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Recurent Coma in Patient with Hashimoto's Encephalites

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Abstract
Encephalitis is a severe inflammatory disorder of the brain with many possible causes and with difficult differentiate of diagnosis. One of the most important challenges is the elucidation of the causes of seizure disorders. Encephalopathy associated with autoimmune thyroid disease (EAAT) is a rare clinical entity, which presents with unspecific neurological symptoms. The clinical presentation is more frequently insidious, with cognitive and behavioural disturbance. Hashimoto's encephalopathy is rare disorder of presumed autoimmune origin characterized by cognitive decline, seizures, neuro-psychiatric symptoms, high titers of Anti-Thyroid-Peroxidase AB, and a positive response to steroids. There is no established correlation between the titer of TPO and severity of autoimmune encephalopathy, or its response to the therapy. Though the cortical and subcortical system may appear to be clinically involved in these cases, brain Magnetic Resonance Imaging (MRI) studies are usually unremarkable.
We presented one case of unexplained reasion of coma and refractory status epilepticus. The patient underwent CT, MRT, EEG examinations, spinal fluid examination on viruses and autoimmune antibodies, feces analysis on Hymenolepios, but rezult was negative. She was performed anticonvulsants therapy, but after short-term improvement patient was returning to the clinic with unexplained coma and convulsions.
We measured serum levels of thyroid autoantibodies and noticed it's elevated level, with statistical significant difference from normal values.

Keywords: Hashimoto encephalitis, TPO antibodies, Refractory and intractable seizures

Introduction
Encephalopathy associated with autoimmune thyroid disease (EAAT) is a rare clinical entity, which presents with unspecific neurological symptoms. The clinical presentation is more frequently insidious, with cognitive and behavioural disturbance that may associate with tremor, myoclonus, or ataxia. More rarely, clinical onset may be acute as stroke-like episodes, epilepsy, or psychosis. Hashimoto's encephalopathy is rare disorder of presumed autoimmune origin characterized by cognitive decline, seizures, neuro-psychiatric symptoms, high titers of Anti-Thyroid-Peroxidase AB, and a positive response to steroids. As seeing in most autoimmune diseases, HE is more prevalent in white females with a mean age of onset 45–55 years old. In general, TPO antibody targets thyroid peroxidase, which is located inside the follicular cell and catalyzes the thyroglobulin iodination process in the production of the thyroid hormones. There is no established correlation between the titer of TPO and severity of autoimmune encephalopathy, or its response to the therapy. The confusing nomenclature applied to autoimmune encephalopathies. The clinical picture, elevation of TPO antibodies, and positive response to corticosteroid therapy are essential for making the diagnosis of HE. Though the cortical and subcortical system may appear to be clinically involved in these cases, brain Magnetic Resonance Imaging (MRI) studies are usually unremarkable. with cognitive impairment reflects the evolution of these disorders. The relationship between HE and Hashimoto's thyroiditis (HT) remains uncertain, since there is no evidence that thyroid autoantibodies react with brain tissue and affect neuronal function, and the level of circulating autoantibodies is not correlated with the severity of neurological manifestations or with the response to treatment. Historically linked to autoimmune thyroiditis, the exact

mechanism of disease is not known. High titers of thyroid autoantibodies are found at the time of diagnosis.

Case

53 year old woman with unknown comas was hospitalized three times in different clinics during 4 months. Each attack was repeated about one month period. In all cases patient's relatives have found her in loss of consciousness and after this they have made patient hospitalized. Because of acute breathing problem artificial respiration was needed.

The patient was found unconscious at home,she had a series of tonic seizures in the limbs.CT was performed in dynamics, as well as, MRI and EEG examinations in dynamics, did not revealed pathological changes (pic 1-2)

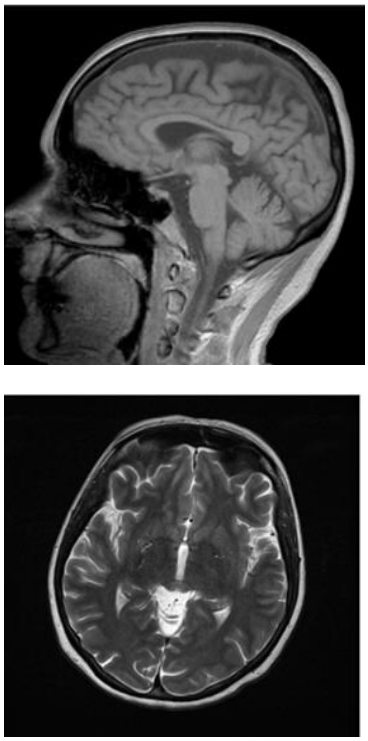

Fig-1

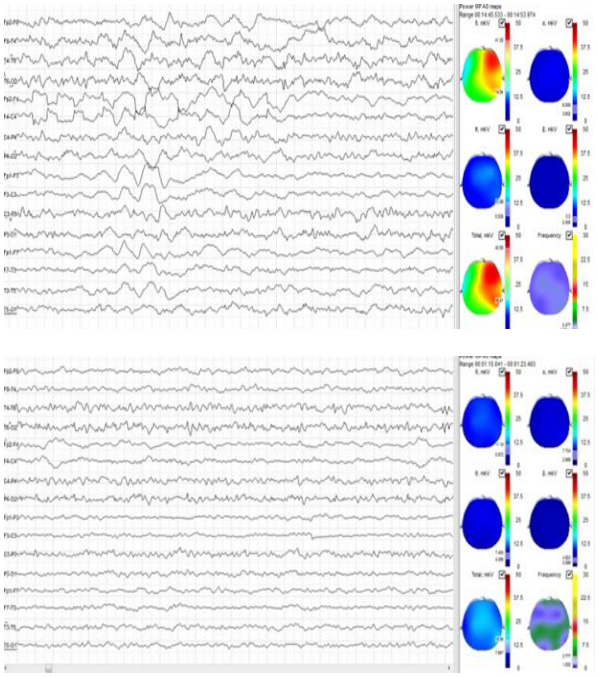

Fig-2

Blood tests –HIV-1.2, TRPA and test on borreliosis was negative. Biochemikal parameter was in normal ranges. Internal organ and thyroid gland ultrasonography investi gation, monitoring of cardiac rithem by holter, echocardiography, and cerebrospinal fluid analyzis could not find pathological changes. Four days later patient became adequate, breathing parameters was normal, and successfully was disconnected from mechanical ventilation. The patient was discharged from the clinic in a stable condition.

After 45 days from episode I the patient was once more discovered in uncon scious state. When arriving at clinic, the patient was contactless, she expressed severe psychomotor agitation, had ambiguous speech, periodic tonic seizures in right limbs, myosis, bilateral pathological reflexes.

She was performed anticonvulsant therapy with Levetiracetam, but for the purpose of seizures relief was added Propofol infusion. On this background for 2 days was still observed unit myoclonic movements mostly in right limbs. There was performed the repeated studies, including: brain CT, MRT examination with contrast enhancement, cerebrospinal fluid examination, EEG in dynamics, hematologic studies in dynamics, 48-hour Holter monitoring. after the aritmologist's recommendation, was inserted the loop recorder, used for diagnosis with recurrent unexplained episodes of palpitations or syncope, for long-term monitoring in patients at risk for arrhythmia and for risk stratification. Recording was examined once a week, also EEG and neurological status assessment. Patient state was improved and was disconected from mechanical ventilation.

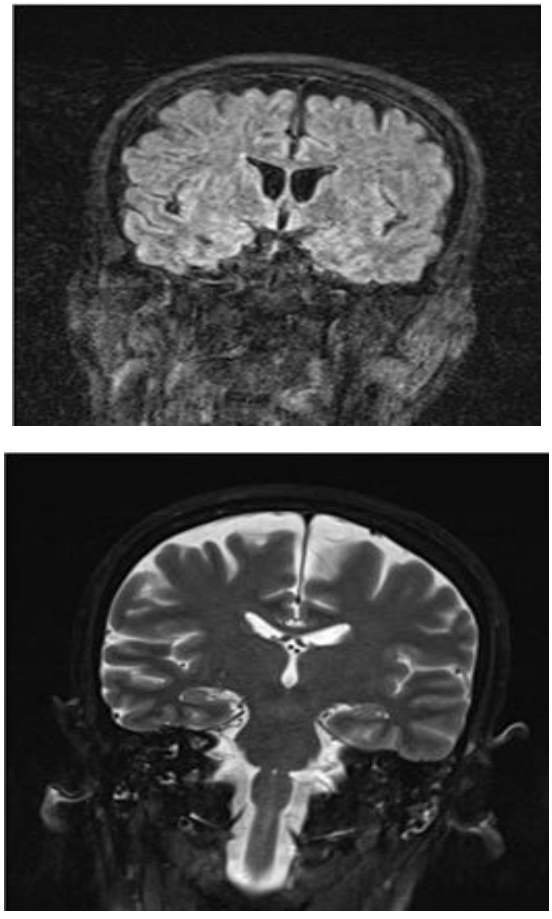

Fig .3: MRI study in dynamic without pathological changes

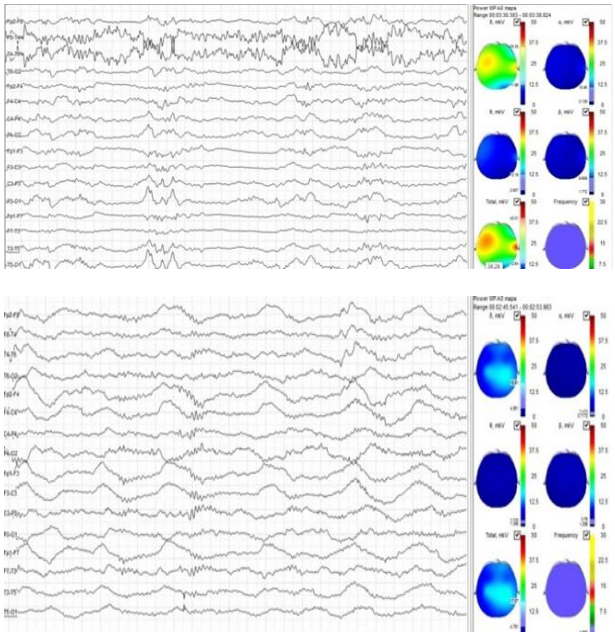

Fig.4:.EEG study in dynamic without speciphic pathological changes

The patient complained of general weakness, loss of appetite, paleness. Several days before the episode III the patient was started of cognitive dysfunction, oligo bradykinesia, bradypsychia, tremor of limb, incoordination.In the hospital, the patient had tonic-clonic seizures several times, with tonic tension in left limb at the onset. There was started propofol infusion, on this background there still continued tonic tensions of limbs, unit and group myoclonic movements in different areas of the body. According to the information gained from Loop recorder cardiological pathology was finally excluded. The patient underwent CT, MRT, EEG examinations (Picture 5 and 6),

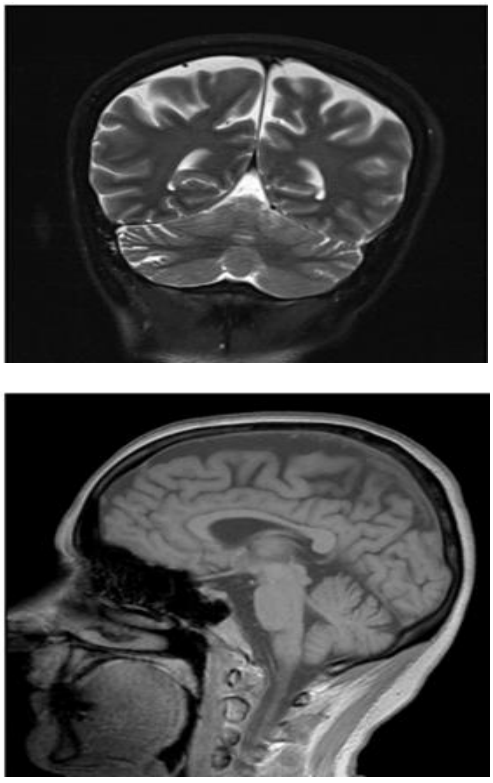

Fig.5

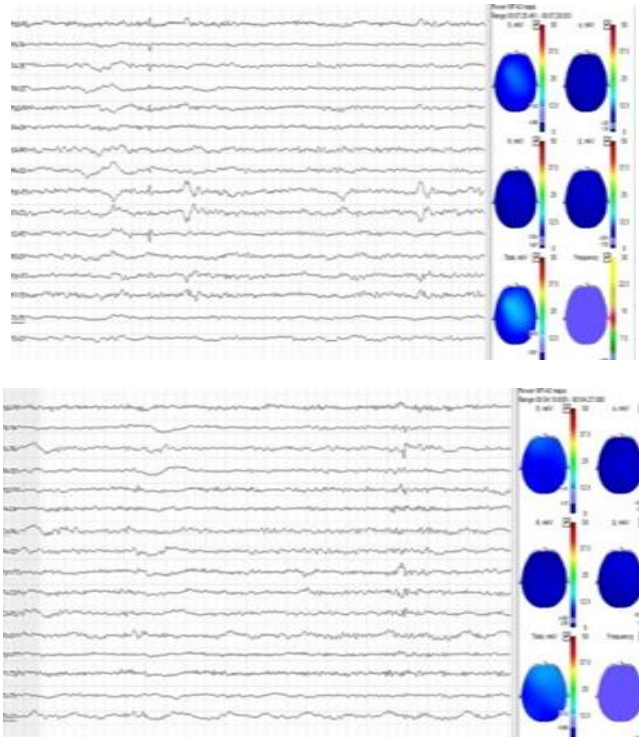

Fig.6

Cerebrospinal fluid examination on viruses and autoimmune antibodies, feces analysis on Hymenolepios was negative. Biochemistry and immunology features were in normal value (Table 1 and Table 2) Table 1

Table.1

Biochemistry	Result	Reference Range
LGI1 (Leucine-rich glioma inactivated protein 1) Antibody	<1:10	<1:10-
GABA-B Receptor Antibody"	<1:10	<1:10-

Table.2

Immunology	Result	Reference Range
Neuronal Nuclear Antibody (Anti-Hu)^	<1:50	<1:50-
Anti-Yo (Purkinje cell)^	<1:50	<1:50-
Anti-CV2 Antibody^	Negative	Negative
Anti-Ma Antibody^	<1:10	<1:10

In several days the patient's condition worsened, patient became inadequate, the movement was restricted, began periodic strong tremor of limbs. The patient started having plasmapheresis.We measured serum levels of the following parameters: antithyroid peroxidase antibodies (antiTPO-Ab), anti-thyroglo bulin antibodies (Tg-Ab), anti-TSH-receptor antibodies (TSHR-Ab), thyroid-stimulating hormone (TSH). We noticed elevated antiTPO-Ab, and anti_TPO-ab, with statistical significant difference from normal values.(Tabl.3).

Table. 3

	Result	unit	Reference Range
Free Thyroxine FT4	9.89	Pmol/l	12-22
Free Triiodthyronine FT3	5.04	Pmol/l	3.1-6.6

Thyroid-stimulat.Hormone TSH	12	mIU/l	0.4-3.8
Microsomal thyroid antibodies	**248**	KU/l	<34
Thyreoglobulin autoantibodies	**235**	KU/l	<115
5-Hydroxyndoleacetic acid/U	**<0.40**	Mg/24h	<8

The patient was started treatment with L-Thyroxin and high doses of oral prednisone -100 mg/day. After 6 day from onset of treatment was observed decresion of anti-TPO and anti-Tg level. (Tabl.4)

Table.4

	Result	Unit	Reference range
Anti-Tpo	16`1.7	IU/ml	<34
Anti-Tg	215.3	IU/ml	<115

PET tomography shows pathological changes: bilateral symmetrical increased FDG uptake of thalami and striatum (autoimmune limbic), bilateral increased FDG uptake of thyroid lobes (thireoiditis). (Pict.7)

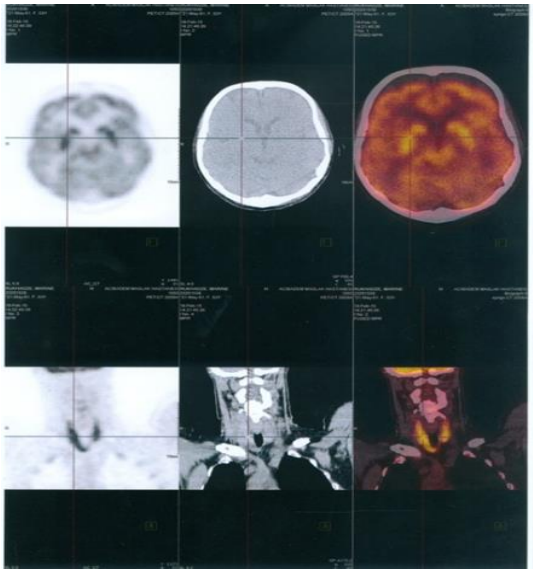

Fig.7

Discussion

We presented a case of encephalopathy associated with thyroid autoantibodies or so-called Hashimoto's Encephalopathy (HE). This is an uncommon and frequently underrecognized autoimmune disorder. Historically linked to autoimmune thyroiditis, the exact mechanism of disease is not known. High titers of thyroid autoantibodies was found in our clinic after multiple investigations in different insititiutions.

The underlying pathophysiology of HE is not well delineated. It does not appear to be directly related to the dysfunction of the thyroid gland as the majority of the patients are clinically and biochemically euthyroid. There is no established correlation between the titer of TPO and severity of autoimmune encephalopathy

.But in our case was observed significant increase of thyroid autiantibodies.

Treatment with immunotherapy is reported to be successful in as many as 64% of patients with autoimmune desease. Plasmapheresis, is reported as therapeutic options for initial treatment., but long-term maintanace immunosuppression with prednisone have being used up tu several months to attain long-term remission. But plasmapheresis in our case was not effective. Based on the clinical picture of fluctuating cognitive Status with subacute onset, seizures and high titer of TPO antibodies, the diagnosis of autoimmune encephalopathy was made. Just PET tomography was remarkable, diagnosing bilateral symmetrical increased FDG up take of thalami and striatum (autoimmune limbic), bilateral increased FDG uptake of thyroid lobes (thireoiditis).

High dose prednisone was initiated, which was followed by significant improvement in her cognitive function and cessation of seizures.

Conclusion

An autoimmune cause of frequent, refractory and intractable seizures was suspected based on presence of neural antibody, inflammatory changes indicated in spinal fluid or on MRI. We presented case, when never have not been found reason of seizures.

Based on the clinical picture: fluctuating cognitive status with subacute onset, seizures, high titer of thyroid antibodies, PET tomography findings, steroid treatment efectivness, there was made the diagnosis of autoimmune thyreoiditis and encephalopathy

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