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REVIEW ARTICLE

Relationship between Hashimoto's encephalopathy and neuropsychiatric symptoms

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ABSTRACT

Introduction: Hashimoto's encephalopathy is an uncommon autoimmune disorder, characterized by the presence of antithyroid antibodies and diverse neuropsychiatric manifestations, which complicates timely diagnosis and clinical management.
Objective: to describe the relationship between Hashimoto's encephalopathy and neuropsychiatric symptoms.
Methods: a descriptive and retrospective systematic review was conducted between 2019 and 2024. The search was performed in international databases (Medline, Embase, Pubmed, Cochrane, Dare), using DeCS and MeSH terms related to hashimoto's encephalopathy, autoimmune encephalopathy, corticosteroids, and neuropsychiatric symptoms. Defined inclusion and exclusion criteria were applied, selecting relevant studies for an appropriate critical analysis.
Development: Hashimoto's encephalopathy is associated with seizures, cognitive decline, psychotic episodes, and behavioral disturbances. Its pathophysiology involves Th17 cells, secretion of proinflammatory cytokines, and activation of immune pathways. Diagnosis relies on excluding other encephalopathies, detecting antithyroid antibodies, and complementary studies. A multidisciplinary approach and early clinical suspicion are essential to optimize prognosis. Corticosteroid therapy shows high efficacy, although up to 25% of patients may be refractory, requiring intravenous immunoglobulins, plasmapheresis, or immunosuppressants.
Conclusions: despite its low prevalence, Hashimoto's encephalopathy represents a diagnostic challenge due to its similarity with primary psychiatric disorders. The identification of antithyroid antibodies and the response to corticosteroids are key diagnostic and therapeutic pillars. Integrating immunological and neuropsychiatric strategies is essential to improve clinical outcomes and prevent recurrences.

Keywords: Brain Diseases; Autoimmune Diseases; Mental Disorders.

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INTRODUCTION

Hypothyroidism is the most common endocrine disorder due to hormonal deficiency and a frequent reason for seeking medical attention, with Hashimoto's thyroiditis being its main cause. Described in 1912, this organ-specific autoimmune disease is characterized by lymphocytic infiltration of thyroid tissue, alterations in cytokine secretion, and progressive destruction of follicles, leading to hypothyroidism. According to the World Health Organization, its prevalence ranges from 1 % to 20 % in the general population, with a higher frequency in women and older adults. Hashimoto's encephalopathy, described in 1966, is a rare neurological manifestation of probable autoimmune origin, with a female predominance, association with other autoimmune diseases, and increasing relevance in the differential diagnosis of encephalopathies of uncertain etiology.[1,2]

The etiology of this condition is unknown, and it is classified as an autoimmune disorder. Humoral cells involved in the pathogenesis of thyroid hypersensitivity (TH) play a dominant role, with helper T cells (Th), specifically Th17 cells, producing IL-17A, a pro-inflammatory factor secreted in other inflammatory and autoimmune diseases such as multiple sclerosis, rheumatoid arthritis, type 1 diabetes, lupus erythematosus, psoriasis, and Sjögren's syndrome. This has led some authors to question whether it is better to rename it autoimmune thyroid disease-associated encephalopathy. This would better explain its etiology and allow us to link it to antibodies of thyroid autoimmunity, and not only to those related to Hashimoto's thyroiditis (anti-TPO).[3]

All cases presenting with acute or chronic encephalopathy are characterized by encephalopathy associated with neuropsychiatric symptoms such as altered consciousness, impaired attention, and changes in behavior and personality. Memory or language impairments may be present in 80 % of cases, and seizures may occur in 60-70 %. Studies of patients have shown that 25-30 % experience stroke-like episodes characterized by sensory or motor deficits and a high production of positive antithyroid antibodies. The most common form of evolution is with relapses and remissions (50 %), and gradual and insidious (40 %).[3]

The complementary tests are unremarkable, with blood work usually normal except in cases of associated encephalopathy and Hashimoto's thyroiditis, which detect markers of autoimmune disease. Among the antithyroid antibodies, antimicrosomal and anti-thyroid peroxidase antibodies at titers greater than 100 times the normal value are the most specific and are present in 100 % of cases; antithyroglobulin antibodies are found in 70 % of these. The most frequent EEG shows slow waves, triphasic waves, or epileptic disturbances. Neuroimaging tests are nonspecific. Therefore, the diagnosis is made by exclusion, once the most common causes of encephalopathy have been ruled out and antithyroid antibodies are detected.[4]

Hashimoto's encephalopathy usually responds favorably to high-dose corticosteroid treatment, with prednisolone typically used at a dose of 1–2 mg/kg. Lack of response is associated with atypical presentations, concomitant neurodegenerative diseases, or other alternative diagnoses. Although corticosteroid therapy is generally effective, relapses can occur after abrupt discontinuation, sometimes necessitating prolonged treatment. In patients with a partial response or refractoriness to corticosteroids, immunomodulatory therapies such as azathioprine, intravenous immunoglobulins, or plasmapheresis have been described as useful. Comprehensive management includes supportive care, seizure control with antiepileptics, and management of neuropsychiatric symptoms with atypical antipsychotics.[5]

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It is necessary for healthcare professionals to have the necessary skills for its diagnosis and treatment due to the complications that its lack of control generates; therefore, the objective of this review isdescribe the relationship between Hashimoto's encephalopathy and neuropsychiatric symptoms.

METHODS

A systematic, descriptive, and retrospective literature review was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The search and selection of scientific evidence took place between June 2019 and June 2024, with the aim of identifying relevant studies related to Hashimoto's encephalopathy, its autoimmune basis, and associated neuropsychiatric manifestations, as well as therapeutic responses to steroids.

Information sources included electronic databases recognized for their scientific rigor: MEDLINE (via PubMed), Pre-MEDLINE, Embase, the Cochrane Library, and the Database of Abstracts of Reviews of Effects (DARE). Additionally, the reference lists of selected articles were manually scanned to identify relevant further studies. Furthermore, indirect consultation of relevant secondary literature and expert opinion in the fields of neurology and endocrinology were considered to ensure broad and up-to-date coverage of the topic. Publications in both Spanish and English were included.

The search strategy was structured using DeCS and MeSH controlled terms combined with Boolean operators. The descriptors used were: Hashimoto's encephalopathy, autoimmune encephalopathy, steroids, and neuropsychiatric symptoms. These terms were combined using the "AND" and "OR" operators to maximize the sensitivity and specificity of the search. The algorithm was adapted to the specific characteristics of each database, maintaining semantic and methodological consistency.

The inclusion criteria included: studies published within the defined period; research directly addressing the clinical, diagnostic, pathophysiological, or therapeutic characteristics of Hashimoto's encephalopathy; and articles describing variables such as patient age, disease severity, inflammatory mechanisms, type of immunological intervention, clinical course, and outcomes related to diagnosis, classification, and prognosis. Duplicate studies, articles without full-text access, publications outside the time frame, research irrelevant to the study objective, and studies focused on sociological factors, patient status, or other autoimmune pathologies unrelated to the hormonal autoimmunity under study were excluded.

The selection process was carried out in several phases. Initially, 91 records were identified in the databases, of which 60 remained after duplicate removal. Subsequently, a review of titles and abstracts led to the exclusion of 20 studies for not meeting the established criteria. Ten additional articles were discarded due to unavailability of the full text or the impossibility of translation. Finally, after a detailed evaluation of 37 full texts, 28 studies met the inclusion criteria and were incorporated into the review. Two additional studies were identified through a reference review. Data extraction included author, year, study design, sample characteristics, and main results. A qualitative synthesis of the evidence was performed, without meta-analysis.

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DISCUSSION

Numerous immune processes and their associated symptoms are related to autoimmune disorders in patients with autoimmune encephalitis. Each of these mechanisms plays a crucial and consequential role in triggering these alterations. Essentially, all of this is interconnected with the patient's presentation and their reaction to the disability inherent in their hospitalization. Depending on these factors, a better understanding of the body's condition can be achieved, as Goertzen,[6] describes, highlighting some neurological alterations in autoimmune processes that are becoming increasingly common.

One of the predisposing factors and the prognosis in patients with autoimmune encephalitis depends on status epilepticus. A study conducted in the inpatient department of the West China Hospital of Sicchuan University reflects a 22 % risk of patients experiencing moderate or severe status epilepticus. Zhang et al.,[7] indicate that, in patients undergoing intervention, Glasgow Coma Scale scores <8 points, abnormal EEG, delayed immunotherapy, and status epilepticus lasting >30 minutes are considered risk factors for a poor prognosis.

There is a strong association between autoimmune disorders and Hashimoto's thyroiditis, as they are linked in one way or another. Ríos-Duarte et al.,[8] in their article, clarify certain related pathologies, such as type 1 diabetes, rheumatoid arthritis, systemic lupus erythematosus (SLE), autoimmune thyroiditis, Addison's disease, and systemic sclerosis (SSc). In other words, there is a 95 % confidence interval indicating a marked exacerbation when several autoimmune disorders are present or are the main triggers of the disease. Non-cutaneous comorbidities (43 %) are also significant, with larger associated conditions, followed by pernicious anemia (31 %) and autoimmune thyroiditis (31 %). Vitiligo stands out in 12 % of cases as a risk factor for autoimmune encephalitis.

Given the close relationship with neurological and autoimmune processes, there is a possibility that the patient may develop psychotic features. Wallengren et al.,[9] published a clinical case of an N-methyl-D-aspartate receptor disorder that initiates a neuropsychiatric phase in children and adolescents. The symptoms of this phase include abnormal behavior, seizures, and neurological symptoms. Notably, paranoid delusions may be present, in which antipsychotic treatment has no effect.

On the other hand, it is important to consider the differential diagnosis in patients with a history of hypothyroidism. Anti-N-methyl-D-aspartate receptor (NMDA-R) encephalitis helps differentiate it from psychosis. Sitterley et al.,[10] present a clinical case where the symptoms began with headache, and neuroimaging showed a small right frontal hyperintensity on fluid-attenuated inversion recovery images. Interestingly, the patient improved rapidly with steroids, but the onset of psychiatric symptoms remained unexplained. Cerebrospinal fluid analysis was positive for antithyroid peroxidase and NMDA-R antibodies. The patient's condition improved after the administration of intravenous immunoglobulin (IVIG) and rituximab.

It is important to mention the differentiation between autoimmune encephalitis and schizophrenia spectrum disorders. This distinction is highly controversial, as some psychiatric symptoms cannot be detected by laboratory tests or imaging studies because these are unavailable. In this approach, Schneider's first-rank symptoms (hallucinations through verbal comments and delusional experiences) were noted in schizophrenia, while altered perception was more frequently found in autoimmune encephalitis. Funayama et al.,[11] explain that Schneider's first-rank symptoms and altered perception, along with neurological and neuropsychological signs, functional status, and past history, can help accurately differentiate these two conditions in patients experiencing a first episode of psychosis.

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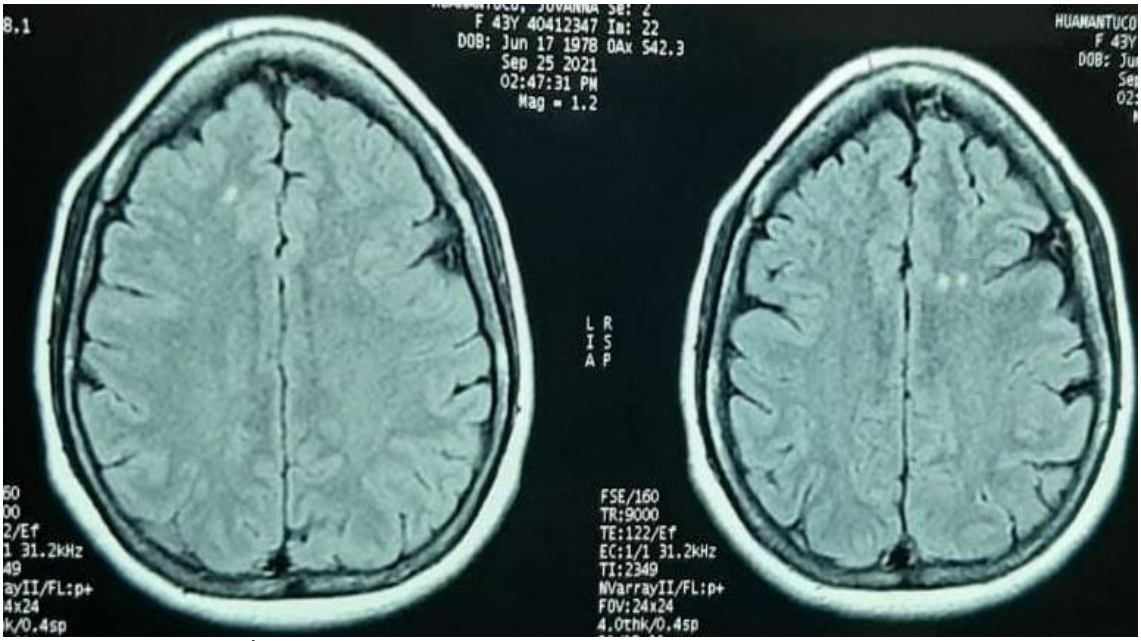
Morioka, et al.,[12] noted in their publication on hospitalized COVID-2 patients who were isolated that they experienced psychiatric disorders and stress in their environment, in addition to their anxieties. They determined that any emotional burden, however small, that causes excessive worry in hospitalized patients will end up being an even greater risk factor when they have an autoimmune disorder that is exacerbated. A crucial factor is the abandonment of hospitalized patients, as they may suffer discrimination, prejudice, and suspensions and even job losses, triggering a worsening of their autoimmune processes.

Clinical presentations with psychiatric characteristics can occur, leading to misdiagnosis and mistreatment. According to Schaller,[13] Hashimoto's encephalopathy presents similarly to paranoid schizophrenia due to its clinical presentation. Consequently, he proposes a thorough medical history in patients with psychiatric symptoms to facilitate early initiation of antipsychotic treatment. Ching-Heng et al.,[14] argue that a history of bipolar disorder followed by catatonia, altered consciousness, and general weakness after a worsening of depressive symptoms should raise suspicion of Hashimoto's encephalopathy, as the neuropsychiatric symptoms mimic a previous mental illness and resolved after receiving pulse steroid therapy.

Defining the psychological symptoms associated with Hashimoto's encephalopathy is crucial for its diagnosis. Ortiz Arce ,[15] notes in his article that altered consciousness, visual hallucinations, and delusions should not be ruled out. Therefore, laboratory tests should include antiperoxide antibodies, thyroid hormone, thyroxine, thyroid-stimulating hormone, and magnetic resonance imaging (MRI) showing bilateral subcortical focal lesions with a nonspecific demyelinating appearance (Fig. 2). An electroencephalogram (EEG) should also be unremarkable. Surpreet et al.,[16] present a clinical case involving generalized tonic-clonic seizures and worsening respiratory status requiring intubation and tracheostomy.



Fountain: Ortiz Arce AD, Sánchez-Rueda H. Debut with psychotic symptoms of Hashimoto encephalopathy: A case report. Medwave. 2022;22(9):e2566.

Fig. 2 Bilateral subcortical focal lesions with a nonspecific demyelinating appearance on magnetic resonance imaging.

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Tomoyuki,[17] characterizes in his article the antibodies against Hashimoto's encephalopathy, as it is one of the autoimmune diseases associated with psychiatric illnesses. There is a high prevalence of antibodies against the N-terminal subunits of the N-methyl-D-aspartate (NMDA) glutamate receptor (GluR). This means that the detection of antithyroid antibodies in psychiatric patients could be a clue for considering certain psychiatric conditions related to antibodies against (GluN1). For his part, Howarth,[18] highlights that anti-NMDA receptor autoimmune encephalitis (NMDARE) is a common pediatric encephalitis that produces neuropsychiatric symptoms. Therefore, objective cognitive assessments in children with this pathology are lacking.

To corroborate the aforementioned information, Yin et al.,[19] published a study on the usefulness of FDG-PET/CT in pediatric patients with suspected autoimmune encephalitis. The children underwent blood sampling, cerebrospinal fluid (CSF) sampling, EEG, MRI, and FDG-PET/CT. The diagnostic criteria for autoimmune encephalitis using FDG-PET/CT were large lobar hypometabolism with or without focal hypermetabolism found on PET/CT, due to its high specificity, sensitivity, and accuracy for diagnosis. Furthermore, Tjong,[20] established a clinical use for quantitative electroencephalography to aid in the diagnosis of steroid-sensitive encephalopathy associated with autoimmune thyroiditis and supported the view that it is a spectrum disorder with protein manifestations.

Hashimoto's encephalopathy often does not respond to steroid treatment in 25 % of cases, particularly when associated with thyroid dysfunction (SREAT), in the context of intermittent cognitive impairment. It is worth noting that it can develop even in patients with a history of partial thyroidectomies. In conclusion, the study published by Adithya-Sateesh,[21] states that timely treatment with glucocorticoids is vital, and a positive response confirms the diagnosis. Maretta,[22] recommends other immunosuppressive therapies such as plasmapheresis, intravenous immunoglobulins, or others when glucocorticoids are not effective, as is the case for most patients.

As described by several authors, patients with steroid-responsive encephalopathy (SREAT) are a common cause of psychosis. A study by Runge,[23] reports a female patient with SREAT whose psychosis continued to worsen despite the gradual reduction of cortisone. This highlights the difficulty in discontinuing steroid treatment and illustrates alternative treatments such as plasmapheresis and rituximab. In another instance, Sorodoc,[24] mentions intravenous immunoglobulin therapy as a primary alternative, citing its proven effectiveness in treating Hashimoto's encephalopathy in 65 % of cases compared to the other therapies mentioned previously.

Key guidelines for the diagnosis and treatment of autoimmune encephalitis include assessment of clinical presentation, brain imaging, cerebrospinal fluid (CSF) findings, antibody detection, and electroencephalography (EEG) findings. Elkhider,[25] notes antibodies against membrane surface antigens, as well as anti-glutamic acid decarboxylase (anti-GAD) antibodies. This indicates that early diagnosis of these disorders requires a high level of suspicion to avoid delaying diagnosis. Jegatheeswaran,[26] reports significant abnormal findings on magnetic resonance imaging, including signal abnormalities in the claustrum, cerebral white matter, and medial temporal lobes.

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We must not forget to mention the coronavirus disease 2019 (COVID-19) detection system published by Kim et al.,[27] in their scientific journalThey point out the importance of preventive control of healthcare personnel and patients following COVID-19. They share the idea of abandonment, as proposed by Morioka,[12] since it is the first consequence of psychotic symptoms and features in hospitalized patients with autoimmune disorders. Elsewhere, Matsumura,[28] not only emphasizes a certain ideology, but also human tolerance in the face of critical situations in hospitalized patients, especially those with cognitive impairments, such as those with Hashimoto's encephalopathy.

CONCLUSIONS

Hashimoto's encephalopathy originates from complex immune processes associated with alterations in the autoimmune response, the proper interpretation of which is essential for understanding its clinical presentation. The co-occurrence of neuropsychiatric manifestations, such as seizures, delirium, and cognitive impairment, contributes to the diagnostic challenge, as these symptoms can mask the autoimmune nature of the disease and are due to diverse pathophysiological mechanisms. In this context, a comprehensive understanding of the interaction between immune processes and neurological manifestations is fundamental for establishing an accurate diagnosis. Treatment is primarily based on the use of corticosteroids, glucocorticoids, and intravenous immunoglobulins—therapies widely used in autoimmune diseases—aimed at controlling immune dysfunction and optimizing the patient's clinical course.

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