Hashimoto's encephalopathy

Hashimoto's encephalopathy, also known as steroid responsive encephalopathy associated with autoimmune thyroiditis (SREAT), is a neurological condition characterized by encephalopathy, thyroid autoimmunity, and good clinical response to steroids. It is associated with Hashimoto's thyroiditis.

Patients with Hashimoto's encephalopathy present with various clinical symptoms and magnetic resonance imaging (MRI) findings.

An alternative diagnosis of glioblastoma should be considered in patients presenting initially as autoimmune encephalitis (AE), especially in patients who do not fulfill the criteria for definite AE and in those with a poor clinical evolution despite initial improvement [1].

Uwatoko et al., from Sapporo, first documented a Hashimoto's encephalopathy with MRI findings mimicking a brain tumor. The patient was a 41-year-old woman with a history of Hashimoto's disease. She experienced gradually worsening Parkinsonism and an MRI revealed a brain tumor-like lesion at the left caudate nucleus. She underwent a brain biopsy that revealed diffuse gliosis and perivascular lymphocyte infiltration with CD3+ T-cell predominance. No pathological signs of a brain tumor were found. Hashimoto's encephalopathy was suspected based on the patient's history and the presence of anti-thyroid antibodies. Her symptoms and the MRI findings improved with glucocorticoid treatment. Although there exist only a few studies on the pathology of Hashimoto's encephalopathy, our findings were consistent with those of previous reports. The findings suggest cerebral vasculitis as an underlying etiology of Hashimoto's encephalopathy. They also emphasize the importance of considering Hashimoto's encephalopathy as a differential diagnosis of brain tumors [2].

Gauthier and Baehring, describe the case of a 55-year-old woman who presented with subacute cognitive decline and ataxia. Neoplastic, paraneoplastic, infectious, and metabolic etiologies were ruled out. Anti-TPO antibody level was markedly elevated at 966U/mL. After one month of 60mg/day of oral prednisone, she felt back to baseline and her Montreal Cognitive Assessment dramatically improved. Physicians should strongly consider this uncommon diagnosis in patients with rapid cognitive decline and no other clear etiology [3].

A 47-year-old man presented to our hospital after suffering transient loss of consciousness and falling to the floor. On admission, his Glasgow Coma Scale score was 11 (E3V3M5), and he exhibited restlessness. Blood examination revealed hyperthyroidism. Computed tomography showed slight traumatic subarachnoid hemorrhage. He developed fever and tachycardia, and was diagnosed with thyroid crisis. Magnetic resonance imaging showed a brain contusion in the right frontal lobe, and encephalopathy signs in the right frontal and insular cortex. Immunocytochemical examinations suggested Hashimoto's disease, and hormone examinations revealed plasma levels were undetectably low of adrenocorticotropic hormone (ACTH) and low of cortisol. Pituitary stimulation tests showed inadequate plasma ACTH and cortisol response, consistent with isolated ACTH deficiency (IAD). The final diagnosis was IAD associated with Hashimoto's disease. Hydrocortisone replacement therapy was continued, and the patient was nearly free from neurological deficits after 18 months. The neuroimaging abnormalities gradually improved with time [4].

A 13-year-old girl presented with an afebrile seizure followed by prolonged confusion and visual hallucinations. Initial investigations in the form of blood tests, cerebrospinal fluid analysis and head imaging by CT, were normal. She represented with two further episodes within a period of 3 weeks. Further investigations considering infective, metabolic and some autoimmune causes of encephalopathy were negative. An MRI head scan was normal. Thyroid function testing disclosed primary hypothyroidism and elevated antithyroid antibodies. She responded well to glucocorticoid therapy for presumed Hashimoto's encephalopathy (HE). HE describes patients with various neurological manifestations with elevated titres of antithyroid antibodies. There are no clear criteria for diagnosis, with many cases labelled as HE. Responses to corticosteroid therapy are favourable. In patients with unexplained encephalopathy, HE should be considered given the favourable response to glucocorticoid therapy [5].

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