Condition of Discirculatory Encephalopathy in Hypothyroidism

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A B S T R A C T

In the scientific literature there are works devoted to the study of certain aspects of hemodynamics in hypothyroidism, but there is a lack of information about the features of cerebral circulation in conditions of deficiency of thyroid hormones. The aim of our study was to identify discirculatory encephalopathy and factors affecting its development in various forms of hypothyroidism.

A R T I C L E I N F O

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The prevalence of hypothyroidism in Uzbekistan is constantly growing. The highest prevalence of hypothyroidism is observed in the age group of postmenopausal women older than 50 years [1]. A clinical manifestation of endothelial dysfunction in patients with hypothyroidism is dyscirculatory encephalopathy (DE), a cerebral pathology that develops as a result of metabolic and vascular disorders [6, 7].

The study included 60 women aged 50-54 with a history of clinical and subclinical hypothyroidism. With all nosological units accompanied by hypothyroidism syndrome, an endocrinologist was obliged to identify neurological disorders. Thyroid status (TSH, f. T4, f. T3) was assessed twice, with an interval of 6 months. Mandatory in the study of the thyroid gland was the determination of antibodies to TPO, because. an increase in antibodies to thyroperoxidase in patients indicated damage to the central nervous system [8]. Hypothyroidism in patients of the main group was due to autoimmune thyroiditis. In order to identify hypertensive encephalopathy in all the subjects, a blood pressure study was carried out, women with blood pressure 125+4.0 / 70+10 mm Hg were included in the study. In addition to the above, the vessels of the neck were examined by duplex scanning using the General Electric Vivid 3.0 device (General Electric Healthcare, USA).Depending on the results obtained, all women included in the study were divided into 2 groups: group 1 - 30 patients with sub compensated hypothyroidism (SH) (TSH> 4.5 mU / l, free T4 within the reference values) and 2- group I - 30 examined with decompensated hypothyroidism (TSH is higher, and St. T4 is below normal). The exclusion criteria were hyperfunction of the thyroid gland, a history of indications of oncological diseases, stroke or heart attack. The studied women had subjective or objective mild cognitive deterioration: decreased memory, attention and intelligence, lethargy, apathy, drowsiness, the degree of these disorders depended on the severity of the decrease in thyroid function.These symptoms preceded neuromuscular manifestations such as Hoffmann's syndrome, hypothyroid myopathy. These states were emotionally colored: against the background of shortness of breath, anxiety, unmotivated fears, and vital anguish appeared. The difference between these states and panic attacks was their duration and stereotyping. It should also be emphasized that the appointment of novopassitis did not reduce the frequency and duration of seizures, however, the

appointment of replacement therapy with thyroid hormones contributed to a decrease in neurological symptoms.

A 50-year-old man was admitted to drowsiness and confusion after three general attacks, each lasting 3-4 minutes, over two days. For the last four months he has been acting strangely, putting things in their places and developing hand tremors. Two months prior to present admission, he was hospitalized elsewhere with a generalized tonic-clonic seizure and disinhibited behavior and was discharged on phenytoin. After that, he became withdrawn and stopped talkingOn admission, he was slightly burnt out and was noted to have bilateral xanthelasma (cholesterol 7.5 mmol/l). He was conscious, but drowsy and disoriented in time, place, and person. He experienced hallucinations and delusions and exhibited disinhibited behavior. Tremors were visible in all four limbs. Muscle tone, strength and reflexes were normal. There was no sensory or cerebellar dysfunction, and there were no signs of meningeal irritation. The white cell count was 13.2 x 109/L and the electrolyte and liver profiles were normal, except for elevated gamma-glutamyltransferase (296 U/L); her blood alcohol level was zero and phenytoin was in the therapeutic range of 15.2 mg/L. The electroencephalogram (EEG) showed a general slowdown consistent with encephalopathy. Head CT was normal, MRI showed a high signal in the right frontal lobe. The cerebrospinal fluid had an elevated protein of 1.67 g/L, but there was no pleocytosis; the polymerase chain reaction for herpes simplex virus was negative, as were the bacterial cultures. He had biochemical evidence of mild hypothyroidism with a TSH of 17 mU/L (normal 0.35-5.0) and an FT4 of 16.2 mmol/L (11-25). Anti-thyroid peroxidase antibody titers were very high, 2699 IU/mL (0-75). Tests for rheumatoid factor, antinuclear antibodies, All initial tests for common causes of metabolic encephalopathy were these negative. The diagnosis of Hashimoto's encephalitis was based on the clinical presentation, high titers of antithyroid antibodies, abnormal thyroid function, and elevated CSF proteins. After 4 days of treatment with oral prednisolone 60 mg daily, he was oriented and behaved much more normally on retesting, TSH rose to 85 mU/L, FT4 dropped to 3.2 mmol/L. He was started on thyroxine. Prior to discharge, his score on the mini mental status exam was 29/30, and his EEG was within normal limits.

The first case of "Hashimoto's encephalopathy" was reported in 1966. 1 In the few reported cases since then, the median age at admission has been 41 years. This condition has been reported in children and has a female predominance (ratio 3.6:1). Diagnosis is difficult because the underlying thyroid disorder is often subclinical and symptoms mimic other neurological conditions.(10) Encephalopathy tends to present acutely with confusion, focal or generalized seizures, and stroke-like episodes. 3 Other manifestations are dysarthria, hallucinations, stupor, headaches and myoclonus. 2 Diagnosis is confirmed by detection of elevated antithyroid antibodies (antithyroglobulin, antithyroid peroxidase, anti-TSH receptor, anticytoplasmic). (15.6) The main EEG abnormalities are generalized slowing, frontal rhythmic slowing, and triphasic waves. Cerebrospinal fluid protein is higher than normal in 75% of patients, and oligoclonal bands may be observed. 2MRI scans are normal in most patients, and reported abnormalities include generalized cerebral atrophy and reversible subcortical signal abnormalities. Single photon emission computed tomography showed several areas of hypoperfusion in several cases.Hashimoto's encephalopathy is thought to be caused by autoimmune vasculitis, and this notion is supported by the identification of the α-enolase autoantigen. Anti-α-enolase antibodies are associated with other forms of autoimmune vascular disease, including systemic lupus erythematosus. Some workers object to the term Hashimoto's encephalopathy, arguing that thyroid autoantibodies may simply be a marker for other autoantibodies, not yet identified, that cause cerebral vasculitis. In the absence of evidence of a causal relationship between thyroid autoantibodies and encephalopathy, a more appropriate name may be autoimmune thyroiditis-associated encephalopathy. (12.13) Steroids appear to be an effective treatment in these conditions, although no formal trials have been conducted. Other immunosuppressants have been reported to be effective. Some patients with hypothyroidism have responded completely to levothyroxine.conclusions. The decrease in blood flow in patients with clinical hypothyroidism suggested a more pronounced atherosclerosis and more precise diagnosis in order to identify atherosclerotic plaques. The appearance of connections between thyroid hormones and indicators of the common carotid artery, especially TSH and St. T3 was associated with a change in metabolism, which also led to endothelial dysfunction and further encephalopathy. This is also indicated by the level of antibodies to thyroperoxidase.

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