Young woman with confusion and cognitive impairment: a case report

Sumišimas ir kognityvinių funkcijų sutrikimai jauno amžiaus moteriai: klinikinio atvejo analizė

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SUMMARY

This clinical case is about a 34-year-old woman who was admitted to mental hospital for psychiatric treatment for the episodes of anger, agitation, confusion with verbal hallucinations, shifting between insomnia or hypersomnia, apathy, lack of interest in daily life and cognitive impairment. The patient was misdiagnosed and unsuccessfully treated for two years before she was diagnosed with Hashimoto's encephalopathy (HE). During the treatment period, the differential diagnoses of this case included potential mental disorders, neuroinfections, diseases caused by parasites, metabolic disorders, epilepsy and neurodegenerative diseases of the brain. The response of psychiatric symptoms to the treatment with antipsychotics was limited: only little adjustment was made on both the states of confusion and the episodes of aggressive behaviour and agitation. Only a complex evaluation of thyroid axis hormone concentrations, anti-thyroid peroxidase (anti-TPO) concentration, changes in cerebrospinal fluid and the course of the disorder allowed diagnosing autoimmune thyroid disease named HE. Treatment with glucocorticosteroids was prescribed, and full recovery with complete recovery of cognitive functions, that was reached as result of the treatment, reaffirmed the correctness of the diagnosis established.

This clinical case provided us with new clinical experience, and once again has drawn attention to the importance of parameters of thyroid axis function in terms of the development and course of and recovery from mental disorders.

Key Words: Thyroid, Hashimoto's encephalopathy, Anti-thyroid peroxidase, Cognitive impairment.

SANTRAUKA

Šiuo klinikiniu atveju pristatoma 34 metų moteris, gydyta psichiatrijos skyriuje dėl pykčio, sujaudinimo, sumišimo būsenų su klausos haliucinacijomis, miego sutrikimų- nuo visiškos nemigos iki mieguistumo, apatijos, nesidomėjimo kasdiene veikla ir kognityvinių funkcijų blogėjimo. Sutrikimo priežastis nebuvo nustatyta ir pacientės psichikos sutrikimo simptomai buvo nesėkmingai gydomi du metus. Gydymo metu pacientės būklė diferencijuota tarp endogeninio psichikos sutrikimo, neuroinfekcijos, parazitų sukeltos ligos, medžiagų apykaitos sutrikimų, epilepsijos bei neurodegeneracinių ligų. Atsakas į medikamentinį gydymą antipsichotiniais vaistais buvo ribotas: tiek sumišimo būsenos, tiek agresyvaus elgesio ir sujaudinimo epizodai mažai koregavosi. Tik kompleksiškai įvertinus skydliaukės ašies hormonų koncentracijas, antikūnų prieš skydliaukės peroksidazę (anti-TPO) pokyčius, likvoro pokyčius ir sutrikimo eigą dinamikoje, pavyko nustatyti sutrikimo priežastį - autoimuninę skydliaukės ligą, vadinamą Hašimoto encefalopatija. Paskirtas gydymas gliukokortikosteroidais ir gydymo eigoje pasiektas visiškas pasveikimas su visišku kognityvinių funkcijų atsistatymu ir psichikos simptomų korekcija dar kartą patvirtino nustatytos diagnozės teisingumą.

Šis klinikinis atvejis mums suteikė naujos klinikinės patirties, ir dar kartą atkreipė dėmesį į skydliaukės ašies funkcijos parametrų svarbą psichikos sutrikimų išsivystymui, eigai ir sveikimui.

Raktiniai žodžiai: Skydliaukė, Hašimoto encefalopatija, Antikūnai prieš skydliaukės peroksidazę, Kognityvinių funkcijų sutrikimas

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INTRODUCTION

Cognitive impairment is a syndrome, the expression of illnesses, but not a disease itself. Patients with cognitive impairment experience difficulties related to such mental functions like memory, paying attention, thinking, understanding and communication. These conditions are usually caused by the underlying disease; this could be a short-term problem or a permanent condition [1, 2[. There are known many causes of cognitive impairment. It is quite difficult to determine the origin of this condition in young people or in atypical cases. Common causes of cognitive impairment in young adults include the side effects of psychotropic medications, mental illness, epilepsy, substance abuse or withdrawal. It could be associated with hormonal misbalance, such as thyroid dysfunction [3, 4] or diabetes mellitus; some infectious agents, such as syphilis, HIV, diseases caused by parasites or other infectious diseases, or with early stages of Lewy body dementia [5]. Cognitive impairment might result from exposure to heavy metals, high levels of serum calcium, nutritional disorders, Wilson disease, rapid changes in sodium level, etc. [6, 7].

Research on the relationship between the disturbed thyroid function and cognitive impairment have been carried out in many studies. Hypothyroidism or hyperthyroidism, or even subclinical thyroid dysfunction are known to have a potential negative effect on cognitive functions. Therefore, screening of thyroid function is recommended in cases of cognitive impairment of all types, and serum Thyroid stimulating hormone (TSH) level is known as a standard screening tool in this case [8]. On rare occasions, it is not enough to test thyroid hormones because thyroid function in these cases is clinically and biochemically normal. When the disorder is of autoimmune origin, high titres of anti-thyroid peroxidase antibodies are present [3, 9, 10]. This interesting clinical case is about a young woman who suffers from reversible cognitive impairment and confusion, and demonstrates psychotic episodes due to autoimmune thyroid disease named as Hashimoto‘s encephalopathy (HE) or ”steroid responsive encephalopathy associated with autoimmune thyroiditis“.

CASE

A 34-year-old woman was admitted to the Department of Psychiatry of the University Hospital due to episodes of anger, agitation, confusion with episodic verbal hallucinations, shifting between insomnia and hypersomnia, apathy, lack of interest in daily life, and cognitive impairment.

Psychosocial history: the grandmother of the patient was diagnosed with Alzheimer’s disease, both parents and the younger brother are not suffering from mental disorders. The early psychomotor development of the patient was timely, but since her childhood demonstrates a more pronounced sensitivity, gets angry easily, is prone to impulsive behaviour, even aggression during conflicts. Started attending school timely, completed 12 years of education at the secondary school, the learning outcomes were relatively mediocre, the patient got no further education, the pursuit of knowledge is quite limited. Got married when was 18 years old, but divorced quite quickly, does not explain the reasons for this. She has been living in a second marriage for 15 years, has two children, resides in a village. For the last 5 years she has been working as an auxiliary farm worker in a grain warehouse. She was a non-smoker, reported mild alcohol consumption, denied drug use. 15 years ago she was involved in a car accident, suffered a head injury with loss of consciousness, was admitted to inpatient treatment, but arbitrarily withdrew herself from the medical facility before completing treatment; did not apply anywhere, was feeling good. No previous history of other somatic diseases. 14 years ago, when the patient was 20 years old, after the first child delivery, episodes of inappropriate behaviour were observed: the patient became irritable, angry, conflicted with her family members and the husband, was oblivious, the patient complained that she cannot remember where did she place the things. Such a period of exacerbated irritation lasted for about 5 years, but the patient did not seek help anywhere. When the patient was 25 years old, after the second child delivery, the patient’s behaviour became normal again: she became calmer, conflicted less, though episodically complained of bad memory.

Two years ago, when the patient was 32 years old, her behaviour has changed for no apparent reason: she would episodically become agitated, aggressive, episodes of anger were triggered for no reason, during them the patient could not control herself, she beat her husband, began forgetting where she put the things, what happened during the day, could no longer cope with daily activities, could not sleep during the nights but used to spent all day lying in bed. Memory of the current events (a fixative memory) particularly deteriorated – the patient would forget the things that have just happened. Due to changes in mental state, at the initiative of her relatives, the patient sought help, was hospitalized and treated for 2 months in the Department of Psychiatry of the regional hospital. The patient was prescribed with Haloperidolum 10mg per day, Diazepamum 20mg per day and Valproic acid 1,5 g/ per day to treat psychomotoric agitation and aggression. The patient’s condition worsened during the treatment, episodes of agitation and aggressive behaviour occurred more frequently, disturbed fixative memory was observed. Computed tomography (CT) scan of the brain did not show any changes, despite the visible plaques in basal ganglia. The cause of the mental disorder was differentiated with thyroid dysfunction, the human immunodeficiency viruses (HIV) or syphilis. But these disorders were excluded following laboratory tests. For memory disorders that were gradually worsening, and as the episodes of aggressive behaviour persisted, it was recommended by a neurologist to differentiate the condition with cerebral frontotemporal degeneration, and the patient was referred for diagnosis clarification and treatment to the University Hospital.

During the treatment in the Department of Psychiatry of the University Hospital, impaired orientation in time and place was observed (the patient stated that she is in another city, was confused about the exact month and season of the year). She knew her forename and surname but did not remember her correct date of birth. The patient spoke slowly, quietly, thinking was inconsistent, episodically became angry, spoke with a raised tone of voice. Emotions were rapidly changing; anger reactions were triggered for no reason. The general mood was lowered, the patient was indifferent to the environment and staying in the hospital, spending all the days lying in bed. The patient often waked up at night, became agitated and fearful,

when walking down the hallway kept asking the staff where she is and why she cannot go home. Sometimes she behaved like hearing voices: she spoke with "her children", was afraid of something. Short-term (fixative) memory impairment was observed.

This condition was evaluated as dementia (unspecified) and the plan of tests and examinations was drawn up to determine the origin of dementia.

Laboratory tests carried out: general blood test (normal), biochemical blood test – in thyroid hormone test – Free thyroxine (FT4) and TTH concentrations (normal), Free triiodothyronine (FT3) slightly decreased – 3.17 pmol/l (norm 3.34–5.14 pmol/l), anti-thyroid peroxidase (anti-TPO) indicator increased –236.73 kU/l; (norm 0–3.2 kU/l), but was evaluated as clinically insignificant. Glycaemia slightly increased – 7.61 mmol/l; (norm 4.1–6.6 mmol/l), but was decreased to the norm in dynamics. Decreased ionized calcium - 0.99 mmol/l, (norm 1.2–1.43mmol/l). Concentrations of copper, phosphorus – within the normal range.

Cerebrospinal fluid (CSF) test revealed the elevated general protein – 1.15 g/l (norm 0.15–0.45) and cytosis – leukocytes 17 x 106/l (norm <8 x106/l).

Tests on infectious agents: test for antibodies against tick-borne encephalitis virus, Lyme disease, syphilis, HIV (1/2 antibodies and p24 Ag antigen), Yersinia pseudotuberculosis, Yersinia Enterocolitica – showed negative results. Tests for toxoplasmosis- Toxoplasma gondii Antibodies, IgG – showed positive results, and Toxoplasma gondii Antibodies, IgM - negative results. The results were evaluated by infectious disease specialist was evaluated as a result of old former contact with infection, but to be unable to cause an existing cognitive impairment.

Electroencephalogram (EEG) was carried out – within the normal range.

Ultrasound imaging of the upper part of the abdomen – within the normal range. X-ray of the chest – slight fibrotic changes on the right side of the lower lungs that have been evaluated as clinically insignificant.

Magnetic resonance imaging (MRI) of the brain: the hypointense foci, probably caused by calcinates described by cerebral CT, are observed in T2W/GRE images of globus pallidus area on both sides and has been evaluated as a variant which is possibly within the normal range. Slightly more sharply visible mucosa of frontal and parietal sinuses.

Cognitive testing showed difficulties in orientation in time, partly – at the place, decreased extent of fixative memory, reduced ability to reproduce information to the maximum, impaired storage of the information in long-term memory. Concentration of attention was fluctuating, which makes it difficult to remember: the patient had difficulty concentrating and shifting attention and reproducing information. Mini mental state examination (MMSE) test results – 17 points, indicated moderate cognitive impairment.

Neurophthalmologist diagnosed retinopathy and retinal vascular lesions (light stasis at the bottom of the eyes). Kayser–Fleischer rings were not visible, the optical media were transparent, discs of the optic nerve were pink. It was recommended to follow up.

Consultation of neurologist: considering the tests and examinations carried out, the data for specifying the dementia type was insufficient. Due to the prevalent clinical picture - disorientation, progressive deficits in cognitive function, significant behavioural impairment, it was recommended to carry out Positron emission tomography (PET) for the purpose of differentiating with frontotemporal neurodegeneration, Alzheimer's disease, or other neurodegenerative disorders. Unfortunately, the PET examination was terminated due to the patient's aggressive and agitated behaviour during the examination.

During psychiatric treatment with Haloperidol up to 10 mg daily in combination with Quetiapine up to 500 mg daily, the control of impulses was improved and aggressive behaviour was reduced. However, the patient's condition was changing: episodes of more adequate behaviour lasting from 15 minutes to half an hour were periodically observed. During them, the patient demonstrated consistent thinking, her contact with others was good, she was interested in her condition, test and treatment plans, and all this was followed by physical and mental asthenization, the patient used to become irritable, she used to become angry, propensity to aggression persisted. During the night, episodic hearing hallucinations were persisted (the patient could hear children crying) which resulted in episodes of psychomotor agitation. The condition was evaluated as an organic hallucinosis. During 40 days of treatment type of dementia could not be specified and without significant changes in mental state the patient was discharged for outpatient treatment and nursing, the patient was advised to continue outpatient treatment with Haloperidol tab. 10 mg/ per day and Quetiapine tab. 150 mg/per day.

For the second time, the patient was hospitalized to the University Hospital after 1.5 year, at the initiative of her relatives, since the patient's condition ranged from episodes of agitation and aggression to complete apathy, avolition, episodic hearing hallucinations, and impaired memory, both fixative and long-term.

Laboratory tests: moderate anaemia – erythrocytes 3.7 x1012/l (norm 3.9–5.1), haemoglobin – 126 g/l (norm 119–146), leucocytosis 9.99 x 109/l (norm 4.4–9.7); increased the erythrocyte sedimentation rate (ESR) – 53 mm/h, (norm 0–30)), calcium and phosphorus – within the normal range thyroid axis hormone tests – decreased FT3 –2.74 pmol/l, FT4 – 12.15 pmol/l (normal), TTH normal, anti-TPO remained elevated (97.49 kU/l). CSF was transparent, but total protein remained elevated to 0.7g/l, glucose, lactates, chlorides - within the normal range, mononuclear cells 100%, elevated leukocytes to 7 x 106/l – but the changes compared with the first hospital stay – decreased.

During the discussion of psychiatrist, neurologist and endocrinologist, considering the medical history, clinical picture and the results of laboratory tests, CSF and CT, a preliminary diagnosis was formulated: autoimmune thyroiditis and HE. Treatment with pulse therapy with the glucocorticoids – Methylprednisolone injections 1 gram per day for 3 days was recommended – by monitoring the general and mental state of the patient for possible exacerbation of the psychotic disorder. After three days the treatment with Prednisolone tab. orally 1.5 mg/kg should be continued (the patient weighs 124 kg), then dose decreasing by 5 mg every 2 days until stopping the medication completely.

During the treatment with glucocorticoids, blood glycaemia

level up to 15mmol / l was observed, it was corrected with short-acting insulin injections.

Already after the first 10 days of treatment, the patient's mental condition started to improve, agitation gradually decreasing, confusion episodes and auditory hallucinations disappeared. Doses of antipsychotic medications have been reduced to a minimal. However, lack of interests, poor hygiene and apathy was persisted. Concentration of attention, short-term memory, orientation in place and in time was improved. Psychological testing of cognitive functions: MMSE results – 22 points, indicated mild cognitive impairment. The patient started to show the interest in her treatment results and after 3 weeks of treatment with glucocorticoids she was discharged from the hospital for outpatient treatment with recommendations to take Prednisolone under the regimen prescribed, gradually reducing the dose until complete withdrawal. After completion of the treatment, follow up of thyroid hormone tests and anti-TPO concentration was recommended.

The information about the patient's condition was received from the patient's relatives after 6 months period. They informed that the course of glucocorticoid treatment was successfully completed in an outpatient setting, the concentrations of thyroid axis hormone tests were within normal range. The patient's mental condition improved, episodes of confusion and psychosis disappeared, her memory improved. The patient managed to do daily activities, to take care for the family. However, the temper of the patient remained quite impulsive (as it has been the case since her very young days), but it was not seen as a mental disorder.

DISCUSSION

This case report is about the patient who was misdiagnosed and unsuccessfully treated during two years period before she was diagnosed with HE. The case was differentiated between mental disorder, diseases caused by parasites, metabolic, endocrine disorders, epilepsy, neuroinfections and neurodegenerative diseases of the brain. Response to treatment with antipsychotics was limited and cognitive function deteriorated in dynamics. The patient fully recovered only after she was diagnosed with HE and received treatment with gliucocorticosteroids.

HE is related to Hashimoto's thyroiditis and also is known as Steroid Responsive Encephalopathy (The word encephalopathy can refer to all kinds of brain damage and diseases). First time HE was described in medical journal The Lancet Brain et al in 1966 (11). Number of clinically diagnosed and treated cases of HE increased only in early 2000. The incidence of HE is greater in women. The average age of the patients who develop HE is about 40 years [12]. Thyrotropin-releasing hormone was originally thought to have a toxic effect on the central nervous system, but today it is known that encephalopathy is associated with high titers of anti-thyroid peroxidase antibodies and anti-thyroglobulin (anti-Tg) [13]. Often enough, HE remains undiagnosed and untreated for a long time because of insufficient disease-specific signs and symptoms, and because of strongly varied clinical picture and highly variable neuropsychiatric symptoms [14]. In general, in clinical practice HE is a rare disease. It can be often undiagnosed because of varied clinical expressions, or it can be misdiagnosed when confused with other psychiatric (depression, schizophrenia) or neurological disease (Alzheimer's disease, epilepsy, viral encephalitis, other forms of autoimmune encephalitis including anti-NMDA receptors encephalitis) [4, 15].

The diagnosis of HE should be considered in younger patients (under 50 years of age) when unexplained cognitive impairment with psychiatric symptoms, clinical or subclinical hypothyroidism is present. The symptoms usually develop gradually and occur within 1 to 12-month period. HE typically manifests itself as slowly progressing cognitive impairment until it evolves into dementia, episodic psychosis, somnolence or confusion. It may also include focal and generalized seizures, myoclonus, pyramidal tract signs [16, 17]. Laboratory (increased liver enzyme levels and erythrocyte sedimentation rate, pathological cerebrospinal fluid findings, increased anti-thyroid peroxidase antibodies and antithyroglobulin antibodies) and radiological (magnetic resonance imaging, single photon emission computer tomography) findings should be considered to diagnose HE. Diagnostic criteria of HE have been proposed by Graus et al. The diagnosis can be established when all six of the following criteria are met:

1. Encephalopathy with seizures, myoclonus, hallucinations, or stroke-like episodes; in our clinical case – the encephalopathy and hallucinations were observed.

2. Subclinical or mild overt thyroid disease (usually hypothyroidism); in our case TTH and FT4 were normal, with slightly decreased FT3.

3. Brain MRI normal or with non-specific abnormalities; in our case- without specific MRI changes.

4. Presence of serum thyroid (thyroid peroxidase, thyroglobulin) antibodies; in our case anti-TPO remained elevated.

5. Absence of well-characterized neuronal antibodies in serum and CSF; in our case- only elevated total protein and leucocytes in CSF were observed.

6. Reasonable exclusion of alternative causes; in our case-other causes were excluded [18]. Corticosteroids are the main curative remedy for HE, and most of the patients respond well to them [19]. 90 to 98% of patients with HE often fully recover from the disease after the treatment with steroids [9].

In the case of our patient, the differential diagnosis included the possibility of mental disorders. The personality of our patient has been complicated since her very young age, with a tendency to impulsive, aggressive behaviour, unmotivated acts (divorce with the first husband, arbitrary withdrawal from the hospital after the head injury), mood changes from hyperactive to apathetic, changes in mental state after childbirths, which promoted the intention to differentiate with bipolar affective disorder or impulsive personality disorder. However, episodic states of confusion with auditory hallucinations and inflammatory changes in CSF suggested the organic nature of the disease.

In this case we observed behavioural changes that are similar to behavioural variant of frontotemporal dementia (FTD) that manifests itself in the form of psychosis, disinhibition, compulsions, and apathy. But cognitive impairment in early stages of the disease is not typical for FTD. According to Frontotemporal Dementia Consortium (FTDC), a potential diagnosis of behavioural variant of frontotemporal

dementia (bvFTD) requires at least 3 of 6 features, which include shared symptoms such as behavioural disinhibition, apathy, loss of empathy, compulsive behaviours, and deficit in executive function with decline in cognitive impairment. For the definite diagnosis of bv FTD additional diagnostic criteria have to be met, but histopathological changes and/or pathogenic genetic mutation of FTD were not examined [20]. We were unable to obtain any definite proof of frontal and/or temporal hypoperfusion or hypermethabolism. Also, in our case there were no signs of primary progressive aphasia and progressive non-fluent aphasia, and this, therefore, suggested a different cause.

The correct diagnostic decision during the first treatment was delayed by the fact that at normal FT4 and TTH concentrations, a slight decrease in FT3 and an increase in anti-TPO indicator were not considered as clinically significant. This interfered with the timely diagnosis of the autoimmune process and the identification of interfaces with cognitive impairment and confusion states. The period of establishing correct diagnosis prolonged by nearly 2 years because of our inexperience in diagnosing HE and the fact that we decided to choose the method of rejecting other causes of cognitive impairment.

The positive treatment results conclusively confirmed that diagnosis for this patient was established correctly. Thanks to this case we gained additional clinical experience, and once again drew our attention to the importance of thyroid axis function parameters in the development, course of and recovery from mental disorders.

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Received 01 December 2019, accepted 31 December 2019
Straipsnis gautas 2019-12-01, priimtas 2019-12-31