improvement in symptoms.

Discussion: Autoimmune thyroiditis can have atypical presentations, particularly in younger individuals. Genetic predisposition and family history may increase susceptibility. Early recognition is critical in identifying cases such as Hashimoto's encephalopathy, which may resolve completely with appropriate treatment.

Conclusions: We present a case of autoimmune thyroiditis that manifested solely with neurological symptoms. It is crucial to consider hypothyroidism when treating complex, undifferentiated patients.

INTRODUCTION

Autoimmune thyroiditis (AIT), traditionally referred to as Hashimoto's thyroiditis, is the most common autoimmune thyroid disorder and the leading cause of hypothyroidism in iodine-sufficient areas.[1] AIT occurs in approximately 0.3 to 1.5 cases per 1000 persons annually, with a frequency 4- to 10-fold higher in women than in men.[1] The cause of AIT remains unknown; however, the primary risk factor, as with many autoimmune conditions, is thought to be a combination of genetic susceptibility and environmental triggers that result in loss of immunological tolerance. Treatment includes levothyroxine, although case reports have shown that residual symptoms may persist for years despite initiation of treatment.[2]

CASE PRESENTATION

A 20-year-old female with no past medical history presented to an outside emergency department (ED) after an episode of gross aphasia, during which her speech was nonsensical when present. Four days prior to presentation, she developed myalgias, malaise, and congestion that waxed and waned over several days. Physical examination was notable for dysarthria; otherwise, findings were normal. Initial laboratory results, including a basic metabolic panel and complete blood cell count (CBC), are shown in the Table. Her Electrocardiography (ECG) demonstrated normal sinus rhythm. Noncontrast computerized tomography of the head revealed no acute intracranial abnormality.

Medical decision-making at the outside hospital indicated that, in the absence of laboratory or imaging abnormalities, her aphasic episode was most consistent with a panic attack. Psychiatry was not consulted; ED staff made the diagnosis. She was discharged home without further intervention.

The patient returned to our ED the next day with headache and apraxia, in addition to continued episodes of aphasia. Repeat laboratory results are shown in the Table. Due to concerns for encephalitis, a lumbar puncture was performed (see Table). Additional history obtained during this encounter revealed that she had spent the summer working as a camp counselor and had

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experienced a rash during her recent illness that spanned the bilateral upper and lower extremities while sparing the palms of her hands and the soles of her feet; therefore, Lyme serologies were obtained (see Table). CBC showed no leukocytosis, arguing against infection, and the basic metabolic panel revealed no metabolic abnormalities. Cerebrospinal fluid (CSF) cultures and nucleic acid amplification testing were negative for all organisms tested. CSF cell count and glucose were within normal limits, reducing the likelihood of meningitis. Borrelia burgdorferi antibodies were negative, making Lyme disease unlikely. She was discharged with a working diagnosis of an unknown headache disorder.

She returned to the ED the following day after additional episodes of impaired cognition and another apraxic episode during which she was unable to dress herself. She had also developed a bilateral headache, nausea, and vomiting. Neurologic exam was normal; however, neurology was consulted given ongoing neurologic symptoms. Neurology recommended adding paraneoplastic studies and N-methyl-D-aspartate (NMDA) receptor antibody testing to the previously collected CSF, as the workup to date had been unrevealing. Brain magnetic resonance imaging and magnetic resonance venography were obtained and demonstrated no acute intracranial abnormality (Figure). Repeat laboratory testing was suggestive of AIT (Table).

Additional history revealed a family history of hypothyroidism in her mother and both maternal and paternal grandmother; her maternal grandmother also had Sjögren syndrome. Notably, the patient denied changes in weight or appetite, constipation, or cold intolerance.

Electroencephalography, obtained on admission per neurology recommendation, revealed mild diffuse background slowing, consistent with hypothyroidism. Based on her elevated thyroid-stimulating hormone (TSH; 28.3 µIU/mL), low thyroxine (T4; 0.76 ng/dL), and markedly elevated antithyroid peroxidase antibodies (>600 IU/mL), a diagnosis of AIT was made, and levothyroxine therapy was initiated. Given the patient's neurological symptoms, she met diagnostic criteria for Hashimoto's encephalopathy (Box). She was referred for outpatient follow-up with endocrinology.

Review of the patient's medical record 6 months later showed that levothyroxine dosing had been titrated in response to fluctuating TSH levels, ultimately reaching 100 µg daily, with complete resolution of neurologic symptoms. Steroids were not administered, as her symptoms had improved with levothyroxine alone. A timeline of her hospital presentations is provided in the Table.

Table. Timeline of Hospital Presentations and Laboratory Results

Date	Visit
Day 0	Presented to an outside emergency department for an episode of aphasia. Work-up normal, discharged with diagnosis of panic attack.
Day 1	Presented after another episode of headache, apraxia, and aphasia. Repeat lab testing, with addition of Lyme serology, lumbar puncture, and MRI brain are unremarkable. Discharged with working diagnosis of unknown headache disorder.
Day 2	Presented with continuing episodes of impaired cognition and apraxia. Thyroid testing suggestive of autoimmune thyroiditis. EEG with findings consistent with hypothyroidism. Diagnosis of Hashimoto's encephalopathy made.

Abbreviations: MRI, magnetic resonance imaging; EEG, electroencephalography.

Box. Diagnostic criteria for Hashimoto's encephalopathy[6]

Diagnosis can be made when all six of the following criteria have been met:

1. Encephalopathy with seizures, myoclonus, hallucinations, or stroke-like episodes
2. Subclinical or mild overt thyroid disease (usually hypothyroidism)
3. Normal Brain magnetic resonance imaging or with nonspecific abnormalities
4. Presence of serum thyroid (thyroid peroxidase, thyroglobulin) antibodies
5. Absence of well-characterized neuronal antibodies in serum and cerebrospinal fluid
6. Reasonable exclusion of alternative causes

DISCUSSION

Autoimmune thyroiditis (AIT) is a rare autoimmune disorder with diverse and sometimes confusing clinical presentations. While the exact cause remains unknown, genetic susceptibility is supported by epidemiologic data, with siblings of affected patients demonstrating a significantly increased risk. AIT is more common in monozygotic twins (29%-55%) than in dizygotic twins.[1]

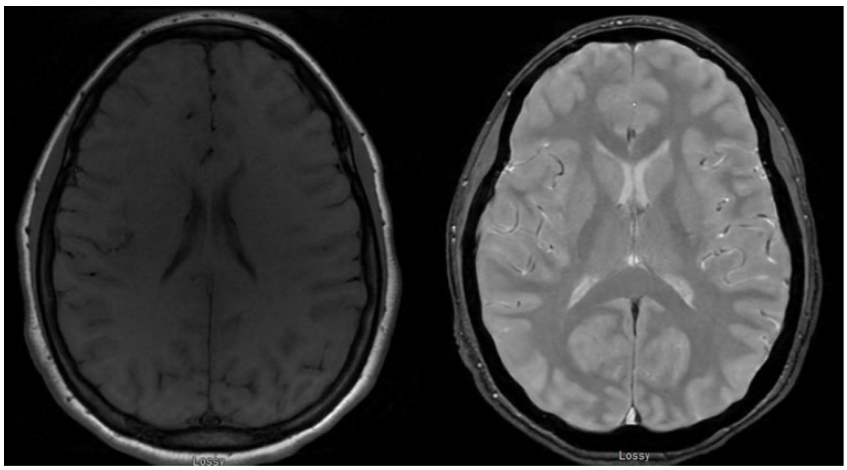
AIT involves both humoral (B cell-mediated) and cellular (T cell-mediated) processes. Pathogenesis includes a breakdown in immune tolerance, resulting in production of autoantibodies against thyroid peroxidase (anti-TPO) and thyroglobulin (anti-Tg), both of which are essential for thyroid hormone synthesis.[1] Antibodies against the TSH receptor may also be present in a subset of patients and can be stimulating, blocking, or neutral.[3] These autoantibodies, along with T cell–mediated cytotoxicity, contribute to the progressive follicular destruction. CD8+ cytotoxic T cells induce apoptosis of thyroid epithelial cells, while CD4+ helper T cells release pro-inflammatory cytokines (eg, IL-2, IFN-γ, TNF-β), exacerbating tissue damage. Genetic predisposition and environmental factors, such as viral infections and excessive iodine intake, further modulate disease progression.[4]

As thyroid tissue is destroyed, thyroid hormone production declines, leading to increased pituitary TSH secretion. Over time, the gland undergoes fibrosis and atrophy, resulting in progressive hypothyroidism.[4] Understanding the pathophysiology is critical for guiding diagnostic evaluation and therapy, including thyroid function monitoring and hormone replacement.

Patients with AIT typically present with hypothyroidism and/or goiter.[5] Clinical manifestations vary by disease stage, with early disease primarily presenting as asymptomatic. Goiter may also be present early. In goitrous AIT, also known as classic Hashimoto

disease, patients have a diffusely enlarged, firm thyroid gland that often has an irregular surface.[5] Thyroid enlargement typically ranges from minimal to massive, but in most cases the thyroid weighs approximately 2 to 3 times normal.[5] Although a feeling of neck tightness is common, thyroid pain and tenderness are rare.[5] In atrophic AIT, patients present without goiter.[5] Rarely, transitory hyperthyroidism (Hashitoxicosis) may occur due to follicular rupture of hormone-containing thyroid tissue, manifesting with symptoms such as irritability, heat intolerance, and diarrhea before progression to hypothyroidism.[7]

Figure. Unremarkable Magnetic Resonance Imaging of the Brain Without Contrast (Left) and Unremarkable Magnetic Resonance Venography of the Brain Without Contrast (Right).



As disease progresses, the thyroid may become normal-sized or small if extensive fibrosis has occurred, and patients may show signs of hypothyroidism.[5] Common symptoms include fatigue, lethargy, dry skin, cold sensitivity, muscle cramps, constipation, and voice changes. Less common manifestations, typically associated with severe or late-stage disease, include sleep apnea, pituitary hyperplasia, myxedema, and carpal tunnel syndrome.[7]

Diagnostic evaluation of AIT includes thyroid function tests and thyroid antibody testing. Thyroid function results vary by stage, with early disease showing transient hyperthyroidism (low TSH, high free T3, high free T4), progressive disease showing subclinical hypothyroidism (mildly elevated TSH, normal free T3/T4), and late-stage disease showing overt hypothyroidism (high TSH, low free T3, low free T4). Thyroid antibody testing is diagnostic, with antithyroid anti-TPO antibodies present in up to 95% of patients and anti-Tg antibodies in 60% to 80% of patients.[8]

The gold standard treatment symptomatic autoimmune hypothyroidism is daily, lifelong oral levothyroxine (L-T4), which addresses hormonal deficiency rather than disease pathogenesis.[7] Dosing is individualized to normalize serum TSH and typically averages 1.6 to 1.8 kg/day.[7] With appropriate therapy and lifelong monitoring, patients typically experience favorable outcomes with adequate symptom management.

A rare complication of AIT is Hashimoto's encephalopathy, an uncommon neurological syndrome with widely variable clinical features, including cognitive impairment, myoclonus, ataxia, seizures, and psychosis.[9] The condition affects approximately 2 per 100 000 individuals.[10] In a review of approximately 3000 patients with positive thyroid antibodies, only 5% met the criteria for Hashimoto's encephalopathy.[11] Diagnostic criteria include encephalopathy with seizures, myoclonus, hallucinations, or stroke-like episodes; dysthyroid state (typically hypothyroidism); normal or nonspecific brain MRI findings; presence of serum thyroid antibodies; absence of well-characterized neuronal antibodies in serum and CSF; and reasonable exclusion of alternative diagnoses (Figure).[6] Our patient met these criteria

Although steroid responsiveness is often observed and may support diagnosis, it is not established as part of the diagnostic criteria. Another case series and literature review described treatment with corticosteroids in combination with management the underlying dysthyroid state.[10] Given this patient's improvement with levothyroxine alone, steroids were deferred. Early recognition and treatment are essential, as timely intervention can significantly improve patient outcomes. Further research is needed to clarify pathogenesis and optimize management.

CONCLUSIONS

We report a rare case of Hashimoto's encephalopathy presenting solely with neurologic symptoms. This case highlights the importance of considering hypothyroidism in complex, undifferentiated presentations, as its presentation can vary widely. Awareness of AIT as a potential etiology for unexplained neurologic symptoms may facilitate earlier diagnosis and treatment, with levothyroxine leading to complete resolution of symptoms.

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