

The encephalopathy as complication of Hashimoto thyroiditis in children: a wide variety of clinical manifestations

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Introduction

Hashimoto encephalopathy is an autoimmune encephalopathy, that is predominantly diagnosed in the adult population. In the pediatric population, the mean age is determined to be 14 years, with the majority of adolescent females. The diagnosis of Hashimoto encephalopathy is clinical and based on the highly variable neuropsychiatric conditions, often affecting more than one area of the central nervous system, the detection of antiperoxidase or antithyroglobulin antibodies in serum, and the elimination of other potential etiologies. The pathophysiology of Hashimoto encephalopathy is still unclear; autoimmune and vasculitic mechanisms can play an active role. A clinical response to corticosteroid therapy is supportive of the diagnosis. The occurrence of Hashimoto encephalopathy is unrelated to the patient's thyroid function status. Neuroimaging studies, EEG, and cerebrospinal fluid analysis can be supportive, although they are not diagnostic. MRI studies in children have mostly shown prolonged T2-weighted signals of the subcortical white matter, suggesting inflammation or demyelination.

The current standard treatment of Hashimoto encephalopathy is the use of corticosteroids in addition to the treatment of any concurrent dysthyroidism. Other options are immunoglobulins and plasmapheresis.

Case report

We describe the case of a 17-year-old girl who admitted due to headache, depression with amnesia and disorientation and anorexia nervosa with significant weight loss. She had a 8-year history of celiac disease. She had normal general and neurological examination, temperature was 36°C, heart rate 90 beats per minute, respiratory rate 17 breaths per minute, blood pressure 110/58 mm Hg. Height 160 cm (25-50 pc), weight 37 kg (<3 pc), and body mass index 14.5 kg/m² (< 3 pc). She had normal transglutaminase IgA antibody, significantly elevated thyroperoxidase (TPO) antibody titers 5.674U/mL in euthyroid function, consistent with the diagnosis of Hashimoto's thyroiditis. The analysis of cerebrospinal fluid showed high protein level and TPO antibodies. Cerebral MRI showed T2 hyperintensity in the periventricular white matter. Electroencephalography was normal. Steroid therapy was initiated with methylprednisolone 1 g intravenously per day for 3 days, followed by prednisone at a dose of 40 mg orally per day.

Results and Discussion

Encephalopathy as a complication of Hashimoto thyroiditis was first described by Brain and Coworkers in 1966 in the adult patient. In recent years have been increasingly recognized in both adult and pediatric patients. Particularly, the association between neuropsychiatric symptoms and Hashimoto thyroiditis should lead to early suspicion of this disorder. Further, this case report aims to raise awareness about the broad spectrum of clinical presentations of the Hashimoto encephalopathy especially in the presence of a known autoimmune disease and the importance of multidisciplinary approach for a early recognition.

References

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