Case Report

iMedPub Journals
www.imedpub.com

Archivos de Medicina
ISSN 1698-9465

2021
Vol.17 No.7:451

Encephalopathy Associated with Hashimoto's Thyroiditis: Case Report

Juan Sebastian Theran Leon*

Department of Epidemiology, University of Bucaramanga, Bucaramanga, Colombia

***Corresponding author:** Juan Sebastian Theran Leon, Department of Epidermology, University of Bucaramanga, Colombia, Tel: 3166316986;

E-mail: jtheran554@unab.edu.co

Received date: December 10, 2021; **Accepted date:** December 24, 2021; **Pusblished date:** December 31, 2021

Citation: Theran Leon JS (2021) Encephalopathy Associated with Hashimoto's Thyroiditis: Case Report. Arch de Medi Vol.17 No.7

Abstract

Hashimoto's Encephalopathy (HE) is a rare disease with variable clinical manifestations and without imaging or laboratory pathognomonic diagnostic criteria. It has been found to be associated with high serum titers of Thyroid Peroxidase antibodies (TPO) and in many cases thyroid function is normal. Usually has a favorable response to steroid treatment.

The case of a 31-years old woman with encephalopathy associated with steroid-responsive autoimmune thyroiditis is presented.

Keywords: Autoimmune encephalopathy; Hashimoto's dise ase; Hashimoto's thyroiditis; Hypothyroidism; Autoantibodies

Introduction

Hashimoto's encephalopathy, also known as corticosteroid-responsive encephalopathy, is pathology of autoimmune nature, with a prevalence of 2.1 per 100,000 inhabitants, mainly between 45 and 55 years of age and predominantly in the female sex [1]. It is considered as a discard diagnosis after excluding encephalopathy of infectious, toxic, neoplastic and metabolic origin [2]. It is manifested by cognitive deterioration accompanied by neuropsychiatric characteristics (hallucinations, delusions or paranoia), tonic-clonic seizures or focal neurological deficits [3] and its diagnosis can be established with the identification of anti-TPO antibodies in serum, euthyroid state or mild hypothyroidism, absence of infection, intoxication or neoplastic processes in laboratory tests (blood, urine or cerebrospinal fluid), absence of structural lesions in neuroimaging and complete or almost complete return to neurological baseline after corticosteroid therapy [4].

Next, a clinical case of HE in a young adult patient with satisfactory evolution is presented.

Presentation of the Case

A 31-year-old female patient with a history of Hashimoto's thyroiditis treated with thyroidectomy (09/2016) with poor adherence to pharmacological management and a history of acute delusional disorder secondary to autoimmune thyroiditis and mood disorder. She is taken to a one-day clinic with a tendency to muteness, isolation, easy crying, psychomotor agitation, palpitations and insomnia, she suspended levothyroxine five months ago; at admission in regular general conditions, with hemodynamic and respiratory stability, at neuropsychiatric evaluation with poor visual contact, dysractile, illogical thinking, bradypsychia and a tendency to mutism, normal neurological examination without targeting. In initial paraclinics, poorly controlled hypothyroidism (ultrasensitive TSH 36.32 uIU/ml, free T4 10.45 pmol/L), positive anti-TPO antibodies (488 IU/mL) and negative antithyroglobulin antibodies (0.016 IU/mL), sodium, potassium, Nitrogen tests and hemogram in normal ranges, thyroid ultrasound concludes changes due to thyroidectomy and the rest of the study without alterations; due to the symptoms presented and the positivity of anti-TPO antibodies, treatment was administered with pulses of methylprednisolone 500 mg intravenously each day, levothyroxine 150 mcg orally each day and concomitant management with quetiapine, after five days the patient presented clinical improvement and was He received a report of a confirmed magnetic resonance of the brain without alterations, for which he was discharged with treatment and outpatient follow-up.

Discussion

HD is a rare autoimmune clinical entity of an autoimmune nature that involves the central nervous system and is mainly associated with the presence of antithyroid antibodies [5]. Although there are no globally accepted diagnostic criteria for HE, this is based on the presence of clinical manifestations with at least one frankly elevated antithyroid antibody (anti-TPO greater than 200 micro IU/mL), in addition to the exclusion of pathologies typical of the central nervous system (tumor, infectious, autoimmune), as well as metabolic pathologies and paraneoplastic syndromes [6-7]; the study of cerebrospinal fluid in 80% of the cases is normal and in some it may present an elevation of proteins that normalize after management [3], eventually antithyroid antibodies can be found in CSF, however, its role in the EH and may remain elevated after the patient's recovery [8], imaging-wise, it is common to find electroencephalogram (EEG) abnormalities and decreased cerebral blood flow on positron emission computed tomography of the brain, while it is It is rare to find abnormalities in the magnetic resonance imaging of the skull, which is normal in 50% of cases and in case of alterations show atrophy, cerebral edema, ischemic lesions and demyelination in T2 and FLAIR

sequence [5,15]. Ischemic lesions can be diffuse and reverse with treatment plus the decrease in serum levels of anti-TPO antibodies [9-10]. The treatment of HE aims to control the autoimmune process and avoid complications, in this way, immunomodulatory management is based on intravenous corticosteroids whose first line is the administration of intravenous methylprednisolone for three to seven days that is continued with high doses dose of oral prednisone and progressive decrease according to improvement of symptoms, likewise, from the beginning of management, thyroid replacement with levothyroxine should be guaranteed [8,11], although, in most cases recovery is complete with treatment, the risk of relapse at two years of follow-up can be 12.5 to 40% of cases [11].

Our case is about a young patient with relapsed HE who meets the clinical, autoimmune criteria, no neuroimaging alterations and the symptomatic improvement after treatment, in the beginning the base mood generated confusion that warranted treatment guided by psychiatry. but it is emphasized that HE, in addition to cognitive deterioration, can lead to alterations in the state of behavior and personality [12], which supports the diagnosis of the patient presented.

It is concluded that HE is an unusual and poorly understood disease, for which it may be thought that it is underdiagnosed, which warrants a high clinical suspicion for its timely determination and treatment, therefore, in cases of unexplained encephalopathy, especially in women, it is It is prudent to think about this entity and request anti-TPO antibody titers which are necessary for the diagnosis of HD given that the thyroid function in most patients is normal, however, care must be taken given the high prevalence of elevated levels of anti-thyroid antibodies in the healthy population [13] with the consequent invitation to think about whether this type of encephalopathy is related to anti-TPO antibodies or is it an autoimmune disease of brain origin.

References

1. Yu HJ, Lee J, Seo DW LM (2014) Clinical manifestations and treatment response of steroid in pediatric Hashimoto encephalopathy. J Child Neurol 29:938-942
2. Montagna G, Imperiali M, Agazzi P, D'Aurizio F, Tozzoli R, Feldt-Rasmussen U GL (2016) Hashimoto's encephalopathy: A rare proteiform disorder. Autoimmun Rev 15:466-476
3. Zhou JY, Xu B, Lopes J, Blamoun J, Li L (2017) Hashimoto encephalopathy: literature review. Acta Neurol Scand 135:285-290
4. Castillo P, Woodruff B, Caselli R, Vernino S, Lucchinetti C, Swanson J, et al. (2006) Steroid-responsive encephalopathy associated with autoimmune thyroiditis. Archives of neurology 63:197-202
5. Cabrera EE, Enríquez CG, Carrillo TD, Clemente PA (2017) Encefalopatía asociada a distiroidismo con anticuerpos antiperoxidasa positivos. Caso clínico y revisión de la literatura. Rev Mex Neurocienc 18:79–87
6. Pinedo TI, Paz Ibarra JL (2018) Conocimientos actuales en encefalopatía de Hashimoto: revisión de la literatura. Medwave 18:e7298
7. Mattozzi S, Sabater L, Escudero D, Ariño H, Armangue T, Simabukuro M, et al. (2020) Hashimoto encephalopathy in the 21st century. Neurology 94:217-224
8. Farrell R, Foster M, Omoruyi A, Kingery S, Wintergerst K (2015) Hashimoto's encephalopathy: a rare pediatric brain disease. J Pediatr Endocrinol Metab 28:721-724
9. Yoneda M (2018) Hashimoto's Encephalopathy and Autoantibodies. Brain Nerve 70:305-314
10. Chen N, Qin W, Wei C, Wang X, Li K (2011) Time course of Hashimoto's encephalopathy revealed by MRI: report of two cases. J Neurol Sci 300:169-172
11. Gauthier AC, Baehring JM (2017) Hashimoto's encephalopathy mimicking Creutzfeldt-Jakob disease. J Clin Neurosci 35:72-73
12. Goh K, Chiu YH, Shen W (2014) Hashimoto's encephalopathy mimicking presenile dementia. Gen Hosp Psychiatry 36:360-e9
13. Kishitani T, Matsunaga A, Ikawa M, Hayashi K, Yamamura O, Hamano T, et al. (2017) Limbic encephalitis associated with anti-NH2-terminal of α-enolase antibodies: a clinical subtype of Hashimoto encephalopathy. Medicine. 96