

Debut with psychotic symptoms of Hashimoto encephalopathy: A case report

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Abstract

Hashimoto's encephalopathy is a rare disease, first reported in 1966, with a prevalence of 2.1 in 1000 inhabitants. We present the case of a 42-year-old woman, with no relevant medical history, who suddenly started having symptoms of altered consciousness, visual hallucinations and delusions. Laboratory tests showed anti-thyroperoxidase antibodies greater than 600 U/ml, thyroxin 0.93 U/ml, and thyroid stimulating hormone 1.60 U/ml. Magnetic resonance imaging showed bilateral subcortical focal lesions with a nonspecific demyelinating appearance. The electroencephalogram was nonspecific. The diagnosis of Hashimoto encephalopathy was made, and symptoms remitted after treatment with steroids. This article highlights the importance of conducting a comprehensive evaluation of patients with atypical psychiatric symptoms and a thorough differential diagnosis.

MAIN MESSAGES

- ◆ Hashimoto's encephalopathy is a differential diagnosis in cases of sudden onset psychosis without premorbid mental pathology.
- ◆ Further long-term follow-up of the this case is not detailed.
- ◆ Psychiatric manifestations such as nihilistic delusions, delusions of persecution and bizarre delusions can part of Hashimoto's encephalopathy onset.

INTRODUCTION

Hashimoto's encephalopathy is associated with Hashimoto's disease and was first described in 1966. This disease is believed to be an immunologically mediated disorder that affects the central nervous system independently from low thyroid hormone levels [1]. Its exact pathophysiology is unknown, but most evidence points to an autoimmune vasculitis or other inflammatory processes, possibly associated with immune complex deposition and alteration of the cerebral microvasculature. Like most autoimmune diseases, it is more frequent in women with a ratio of four to one [1], and it can be concomitant with other autoimmune pathologies in up to 30% of patients [2].

Hashimoto's encephalopathy has been seen to occur independently of Hashimoto's disease. As it is linked to an increase in autoimmune antibodies related to thyroid pathology beyond the antithyroid peroxidase antibody, some propose a different

eponym: steroid-sensitive encephalopathy associated with autoimmune thyroiditis [3].

Its clinical presentation is characterized by neuropsychiatric symptoms of varying intensity that, in some cases, may end in a coma. Neurological symptoms include stroke-like episodes, seizures, dementia, myoclonus, and myelopathies compatible with nonspecific encephalopathies [3]. Among the psychiatric symptoms, the most frequent are sensory-perceptual disturbances but behavioral and thought disturbances may also be present [4]. Finding at least one frankly elevated antithyroid antibody is a fundamental criterion. The literature indicates that the antithyroid peroxidase antibody should be greater than 200 milli-international units per milliliter (more than five times the average value). Some authors even place a cut-off point of 500 milli-international units per milliliter [5]. The first line of treatment is corticosteroids at high doses for 3 to 5 days, associated with drugs for disease complications and most patients show complete improvement after management with corticosteroids [2]. We present the clinical case of a female patient who presented with Hashimoto's encephalopathy with a psychotic episode as a debut.

Table 1. Laboratory tests.

Laboratory tests	Values
Hemoglobin	15 gr%
Hematocrit	43.4%
Platelets	311 x mm ³
Leukocytes	6.9 x mm ³
Banded neutrophils	0%
Segmented neutrophils	73%
Eosinophils	0%
Basophils	0%
Lymphocytes	18%
Monocytes	9%
Glucose	99 mg/dl
Creatinine	0.65 mg/dl
Urea	27 mg/dl
Chlorine	104 mmol/L
Potassium	3.5 mmol/L
Sodium	140 mmol/L
C-reactive protein	1.3 mg/L

gr%: grams percent. mm³: cubic millimeters. mg/dl: milligrams per deciliter. mmol/L: millimoles per deciliter. mg/L: milligrams per liter.
Source: Prepared by the authors of this study.

CASE REPORT

For the report of this case, informed consent and institutional authorization were requested.

This is the case of a 42-year-old woman of mixed race, cohabitant, and housewife with no relevant family or medical history. Four months before her admission, she presented with constant headaches that partially resolved with outpatient analgesics. In September 2021, she had an emergency consultation at the medical service of Goyeneche Hospital with a sudden episode of confusion, psychomotor agitation, and sensory-perceptual disturbances (auditory and visual hallucinations). Clonazepam and mannitol were administered, suspecting a cerebral vascular accident. Since there was no favorable response, hospitalization was decided.

Physical examination showed normal vital signs with no other particularities. The mental examination showed a confused patient, disoriented in time and space and partially in person, with incoherent language and thoughts, the presence of nihilistic delusions ("My son is dead", "My mother is dead", "They

Table 2. Cerebrospinal fluid biochemistry.

Cerebrospinal fluid biochemistry	Values
Volume	2 ml
Color	Colorless
Appearance	Transparent
Cell count	2 x mm ³
Mononuclear	0/μL
Polymorphonuclear	100/μL
Leukocytes	0-1/μL
Red blood cells	0-1/μL
Normal border	90/μL
Crenulated border	10/ul
Germs	None
Glucose	61 mg/dl
Pandy	Negative
Proteins	26.7 mg/dl
Culture	Negative

mm3: cubic millimeter. mg/dl: milligrams oer deciliter. μL: microliter. ml: milliliter.

Source: Prepared by the authors of this study.

have taken all my blood"), delusions of persecution ("The police are looking for me"), bizarre delusions ("How they are going to glue the mouse skin on me? They have scraped my whole body and put blond wool on me, I am a brunette", "My head is empty, light, flat, they have put much garbage in me, they have put glass in me"), pejorative auditory hallucinations, cenesthetic hallucinations ("I have excrement behind my head, clean it"), affective incontinence, hypoprosxia, dyscalculia, and null judgment and introspection.

In the neurological examination, anosmia was found without alteration of cranial pairs, and the rest of the neurological examination was normal. Paraclinical examinations were requested (Table 1) to rule out infection or electrolyte alteration, with results within normal parameters. A lumbar puncture was performed to rule out a neurological compromise, and

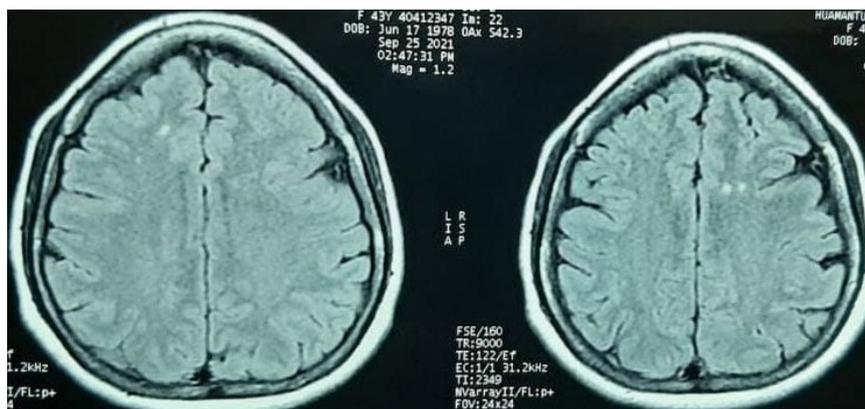
resulted in normal cerebrospinal fluid (Table 2). The electroencephalogram showed no particularities, and the computed axial tomography showed a normal brain. Magnetic resonance imaging of the brain showed bilateral focal subcortical lesions with a nonspecific demyelinating aspect (Figure 1), so serological, immunological, and biochemical tests were requested. Tumor markers were negative, thyroid-stimulating hormone (TSH), thyroxine (T4), and thyroglobulins were normal. The antithyroid peroxidase antibody was elevated (600 U/ml) (Table 3). Even though the patient was clinically euthyroid, these findings supported the diagnosis of Hashimoto’s encephalopathy.

With a presumed diagnosis of Hashimoto encephalopathy, the patient started treatment with prednisone 50 milligrams per day for seven days, followed by methylprednisolone one gram per day for three days. During hospitalization, remission of neuropsychiatric symptoms was observed, so the patient was discharged with outpatient follow-ups, which she did not attend.

DISCUSSION

Hashimoto’s encephalopathy is a clinical entity that may present with multiple neuropsychiatric manifestations and represents a challenge for diagnosis and treatment. The age of presentation of the present case is similar to findings from a systematic review that analyzed 46 cases of Hashimoto’s encephalopathy, finding an age range of presentation at 50 years [6]. The clinical presentation, acute onset, and debut with profound atypical psychiatric manifestations (nihilistic delirium, delirium of persecution, bizarre delirium), associated with anomia, are not very frequent in the reported literature [7,8]. Chang, Jan Shun, and colleagues report three cases in which there is a predominance of visual and auditory hallucinations and, in one of them, persecution delusions associated with frank alterations of the thyroid profile and subsequent evident clinical manifestations [9]. Likewise, Amamou et al. report a case of a 39-year-old male patient who presented hypochondriac delirium and depressive

Figure 1. Axial view of magnetic resonance imaging of the brain, with bilateral focal subcortical lesions of nonspecific demyelinating appearance.



Source: Prepared by the authors of this study.

Table 3. Immunological tests.

Immunological tests	Values
Procalcitonin	0.04 ng/ml
Lactate dehydrogenase	210 μ I/L
Complement C3	133 mg/dl
Complement C4	29 mg/dl
Antithyroid peroxidase antibodies	> 600 U/ml
CA 125	21.95 U/ml
CA 15-3	7.35 U/ml
CA 19-9	10.21 U/ml
CA 72-4	0.79 U/ml
Carcinoembryonic antigen	3.55 U/ml
Thyroxine	0.86 U/ml
Thyroid-stimulating hormone	4.52 U/ml
Vitamin B12	303.6 U/ml
Parathormone	37.9 U/ml

ng/ml: nanograms per milliliter. μ I/L: microliter per liter. U/ml: international units per milliliter. mg/dl: milligrams per deciliter.
Source: Prepared by the authors of this study.

symptoms [8]. Churilov L et al. reported 17 cases of Hashimoto's encephalopathy in patients with schizophrenia, in whom phobias and sleep disorders were identified in up to 52% and hallucinations in 18% of the series of cases studied [7]. Case reports with similar demographic characteristics highlight the history of previous thyroid or psychiatric pathologies associated with this entity [10,11]. Our report highlights the onset of encephalopathy with no previous medical history.

An elevated level of antithyroglobulin antibodies, in addition to cerebrospinal fluid studies, electroencephalogram, and imaging studies without significant alterations, are criteria that corroborate our clinical suspicion [7]. Finally, the recommended treatment guidelines were applied [2,10], obtaining a clinical improvement in the patient.

One of the limitations of our study is that we do not have a long-term follow-up of the presented case and lack of exclusion of other autoimmune pathologies.

We highlight the importance of comprehensively evaluating patients with atypical psychiatric symptomatology and deepen the differential diagnoses.

CONCLUSIONS

We recommend having special attention to sudden onset psychosis in patients without premorbid mental pathology. We also emphasize the importance of taking a detailed clinical history that will provide valuable information about the origin of psychosis since organic etiology should always be present in a differential diagnosis of psychiatric pathology and to reach a timely diagnosis and treatment.

Notes

Contributor roles

ADOA, HSR performed the conceptualization, literature analysis, writing, review, editing, final approval of the version to be published, and commitment to be responsible for all aspects of the report.

Competing interests

The authors declare that there are no conflicts of interest.

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Ethics

Informed consent was signed and institutional authorization was obtained.

Data sharing statement

The authors declare their willingness to provide data on request.

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Debut con síntomas psicóticos de encefalopatía de Hashimoto: reporte de caso

Resumen

La encefalopatía de Hashimoto es una enfermedad rara reportada por primera vez en 1966 con una prevalencia de 2,1/1000 habitantes. Se presenta el caso de una mujer de 42 años, sin antecedentes médicos de importancia, quien inició un cuadro de manera súbita con alteración de conciencia, alucinaciones visuales y delusiones. En los exámenes de laboratorio se tuvo anticuerpos antiperoxidasa tiroidea mayor a 600 U/ml, tiroxina 0,93, hormona estimulante de la tiroides 1,60 U/ml, resonancia magnética con lesiones focales subcorticales bilaterales de aspecto desmielinizante inespecífico, electroencefalograma sin particularidades. Se realizó el diagnóstico de encefalopatía de Hashimoto y el cuadro remitió luego del tratamiento con corticoides. El artículo resalta la importancia de realizar una evaluación integral de los pacientes con sintomatología psiquiátrica atípica y ahondar en el diagnóstico de exclusión.



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