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Review

Status epilepticus in Hashimoto's encephalopathy

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ABSTRACT

Hashimoto's encephalopathy is a non-infectious, probably autoimmune encephalitis, characterized by varied signs coupled with elevated levels of anti-thyroid antibodies and, often, good response to corticosteroid therapy. Seizures, namely focal and generalized tonic-clonic seizures, myoclonus, and status epilepticus, are frequent manifestations of Hashimoto's encephalopathy. Typically, seizures in these patients respond poorly to anti-epileptic drugs. Although cases of Hashimoto's encephalopathy with status epilepticus have been reported in literature, they vary in demographic, clinical, and treatment characteristics. We could not identify any systematic review summarizing the evidence in regard to factors predicting the occurrence of status epilepticus in Hashimoto's encephalopathy and the responsiveness of status epilepticus to anti-epileptic drugs, steroids and other immunomodulatory medication. Therefore, we performed an extensive review of the literature to identify and compare Hashimoto's encephalopathy patients presenting with and without status epilepticus. In 31 patients with status epilepticus and 104 patients without status epilepticus, thyroid status, anti-thyroid antibodies, cerebrospinal fluid analysis, brain MRI/CT/SPECT scan did not predict occurrence of status epilepticus of variable phenomenology. Status epilepticus did not respond to anti-epileptic drugs but completely remitted under steroid treatment, alone or in combination with other immunomodulatory medication, in about three quarter of patients. Generalized convulsive status epilepticus might be a factor negatively influencing outcome.

1. Introduction

Hashimoto's encephalopathy (HE), also known as steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT), is a rare disorder with an estimated prevalence of 2:100.000, a reported woman to men ratio of 4:1 [1], and a relatively greater frequency in the adult population [2].

HE is defined by signs and symptoms of an encephalopathy, exclusion of other causes (infectious, toxic, metabolic, or neoplastic processes), presence of elevated anti-thyroid antibodies (usually anti-thyroid peroxidase (TPO) antibodies and/or anti-thyroglobulin (TG) antibodies), a euthyroid or mildly hypothyroid state, no specific neuroimaging finding, and good clinical response to steroids [3,4].

HE may have a variety of acute/subacute presentations characterized by cognitive and neuropsychiatric manifestations, focal neurologic deficits and seizures [3]. Typically, seizures in HE patients respond poorly to anti-epileptic drugs (AED) even though, like other HE symptoms, they may respond well to steroid therapy [4]. Although cases of HE with status epilepticus (SE) have been reported in literature, they vary in demographic, clinical, and treatment characteristics [5–27]. We could not identify any systematic review summarizing the evidence in regard to factors predicting the occurrence of SE in HE and the responsiveness of SE to AED, steroids and other immunomodulatory medication. Owing to the low prevalence of HE, we addressed this knowledge gaps by reporting our own clinical observation of a patient who developed HE and by reviewing the existing reports of HE patients manifesting SE.

2. Materials and methods

A computer-assisted review of the literature using PubMed as the search engine was performed to identify relevant articles. The search was run from January 1966 (the year of the first description of HE) [28] to March 2019. The terms Hashimoto's encephalopathy, Hashimoto's thyroiditis, Hashimoto's disease, steroid-responsive encephalopathy associated with autoimmune thyroiditis, SREAT, hypothyroidism, and autoimmune thyroiditis were used as key words, alone or in combination with terms like "status epilepticus" and "seizures". By application of predefined criteria, articles had to be original full texts describing patients with HE and acute/subacute onset of neurological symptoms. Cross-references of selected papers were also screened.

Overall, 179 papers were identified, including case reports and

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series, reviews, and letters to the editor. Eighty papers did not satisfy inclusion criteria or did not provide sufficient information about relevant demographic variables including age, gender, thyroid status, classification of SE, clinical features, presence of antithyroid antibodies, cerebrospinal fluid (CSF) and imaging finding and responsiveness to treatment. Ninety-nine articles describing 30 HE patients who presented with SE [5–27] and 104 HE patients manifesting symptoms other than SE [29–48; see also Appendix A] were finally analyzed.

Statistical analysis was performed by the Stata 11.0 package (Stata Corporation, College Station, TX). Data were expressed as mean and standard deviation (SD) unless otherwise indicated. Differences across groups were analysed by *t* test, Fisher test (two tailed) or Chi-squared test, as appropriate.

3. Results

3.1. Case report

A 35-year-old woman was admitted at our clinic because of two focal seizures with impaired awareness. On admission, intercritical electroencephalogram (EEG) and brain magnetic resonance imaging (MRI) were normal. In the following days, the patient insidiously manifested hallucinations, altered cognition, and impaired consciousness. Concomitantly, EEG showed diffuse, specific abnormalities consistent with non-convulsive status epilepticus (NCSE) that did not respond to diazepam, or oxcarbazepine or phenytoin. On brain MRI follow up, bilateral and asymmetric cortical thickening and increased T2/FLAIR signal intensity became evident in several areas, including mesial temporal lobes, limbic system, basal ganglia and frontoparietal lobes. MRI and laboratory investigations did not provide any evidence of infective, metabolic, toxic, degenerative, and neoplastic processes. Full-body computed tomography (CT) scan, mammography, breast ultrasonography, transvaginal ultrasound did not provide any evidence of neoplastic process. Thyroid hormone serum levels were normal, while serum anti-TPO-antibodies and anti-TG- antibodies were elevated. Serum autoantibodies against the NH2-terminal of alpha-enolase were not detected. Oligoclonal bands were found on CSF examination, whereas antibodies commonly associated with paraneoplastic and autoimmune encephalitis did not (antibodies anti-HU, anti-YO, anti-RI, anti-amphiphysin, anti-CV2, anti-PNMA2, anti-recoverin, anti-SOX1, anti-titin, anti-GAD65, anti-Zic4, anti-Tr, anti-NMDA, anti-AMPA1, anti-AMPA2, anti-VGKC, anti-CASPR2, and anti-LGI1). Intravenous methylprednisolone 1 g/day for five days followed by oral prednisone yielded remission of NCSE as well as of psychic and cognitive symptoms. After a few days, relapse of hallucinations and fluctuating consciousness (but not of NCSE) was successfully managed by intravenous immunoglobulin (IVIg, 0.4 g/kg/day for 5 days). MRI performed six months after clinical onset showed almost complete disappearance of brain lesions.

3.2. Analysis of HE patients with and without SE

The overall study group included 31 case patients with HE and SE (30 from the literature [5–27] and 1 from our clinic) and 104 control HE patients who presented with acute/subacute onset of symptoms other than SE. The two groups were comparable for sex (25 women and 6 men vs. 75 women and 29 men, p = 0.48) and age at HE (40.7 ± 23.3 years [range, 8–83] vs. 40.4 ± 20.1 years [range, 9–82], p = 0.94). In the SE group, generalized convulsive SE (GCSE) was reported in 16 patients (52%) [7,9,10–12,14,16–19,24–26], NCSE in 9 patients (29%) [6,15,20,21,23, and our case], focal SE (FSE) in 4 patients (13%) [5,22,26,27], generalized absence SE (GASE) in 1 patient (3%) [8], and epilepsia partialis continua (EPC) in 1 patient (3%) [13]. In the non-SE group, the most frequently reported problems were psychiatric symptoms, seizures, cognitive abnormalities, and movement disorders [29–48; see also Appendix A].

Table 1
Clinical and laboratory findings in patients with Hashimoto's encephalopathy and status epilepticus or neurological symptoms other than status epilepticus.

	Patients with Status epilepticus (n. 31)	Patients with neurological symptoms other than status epilepticus (n.104)	P
Thyroid status [n. patients (%)]:			
Euthyroidism	12 (41%)	56 (55%)	
Clinical Hypothyroidism	7 (24%)	30 (29%)	
Subclinical Hypothyroidism	8 (28%)	9 (9%)	0.11
Clinical Hyperthyroidism	2 (7%)	6 (6%)	
Subclinical Hyperthyroidism	0	1 (1%)	
Not reported	2	2	
Anti-thyroid autoantibodies [n. patients (%)]:			
Anti-TPO and/or Anti-TG and/or NAE	31 (100%)	104 (100%)	
Anti-TPO	9 (29%)	38 (36%)	0.44
Anti-TG	3 (10%)	10 (10%)	1
Anti-TPO + anti-TG	19 (61%)	51 (49%)	0.23
Anti-TPO + anti-TG + Anti-NAE	0	5 (5%)	
Not reported	0	0	
Cerebrospinal Fluid analysis [n. of patients (%)]:			
Normal	11 (41%)	32 (36%)	0.65
Abnormal	16 (59%)	57 (64%)	
High protein level	13 (47%)	46 (51%)	
High cells count	0	4 (5%)	
Oligoclonal Band	1 (4%)	1 (1%)	
High Protein Level + High cells count	1 (4%)	4 (5%)	
High cells count + Oligoclonal Band	1 (4%)	1 (1%)	
High cells count + High cells count + Oligoclonal Band	0	1 (1%)	
Not reported	4	15	
Brain Magnetic Resonance Imaging/Computed Tomography scan [n. patients (%)]:			
Not reported	4	0	
Normal findings	16 (59%)	64 (62%)	0.82
Abnormal findings	11 (41%)	40 (38%)	
Brain SPECT scan [n. patients (%)]:			
Not reported	26	86	
Normal findings	1 (20%)	7 (39%)	0.62
Abnormal perfusion	4 (80%)	11 (61%)	
Treatment [n. patients (%)]:			
Steroids	20 (65%)	87 (84%)	0.02
IVIg	0	0	
Plasmapheresis	1 (3%)	0	
Steroids + IVIg	5 (16%)	7 (6%)	
Steroids + Plasmapheresis	1 (3%)	5 (5%)	
Steroids + immunosuppressive medication	1 (3%)	3 (3%)	
Steroids + IVIg + Plasmapheresis	3 (10%)	1 (1%)	
Spontaneous remission	0	1 (1%)	
Clinical responsiveness to immunomodulatory treatment [n. patients (%)]:			
Complete remission	24 (77%)	85 (83%)	
Partial remission	4 (13%)	18 (17%)	0.59
Death	3 (10%)	0	

Anti-TPO: anti-thyroid peroxidase antibodies; anti-TG: anti-thyroglobulin antibodies; anti-NAE: anti-NH2-terminal of α-enolase antibodies; IVIg: Intravenous immunoglobulin.

Clinical and laboratory features of HE patients with and without SE are summarized in Table 1. The two groups did not significantly differ for thyroid status (most patients were euthyroid or mildly hypothyroid), presence of anti-thyroid antibodies, CSF analysis parameters, brain MRI/CT scan findings (the most common abnormalities were focal or confluent white matter lesions, diffuse or asymmetrical atrophy, meningeal enhancement) and single-photon emission

computed tomography (SPECT) scan findings (demonstrating in most cases focal or global hypoperfusion in multiple brain areas). Of note, SPECT data were not available in the majority of patients (Table 1).

Conventional AEDs were ineffective in managing SE or seizures in non-SE patients (data not shown). Most patients in both groups received steroids treatment (oral prednisone 1–2 mg/kg/day; intravenous methylprednisolone, 500–1000 mg/day). A significantly greater percentage of SE patients than non-SE patients (35% vs. 13%, p = 0.02) required further treatment with IVIg, plasmapheresis, or other immunosuppressive medication (i.e. cyclophosphamide, azathioprine) alone or in combination with steroids (Table 1). By this approach, 77% SE patients and 83% of non-SE patients achieved a stable and complete remission of symptoms (Table 1) while incomplete response or death was observed in 23% of SE patients and 17% of non-SE patients (p = 0.59). Residual symptomatology in patients with incomplete response included motor sequelae (cerebellar ataxia, arm tremors, multifocal myoclonic movements, and hemiparesis) and cognitive abnormalities (memory impairment, learning difficulties, time disorientation, and executive functioning impairment) [14,18,22,24,32,36–38,41,43,44,47,48]; see also Appendix A]. No differences in residual symptoms were observed between patients with and without SE (data not shown).

In the SE group, there was a trend for a non-significant association between GCSE and treatment outcome (p = 0.08) whereas age, sex, serum anti-thyroid antibodies, CSF analysis parameters and brain imaging findings were not associated with the outcome (Table 2).

4. Discussion

We identified 31 HE patients manifesting SE characterized by a wide semeiology variability. GCSE accounted for more than a half of cases (52%) [7,9,10–12,14,16–19,24–26], and NCSE, FSE, GASE, and EPC were reported in the remaining patients [5,6,8,13,15,20,21–23,26,27, our case]. Comparison of SE and non-SE patients did not reveal any difference between study groups in thyroid status, presence of antithyroid antibodies, CSF analysis parameters, and brain MRI/CT/SPECT scan findings. In this regard, a few points can be made. Although hypothyroidism may be associated with SE [49,50], we found no evidence of such an association in this sample. Rather, the thyroid function spanned the entire spectrum of overt (though mildly) hypothyroidism, subclinical hypothyroidism, euthyroid or, rarely, even hyperthyroidism. The similar occurrence of antithyroid antibodies in SE and non-SE patients is in line with the still discussed role of such antibodies in HE. A direct toxicity has never been demonstrated and antibodies could merely be an innocent bystander or a marker for the existence of other antibodies that are directly pathogenic [3]. Finally, comparison of CSF findings was also unrevealing and consistent with data from the literature showing normal CFS analysis in a consistent minority of patients or, more frequently, mild inflammation mostly characterized by elevated proteins and rarely by oligoclonal bands. The findings from MRI either returned a normal study or, less commonly, showed non-specific focal or diffuse white matter hyperintensities that had no diagnostic specificity for SE. Functional imaging data were available in a small number of patients and mostly showed widespread non-specific hypometabolism.

SE in HE did not respond to AED, and steroid treatment was not always effective in suppressing SE. More intensive and combined immunomodulatory treatment with IVIg, plasma exchange, or other immunosuppressive medication was necessary in a greater percentage of SE patients to reach a favorable clinical outcome. Overall, about 4/5 of both Se and non-SE patients yielded complete remission by this sequential therapeutic approach with steroids and other immunomodulatory medication. According with the results of our review, GCSE might have a worse response to immunomodulatory therapy than other forms of SE, whereas thyroid status, presence of antithyroid antibodies, CSF analysis parameters, and brain MRI/CT/SPECT scan findings were not related to SE outcome.

Our analysis has limitations. In reviewing the results of antibodies assay, we did not consider quantitative data because of the involvement of different laboratories. Despite of this limitation, it is to be considered that a causal or linear relationship between antibody titers and HE has never been established. The relatively low number of SE and non-SE patients might have contributed to the lack of association between SE and some clinical/laboratory parameters. This may have been

Table 2
Clinical and laboratory findings in patients with Hashimoto's encephalopathy and status epilepticus yielding complete/partial remission or death following immunomodulatory treatment.

	Patients undergoing complete remission following immune-modulatory treatment (n. 24)	Patients undergoing partial remission/death following immune-modulatory treatment (n. 7)	P
Mean age at onset ± SD	39.3 ± 24.8	45.6 ± 17.9	0.53
Women sex (%)	19 (79%)	6 (86%)	1
Classification of status epilepticus [n. patients (%)]:			
GCSE	10 (42%)	6 (86%)	0.08
Other types of SE	14 (58%)	1 (14%)	
NCSE	9 (38%)	0	
FSE	3 (12%)	1 (14%)	
GASE	1 (4%)	0	
EPC	1 (4%)	0	
Anti-thyroid antibodies [n. patients (%)]:			
Anti-TPO or Anti-TG or NAE	9 (38%)	3 (43%)	1
Anti-TPO + anti-TG or Anti-TPO + anti-TG + Anti-NAE	15 (62%)	4 (57%)	
Cerebrospinal fluid analysis [n. patients (%)]:			
Normal findings	10 (48%)	5 (83%)	0.18
Abnormal findings	11 (52%)	1 (17%)	
Not reported	3	1	
Brain Magnetic Resonance Imaging/Computed Tomography scan [n. patients (%)]:			
Normal findings	14 (64%)	2 (40%)	0.37
Abnormal findings	8 (36%)	3 (60%)	
Not reported	2	2	

GCSE: Generalized convulsive status epilepticus; NCSE: Non-convulsive Status Epilepticus; FSE: Focal status epilepticus; GASE: generalized absence status epilepticus; EPC: Epilepsia partialis continua; Anti-TPO: anti-thyroid peroxidase antibodies; anti-TG: anti-thyroglobulin antibodies; anti-NAE: anti-NH2-terminal of α-enolase antibodies.

particularly relevant for MRI and SPECT findings, so that we do not know whether the lack of association between MRI findings/SPECT hypoperfusion and SE merely resulted from low study power. Likewise, the small number of SE patients might have contributed to the lack of significance in the association between GCSE and treatment outcome.

5. Conclusions

Despite of the foregoing limitations, we can conclude that: (a) SE occurring in the course of HE may have variable phenomenology and awareness of this variety by clinicians would facilitate early HE diagnosis; (b) we could not find any association with clinical/laboratory parameters related to HE diagnosis and possibly reflecting brain inflammation and damage; (c) SE in HE does not respond to AED but may well respond to steroids alone or combined with other immunomodulatory treatment; (d) regardless of the treatment schedule about one quarter of SE patient can not achieve complete remission. GCSE might be a factor negatively influencing outcome, which may reflect hemodynamic and metabolic complications that are usually associated with convulsions rather than factors more closely related to HE. This is suggested by the lack of correlation between treatment outcome and serum anti-thyroid antibodies as well CSF and MRI finding.

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Declaration of Competing Interest

None to be declared.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.seizure.2019.06.020.

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