Hashimoto's encephalopathy is an autoimmune neuroendocrine disorder caused when the thyroid antibodies seen in Hashimoto's thyroiditis affect brain tissue.

**Autoimmune Dementia**

Hashimoto's encephalopathy (HE), which has recently been designated steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT), is an autoimmune disorder that can cause memory impairment, cognitive changes, dementia and associated neurological symptoms. HE can occur in patients with hypothyroidism related to Hashimoto's thyroiditis and in patients with normal thyroid function.

In some cases, particularly when HE occurs in association with non-thyroid related disorders such as Sjogren's syndrome or systemic lupus erythematosus (SLE), it's called nonvasculitic autoimmune meningoencephalitis (NAIM).

**History**

The first cases of Hashimoto's encephalopathy were reported in 1966 when the existence of thyroid antibodies in the blood of patients with the autoimmune hypothyroid disorder Hashimoto's thyroiditis was first demonstrated. SREAT is caused by the same thyroid antibodies that cause Hashimoto's thyroiditis although in SREAT, rather than destroying thyroid tissue, these antibodies attack and destroy brain cells known as neurons.

Most experts believe that SREAT is under-diagnosed and that many patients thought to have Alzheimer's disease actually have SREAT, which is a treatable disorder. Hashimoto's encephalopathy has been reported worldwide, and cases have been documented in patients ranging from 12-82 years with women more likely to be affected than men.

**Encephalopathy**

Encephalopathy is a general term referring to an inflammatory brain disease that alters the brain's structure or function. Encephalopathy is suspected in patients showing signs of an altered mental state.

**Symptoms**
Common symptoms of encephalopathy include stroke-like symptoms of memory loss, difficulty concentrating, hallucinations, irritability, restlessness, amnesia, diminished cognitive ability, myoclonus (involuntary muscle twitching), tremors, nystagmus (rapid, involuntary eye movement), muscle weakness, dementia, seizures, convulsions, difficulty swallowing, impaired speech, confusion, disorientation, psychosis, headache, right-sided hemiparesis or partial paralysis, and fine motor problems, including incoordination of arms, hands and fingers.

**Diagnosis**

HE is diagnosed in patients with high titers of thyroid peroxidase (TPO) antibodies who show signs of cognitive impairment responsive to corticosteroids. Lymphocytic vasculitis of the veins and venules of the brain stem in HE supports the notion that HE may be an autoimmune vascular disorder. Vasculitis as a contributing factor to HE is also supported by the presence of anti-alpha-enolase antibodies in HE.

These antibodies are also seen in other conditions of vasculitis including systemic lupus erythematosus (SLE) and ANCA-associated vasculitis. Other diagnostic changes seen in HE include an elevated cerebrospinal fluid protein, EEG abnormalities (diffusely slowed), and perfusion deficits in the presence of normal structural neuroimaging.

Although hypothyroidism may be present, most patients have normal levels of circulating thyroid hormone, suggesting that inflammation rather than myxedema is responsible for the cognitive defects. CT scans may be normal or show cerebral swelling (patchy edema) secondary to diffuse white-matter edema appearing as diminished attenuation with increased signal intensity on T2-weighted matter MRI images. The variation in imaging test results may represent different stages or subtypes of HE.

**Relapsing Disease**

HE may, like multiple sclerosis, also cause a relapsing form of encephalopathy with imaging test results varying depending if the disease is an active or relapsing mode. Relapsing white-matter edema is the usual presentation.

Most patients have elevated titers of TPO antibodies and some patients have thyroglobulin antibodies. However, because 20 percent of the older population, especially women, may have these antibodies, antibody test results must be interpreted with caution. Patients with HE are also reported to occasionally have antinuclear antibodies (ANA) and anti-parietal cell antibodies. Fine needle aspiration (FNA) studies in patients with HE show lymphocytic thyroiditis.

**Treatment**

Patients with SREAT show a good response to corticosteroids such as prednisone and related immunosuppressants because of the ability of these medications to reduce thyroid antibody production and reduce inflammation. Researchers in India report a case of
SREAT that did not respond to corticosteroids but showed a very favorable response to plasma exchange, a technique used to remove circulating antibodies,


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